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OUTCOME OF LATERAL INTERNAL SPHINCTEROTOMY AS COMPARED TO MANUAL ANAL DILATATION

**A PROSPECTIVE STUDY AT
KENYATTA NATIONAL HOSPITAL** //

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**A DISSERTATION SUBMITTED AS PART FULFILMENT FOR THE
DEGREE OF MASTER OF MEDICINE IN SURGERY
UNIVERSITY OF NAIROBI**

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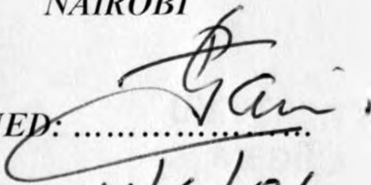
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LIST OF ABBREVIATIONS

MAD – Manual anal dilatation

LIS – Lateral internal sphincterotomy

HIV – Human immuno deficiency

AIDS – Acquired immuno deficiency syndrome

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SUMMARY

Background

Fissure in ano (anal fissure) is a common anorectal condition. Concern is drawn here because if acute, the degree of patient discomfort and disability far exceeds that which might be expected for any otherwise rather trivial lesion.

Objectives

The study evaluated the outcome of lateral internal sphincterotomy as compared to manual anal dilatation as a basis for future practice.

Anal fissure can be classified into two: acute and chronic fissure. It can occur at any age, but is usually a condition of young adults. Sex distribution is males to females is 1:1, however, women are much more likely to develop this condition than men. Fissures in ano can occur anteriorly at 12 o'clock or posterior at 6 O'clock. They are commonly found in the midline, this is because of the elliptical arrangement of the external sphincter, which is felt to provide less support to the anal canal in the antero-posterior axis thus rendering this location most susceptible to trauma. Blood supply, which is already tenuous, may be further compromised by compression and contusion as the branch of the inferior rectal artery passes through the internal anal sphincters.

Clinical diagnosis of fissure in ano is made from history and physical examination.

Setting

A prospective study carried out at Kenyatta National Hospital wards 5A, 5B and 5D.

Methods and patients

In the study period of nine months, from June 2004 to February 2005, both months inclusive, we sampled seventy-eight patients of whom forty underwent lateral internal sphincterotomy and thirty-eight underwent manual anal dilatation (Lords procedure).

Procedures were carried out under spinal or general anaesthesia. Early complications noted were such as bleeding and haematoma formation. These were noted immediately after surgery and up to 24 hrs post surgery. Late complications were noted from the third day, and these were such as incontinence of flatus, incontinence of stool, pruritus ani, and abscess formation.

Results

Fifty six percent of the patients who underwent manual anal dilatation developed stool incontinence while none developed this with lateral internal sphincterotomy. Eighty four point eight percent of those who underwent manual anal dilatation developed incontinence of flatus. While 15.2% of those who underwent lateral internal sphincterotomy developed incontinence of flatus. Eighty percent developed pruritus ani in manual anal dilatation while 20% developed this in lateral internal sphincterotomy. Abscess formation was seen in 92% of those who underwent manual anal dilatation while 8% of this was seen in lateral internal sphincterotomy. On discharge the patients were reviewed and the early complications resolved. Late complications were seen up to four weeks post surgery these resolved with time and conservative management. This tells us that complications were temporary.

Those who underwent lateral internal sphincterotomy had a short duration of stay in hospital while those who underwent manual anal dilatation had a longer duration of stay for the complications to be treated. Recurrence was found to be more in manual anal dilatation than lateral internal internal sphincterotomy whereby of those who underwent MAD, 14 (36.8%) had recurrence whereas in LIS 8 (20%) had recurrence.

The study showed that more men suffered than women to the ratio of 1.66:1. More complications were seen with manual anal dilatation than lateral internal sphincterotomy therefore patients were forced to stay longer in hospital for the complications to be treated. Since our patients came to us after two months (chronic fissures) conservative management had no role.

Conclusion

Therefore, lateral internal sphincterotomy remains the attractive option for many patients suffering from this painful condition.

INTRODUCTION

Chronic anal fissure is characterised by severe pain after defecation and often fresh anal bleeding. Physical examination reveals a tear in the anal canal. Most patients with chronic anal fissures have a high resting anal pressure, which impairs blood flow through the anal sphincters to the anal mucosa and prevents healing.

The primary aim of treatment of anal fissure is to reduce pressure generated by the anal sphincter mechanism to improve blood flow and allow mucosal healing. Due to the complications seen in our SOPC clinic following manual anal dilatation, done from the periphery and some units in KNH we were prompted to study the two modalities.

Lateral internal sphincterotomy being the conventional method for the treatment of anal fissure as compared to manual anal dilatation, the aim of the study was to evaluate the outcome of the two modalities as a basis for future practice.

LITERATURE REVIEW

According to Antropoli, the oldest proctologist, pathologies of the anal canal are extremely common. About 30 – 40% of the population suffers from proctological pathologies at least once in their lives. Anal fissure was recognised as a clinical entity in 1934. It is a longitudinal defect of the anal canal mucosa and anoderm extending usually from the dentate line to external verge of the anal canal. This defect exposes the lower half or even most of the fibres of internal anal sphincter. Anal fissure is almost always accompanied by extensive tension of these muscles. Anal fissure affects all age groups but predominantly occur in the third and fourth decades of life (1). The longitudinal tear in the squamous epithelium of the anal canal, frequently precipitated by the passage of hard stools, though in small proportions of patients, it may follow an episode of diarrhoea (2). Majority of patients have been mis-diagnosed to have haemorrhoids and in the real sense have fissure in ano. HIV/AIDS presents with wounds around the anal region and the stigma of the disease has made many keep away due to the embarrassment that may be caused on them, therefore they present late. (2)

AETIOLOGY

Fissures can be classified as primary or secondary depending on the cause.

The cause of idiopathic fissures is not known, however several theories have been postulated and some of these are: -

- 1) The **mechanical forces** imposed on the anal canal during the passage of stool. Hard stools are most commonly implicated, but forceful liquid stool can also produce the same results. (1,3)
- 2) **Tears** in the anus caused by foreign bodies such as instrumentation or undigested bone spicules or any other hard foreign body. (1,3)
- 3) **Laxatives** – habitual users of this may also develop fissures. There is fairly liquid stool for a long time leading to tightly fibrosed anal canal. If this patient passes hard stool the skin splits leading to fissures in ano. This is also due to the spasms of the internal sphincter muscles. (1,3)
- 4) **Childbirth** – A number of women end up with fissures especially anterior fissures following complicated childbirth. (1,3)

- 5) **Fissures** in ano may be secondary to other anal diseases such as fistulas in ano, proctitis as well as fibrous anal polyps. (1,3)

These diseases may also complicate surgery such as laying out of a fistula or haemorrhoidectomy. It may also follow pruritis ani. Not forgetting worm infestations such as Trichuris, Enterobius vermicularis that lay ova around the anal opening (4).

- 6) The **anatomy** of the anal canal plays a role in the aetiology of fissures in ano. This depends on the arrangement of the anal muscles. Lockhart –Mummary felt that the external sphincter structure plays a role in causing fissures. The lower portion of this muscle fibres is not truly circular, but consists of a band of muscles fibres that pass from posterior to anterior and split around the anus. He postulated that the anal mucosa is best supported laterally and is weakest posteriorly and therefore more fissures occur posteriorly (5).

- 7) **Blood supply.**

Klosterhalfen and colleagues did angiographies on cadavers to visualise the inferior rectal artery using vascular injection, they showed that the posterior commissure is less perfused than the other areas of the anal canal, hence, ischaemia may be an important aetiological factor in causing anal fissures in the posterior location. (5)

8) **Secondary causes** of fissures in ano are seen in patients with syphilis and other sexually transmitted diseases, tuberculosis, leukaemia, inflammatory bowel disease such as Crohn's disease, and HIV. (6)

In this secondary fissures, treatment or management will be directed to the primary causes as above. (6)

Physiology of regulation of internal sphincter tone

There are three main influences of internal sphincter tone and function reflecting the sphincter specialization of this muscle (7). The first is intrinsic myogenic tone. This depends on the extra cellular calcium levels entering via L-type calcium channels (7).

The second influence is the enteric nervous system also known as the third division of the autonomic nervous system. This pathway is located in the Auerbach and Meissner's plexi in the wall of the gut and responsible for peristalsis as well as local reflexes such as the rectoanal inhibitory reflex. The nerves are known to be non-adrenergic, non-cholinergic because neither guanethidine nor atropine block their activity, yet tetrodotoxin does. The neurotransmitter has been identified as nitric oxide. This relaxes the internal sphincter, an action blocked by N-nitro-L-arginine.

The third influence is the autonomic nervous system which affects contraction and relaxation of the internal sphincter via sympathetic and para sympathetic post ganglionic fibres respectively. They act directly on the smooth muscles and indirectly on the nerves of the enteric nervous system or both. The sympathetic neurotransmitter noradrenaline contracts the internal sphincter via its action on alpha-receptors, an action blocked by the alpha antagonist phentolamine. (8)

Physiological studies

Researchers have been interested in anorectal pressure studies in patients with anal fissures.

a) Duthies and Benneth Measured sphincter pressure, with an open-ended tube connected to a recording device by a sterile gauze (9).

b) Miles and Stewart have also shown that the posterior angulation of the rectum leads to uneven straining at the margins of the anal opening during defecation. (9)

Therefore during defecation the pressure of the hard faecal mass is mainly on the posterior anorectal angle in which event the overlying epithelium is greatly stretched, and being relatively unsupported by muscles, remains an area of fissures formation.

All patients demonstrated spasms of the sphincter on the digital rectal examination, but no increase in resting pressure in the control subjects. Following sphincter stretch, a moderate fall in pressure was noted but it returned virtually to normal by the eighth post-operative day (9).

- c) Gibbons and Read employed perfusion probes of varying diameters in patients with chronic anal fissures. Resting pressure was elevated in all subjects when compared to the controls. He therefore concluded that resting pressure was elevated in patients with fissures in ano (8).
- d) Nothmann and Schuster performed ballon rectosphincter manometry on patients with anal fissure. Resting pressures were twice as high as those measured in control subjects.

Following distention of the rectum by the ballon, there is the expected internal sphincter relaxation, but this is followed by a marked and prolonged contraction above the initial baseline termed the “ overshoot” phenomenon. Therefore they concluded that this reflexly stimulated sphincter spasm and so involved in the aetiology of the condition (9).

ANAL ANATOMY

The anal canal commences at the level where the rectum passes through the pelvic diaphragm and ends at the anal verge. The muscular junction between the rectum and anal canal can be felt with the finger as a thickened ridge – the anorectal bundle or “ring”. The internal sphincter is a thickened continuation of the circular muscle coat of the rectum. (1,2) The involuntary muscle commences where the rectum passes through the pelvic diaphragm and ends at the anal orifice where its border can be felt. The internal anal sphincter is 2.5 cm long and 2-5mm thick. When exposed during life it is pearly white in colour and its individual transversely placed fibres can be seen clearly. Spasm and contracture of this muscle play a major part in fissure and other anal affections. (1,2)

The longitudinal muscle is a continuation of the longitudinal muscle coat of the rectum, intermingled with fibres from the pubo rectalis. Its fibres fan out through the lowest part of the external sphincter, to be inserted into the true anal and peri-anal skin. The longitudinal muscle that are attached to the epithelium provides pathway for the spread of perianal infections, and mark out tight compartment that are responsible for the intense pressure and pain that accompany many localised perianal lesions. Beneath the anal skin lie the space of the corrugator cutti ani muscle.

The external sphincter has its fibres attached posteriorly to the coccyx, while anteriorly they are inserted into the mid perineal point in the male, whereas in the female they fuse with the sphincter vaginae. (1,2)

Between the internal (involuntary) sphincter and the external (voluntary) sphincter muscle mass is found a potential space, the inter-sphincteric plane. The plane contains the eight to twelve apocrine glands which can cause infection and it is also a route for the spread of pus. It can also be opened by a surgeon to provide access for operation of the sphincter muscle. (1,2)

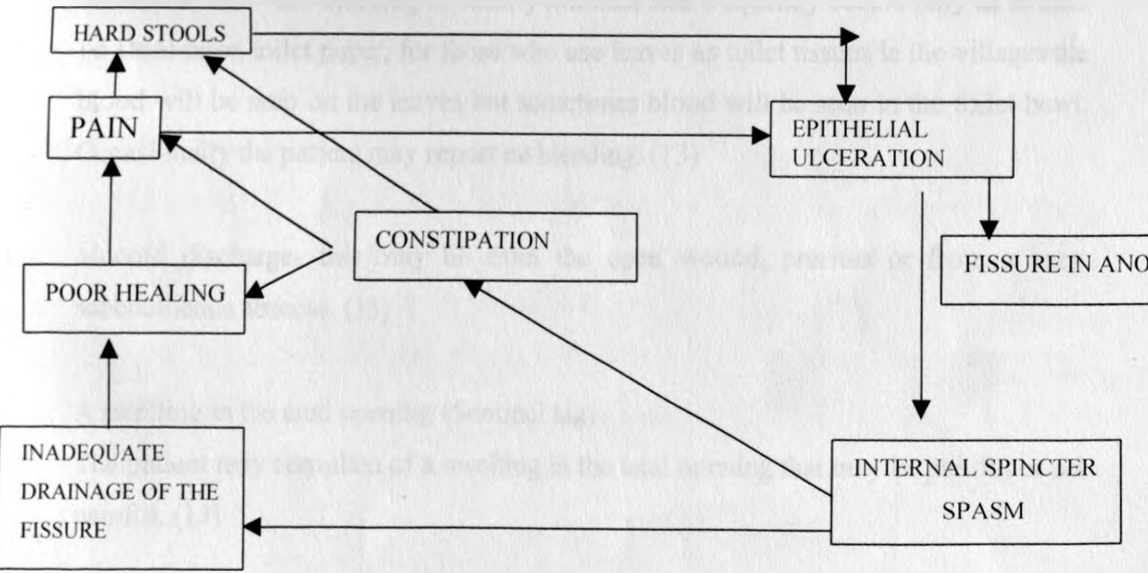
The mucous membrane – the pink epithelium lining the rectum extends through the anorectal ring into the surgical anal canal (1, 2).

Pathology

Acute anal fissures are not normally associated with skin tag formation. Chronic anal fissures are associated with the development of anal tags as a result of inflammatory oedema. The ulcer in chronic anal fissure is cone shaped and at the inferior extreme there is a tag of skin, usually oedematous. This tag is known as a sentinel pile. Sentinel because it guards the fissure. A vicious cycle ensues which in the sub-epithelial inflammation causes spasms of the internal sphincter inhibiting free drainage of the infected fissure and permitting continued inflammation, resulting in a small, chronic inadequately drained abscess. The reflex relaxation of the internal sphincter that normally precedes defecation is lost in patients with fissures in ano. and instead contraction of the internal sphincter occurs. (3).

Diagram as illustrated by the flow chart

Vicious cycle as seen in fissures in ano (3)



Due to the contraction of the internal sphincter and pain sensation, the patient develops the fear to defaecate and this leads to constipation, Stool will be hard and so stretching of the fissure occurs tearing the epithelium more and so more pain. (1,2,3)

Once there is a crack in the epithelium and then a break in continuity of the mucus membrane, the anal canal being a place with micro organisms, infection occurs, and this leads to the chronicity of the fissures. There is repeated injury by the hard stools also. With time the fissures heal with fibrosis and so anal stenosis, may occur. (10)

The “sentinel pile” a hypertrophied anal papilla seen in the majority of chronic anal fissures, results from oedema with contributions made by a combination of infection, lymphostasis, surface irritation by discharge from fissures and squeezing up effect produced by a tight sphincter. (11)

The undermining of the sentinel tag occurs with abscess formation which then drains in the anal canal after rupture due to the tight sphincter and then complicates into a subcutaneous fistula (12).

Clinical features.

From history the patients will complain of the following: -

- 1). Anal pain, usually described as burning or tearing
The pain usually occurs with and immediately after defecation. Usually pain ceases in a few minutes but occasionally it may persist for hours. The pain is also related to bowel movements. (13)
- 2). Bloody stools: - the bleeding is usually minimal and frequently occurs only as streaks on stool or on toilet paper, for those who use leaves as toilet tissues in the villages the blood will be seen on the leaves but sometimes blood will be seen in the toilet bowl. Occasionally the patient may report no bleeding. (13)
- 3). Mucoid discharge- this may be from the open wound, pruritus or from a burst subcutaneous abscess. (13)
- 4). A swelling in the anal opening (Sentinel tag)
The patient may complain of a swelling in the anal opening that may be painful or not painful. (13)

- 5). Others: - They may complain of problems with micturation (retention, urgency, frequency) and dyspareunia due to perinial muscular spasms. Dyspareunia or problems with micturation may occur in both acute and chronic fissures. Loss of appetite due to failure to defecate, constipation and pain may be reported. (13)

Constipation is the antecedent event, but once pain develops the fear of the act of defaecation and refusal to the call to stool can exacerbate tears in the anal mucosa.

This anxiety leads to faecal impactions particularly in children and the elderly. (13)

Physical exam

Patient is placed in left lateral decubitus position with knees drawn up towards the chest.

Inspection is done, of the anal opening, by gentle retraction of the glutea. Due to pain the anal sphincter muscles may be in spasms, therefore digital rectal examination is discouraged unless a local anaesthetic is used where the depth of fissure can be accessed and its orientation to the midline often described using clock orientation of the hour hand. (13)

Palpation may demonstrate a spastic anal sphincter or tight anal canal and will exacerbate patient's discomfort. There may be no fibrosis in the acute phase as the tear is superficial. (13)

Anoscopic examination will confirm the location of the fissures.

Acute fissures are seen to be erythematous and bleed easily.

With chronic fissures, classic fissures triad may be seen (13).

- a) Deep ulcer
- b) Sentinal pile, which forms when base of fissures becomes oedematous and hypertrophic
- c) Enlarged anal papillae. This can be mistaken for tumours. (13)

In chronic cases anoscopy can be accomplished, as pain in these cases is not very severe.

Proctosigmoidoscopy may be performed to rule out the possibility of a tumour or distal inflammatory bowel disease. (13)

Occasionally the base of the ulcer may form an abscess that leads to a fistula. The fistulas are superficial and usually 1-2cm distal to the skin tag. A probe always can go through the fistula but the internal anal sphincter is not traversed. (13)

In patients with severe pain, rectal examination may be carried out under general anaesthesia to rule out other anal pathologies such as haemohroids, fistula and tumours. (13)

Diagnosis

- 1). Patient history and physical examination will often strongly indicate the correct diagnosis
- 2). Endoscopic examination is helpful to exclude higher pathology in the rectum but may be impossible to perform adequately at the initial stages due to discomfort and pain. (3)

Differential diagnosis

The fissures in ano can be differentiated from diseases such as:

Perianal abscess: may mimic a fistula. (14)

Pruritus ani: may have mucosal splits of skin and mucous membrane secondary to irritation from the discharge. (14).

Thrombosed haemorrhoids: with this condition the patient feels a lump in the anal opening. (14)

Proctalgia fugax: the discomfort in this case is not related to bowel action, also the patient feels the discomfort high up and more deeply-seated, while that of the fissures in ano is subcutaneous. (14)

Anal haematoma: may follow surgery of haemorrhoids or trauma in the anal region. (14)

Bartholin's gland abscess in females: could resemble an anterior fissures. (14)

Tuberculous ulcer: it is small in the beginning, but grow with time with undermined margins. (14)

Tumours: these are tumours such as squamous cell carcinoma of the anus or adenocarcinoma of the rectum that invades the anal canal and anal opening. With this other symptoms may be found such as inguinal lymph nodes and a huge mass in the rectum. (14)

Syphilitic chancre: the most characteristic feature here is a symmetrical lesion on the opposite wall of the anal canal. Diagnosis is made by presence of spirochaetes found in the discharge from the wound by dark ground illumination or by the Wassermann reaction. (14)

Crohn's disease: usually has multiple lesions and the diagnosis is always made on sigmoidoscopy, which reveals multiple lesions in the rectum. (15)

Ulcerative colitis is also a differential. Colonoscopy could be done to confirm its diagnosis. (15)

Leukaemia: usually the fissures are off the expected location and are multiple. A diagnosis is by white cell count and a blood film examination. (15)

HIV infection and AIDS: there are multiple wounds in the anal region. (15)

Herpes Simplex: a viral disease affecting the mucous membrane and skin, and can cause fissures. (15)

Hidradenitis suppurativa: infection of the hair follicles around the anal opening. These produce a lot of fluid/ pus. (16)

MANAGEMENT

An anal fissure is a very painful condition, which needs prompt and adequate treatment. This will rid the patient off the agony of pain and restore comfort. (3)

Vicious cycle of the disease has to be broken to inhibit the contraction of the internal sphincters by stopping the spasms and then allowing the free drainage of the fissure or the subcutaneous abscess that may form and so allowing the healing process to take place. One should also take care of constipation to reduce the rate of repeated trauma by hard stools.

Epithelization of the fissure occurs leading to complete closure.

Complications such as fistula formation and sinus formation are therefore prevented.

Conservative

The Conservative or medical management is mostly for acute fissures. It breaks the vicious cycle and allows for the healing to occur. This includes:

The use of the **W.A.S.H.** regime in the treatment of anal fissures.

W --- Warm water shower or Sitz baths after bowel movements.

This is done two to three times in a day using warm salt water. It cleans the fissure and is used also as an antiseptic. It also has a soothing effect in the wound and reduces oedema. (3)

A --- Analgesics (3)

S --- Stool softeners. Such as diotyl sodium sulfosuccinate have been used. Bulking agents such as psyllium (Fiberall, Meta Mucil, Idonsyl) have been used. In Kenyatta National hospital we commonly use dulcolax and sennakot (3)

H --- High Fibre diet – should be encouraged to these patients especially in the initial stages when the symptoms begin, this is to avoid constipation. (3)

Following the above procedure most of the uncomplicated fissures resolve in 3-5 weeks. A chronic fissure may need frequent treatment.

Anaesthetic agents have been used. They only relieve the pain and relax the sphincters, making it possible also for one to examine the patients with very minimal pain. Lignocaine has been used; Anusol and proctosedyl have also been used. One can use them as suppositories or gels. Long acting anaesthetic agents have been discarded. It is aimed at blocking the inferior rectal nerve. (16)

Injections of botulinum toxin are being used. Botulinum toxin is an exotoxin produced by bacteria clostridium botulinum and is a potent neuro toxin that causes botulinism (5). The injections given produce a reduction in the maximum resting anal pressure, which can be sustained for two to three months and this should translate to improved healing. The research done by Giuseppe et al, randomly sampled 50 patients. Injection of 20units of botulinum toxin into the internal anal sphincter of each sides of anterior midline was done twice daily. Botulinum toxin causes denervation of the internal sphincters. It acts faster and prevents the release of acetylcholine by presynaptic nerve terminals. (17) Paralysis occurs within a few hours and the transmission of neuromuscular impulses resumes after the growth of new axon terminals. (17)

Topical application of glyceryl trinitrate ointment twice daily for 8 weeks, can be effected. Glyceryl trinitrate 0.2% ointment provides rapid sustained relief of pain in patients with chronic fissures in ano. It reduces the maximum resting anal pressure and improves pain by increasing the anodermal blood flow as well as reduction in maximum anal pressure. It has side effects such as headaches and burning sensation around the anal opening. All this side effects are short lived. The glyceryl trinitrate ointment breaks down to form Nitrous Oxide a vasodilator, which relieves spasms. This sustains relieve of spasms and the fissure heals. However this needs follow up of patient for a long time. (18)

Isosorbide dinitrate has been used as a gel or a spray applied around the anal opening. It increases the blood flow to the anal sphincter and so improves spasms. Isosorbide dinitrate pharmacological activity is attributed to its active metabolite isosorbide mononitrite. It has side effects such as headaches (7).

Calcium channel blockers such as diltiazem have been tried as a form of chemical sphincterotomy. The drugs used in this class are such as oral nifedipine 20mg twice daily. It reduces maximum resting pressure and so sustaining the healing process. It causes headaches and flushes. This are short lived (18).

Topical diltiazem 2% gel has proved to be effective with less side effects. It demonstrated more profound reduction in maximum resting pressure and therefore better healing process than glyceryl trinitrate.

Antibiotic should be prescribed to patients with fever and discharge indicating infection. (18)

Operative Management

Surgical therapy of anal fissure is reserved for patients who have failed medical therapy, or have developed a fissure-fistula. Most of the methods here involve the disruption of the internal anal sphincter. The operative methods or techniques commonly used for anal fissure include

- (1) Anal stretch well known as manual anal dilation (MAD). (19)
- (2) Open Lateral internal sphincterotomy. (19)
- (3) Closed lateral internal sphincterotomy. (19)
- (4) Tailored sphincterotomy
- (5) Posterior midline sphincterotomy + Fissurectomy. (19)
- (6) Dermal flap coverage of the fissure. (19)

Anal Stretch – M.A.D.

Anal stretch was originally described by Recamier in 1838 for the treatment of proctalgia fugax and anal fissure. (5) Such a procedure can be carried out with a local anaesthetic infiltration, but a brief general anaesthetic is preferable. Patient is placed in the lithotomy position and draped. An index finger of one hand is inserted into the rectum followed by the index finger of the opposite hand. The long finger of the same hand is inserted followed by the long finger of the opposite hand. Gentle lateral retraction with each finger commences for approximately 30 seconds. With four fingers in place, the anal canal stretched cautiously for four minutes. In men, it is easier to stretch the sphincter in the anteroposterior plain because of the narrowness of the pelvic outlet. Sphincter stretch in women should be done transversely as the disruption of the anterior sphincteric support is a real possibility. (20)

The disadvantages of the M.A.D. includes the following:

- (1) With this procedure fissure may be widened and can end up bleeding more. Widening the fissure, more micro organisms may invade the wound and septicaemia can occur. (20)

- (2) M.A.D may cause incontinence of flatus and occasionally faeces, due to interference of the complete closure of the anal opening that leaves a small hole defect due to rupture of the external sphincter fibres. (20)
- (3) Recurrence of the fissure is very common after M.A.D. (20)
- (4) A haematoma may form after M.A.D. (20)
- (5) Subcutaneous anal abscess may form latter especially when the microorganism invade the raw widened fissure after MAD. (20)

Open Lateral Internal Sphincterotomy

The first person to perform this operation was Brodie in 1839. With the pretext that when the sphincter goes into spasm the anal pressure goes up and this makes the fissure worse: (21) Hilton in 1863 gave credit to the operation due to its success. Miles believed that he was dividing what he called the band in his operation and so mistook this for the internal anal Sphincter. (21)

Eisenhammer in 1951 had to work on cadavers and then went ahead to do proper internal anal sphincterotomy by dividing the right muscles. Internal anal sphincter is the continuation of the distal portion of the circular muscles of the rectum. There is a groove in between the internal anal sphincter and the external sphincter that lies lateral. (21)

Internal anal sphincter maintains the anal canal in the closed position, action is involuntary. External sphincter is a striated muscle voluntary. The procedure is done under general anaesthesia with the patient in lithotomy position, a groove between the internal and external sphincters is palpated. An incision is made above the internal sphincter either on the left or right lateral position then with the use of a curved mosquito forceps (Halsted). It is brought out from the incision and cut by use of a knife or diathermy. The wound may be left open and patient goes on saline sitz baths twice a day. (21)

Patients may also be put in a prone Jack-knife position depending on the surgeons preference and availability of appropriate table and the buttocks strapped laterally to expose the anal region. An incision is made on the left or right and latter can be sutured with absorbable sutures. (21)

Post operatively the patient goes on analgesics, saline sitz baths. Time taken for these patients to heal is about six weeks. Complications of open lateral internal sphincterotomy are such as:

1. Ecchymosis around the entrance wound if the closed technique is used. (22)

2. A massive haematoma is usually the result of failure to apply enough pressure to the site. (22)
3. Haemorrhage may occur especially if a blood vessel is severed. (22)
4. Perianal abscess occurs in 1% of the internal anal sphincterotomies these may lead to fistula and anal discharge. (23)
5. Very rarely does incontinence of flatus and faeces occur.

Fissurectomy and midline sphincterotomy

Fissurectomy is used in chronic anal fissure. It involves the excision of the fissure in a triangular manner and removes the fibrotic tissues this will enable the wound to re-epithelialize and so heal. This method was tried long time back by Gabriel in 1948. This operation had many disadvantages, which included the following: - (24)

1. The wound left after the fissurectomy was very large and took too long to heal.
2. The anal opening is a dirty area and so during defecation the wound being raw is contaminated by organisms, which may pass to the bloodstream.
3. The condition, with this method may become worse with reduced chances of healing and may even form a subcutaneous abscess at the site of excision.
4. The patient is fed on fluid diet for not less than seven days and started on stool softeners. This is to allow the healing process to take place. (24)
5. Fistula may form after the operation. (24)
6. Keyhole deformity is another troublesome consequence of the excision. The result may produce symptoms of mucus discharge, pruritus, and soiling of the undergarments.

This method, because of the disadvantages is now obsolete.

Closed lateral internal sphincterotomy:

Closed lateral internal sphincterotomy almost the same as open lateral internal sphincterotomy, and is more recent. (25) It was first tried by Park in 1967 and supported by his colleague Sohn and others. (25)

The method does not need bowel preparation although is necessary. It can be done in a clinic under local anaesthesia, with the patient in a jack knife position or lithotomy position.

General anaesthesia is recommended because all muscles are relaxed with patient in lithotomy position. A finger is put in the anal canal. Internal sphincter palpation is done and the junction between it and the external sphincter delineated. (25)

A blade is passed between the mucosa and internal sphincter up to the junction of the upper third and lower two thirds of the internal sphincter (Dentate line), and then rotated laterally cutting the internal muscle with manual assistance. (25)

Pressure is then applied for two minutes over the mucosa to stop haemorrhage. Sofratule on gauze may be inserted in the opening to stop haemorrhage and removed later when the patient is awake. Patients later go home with stool softeners, analgesics and instructed to have daily sitz baths.

Sentinel tags or piles, oedematous tissue of skin above the fissure should be excised to allow drainage of the fissure.

The advantages in this case outweigh the disadvantages in that:

1. The healing is faster (26)
2. The discomfort is not there post operatively and the anal pressure falls immediately. (26)
3. Incontinence is a rare entity in this case as documented by Callopy and Ryan in 1978. They found out that a very small percentage of patients were incontinent. (26)

The disadvantages of the method include:

1. Haematoma formation at the site (26)
2. Injury to nerves and major vessels as it is done in a closed manner. (26)
3. One may not be sure to have done complete division of the muscle (26)
4. There may be severe bleeding especially when a vessel is severed. (26)

Tailored sphincterotomy

This procedure differs from standard lateral internal sphincterotomy in that the division of the sphincter is more conservative and is carried out cephalad only for the length of the fissure rather than to the dentate line. Flatus and solid incontinent rate are very low and recurrence has not been reported and (7).

Dermal flap coverage of the fissure:

Dermal flap coverage is a new method in proctology. (27). A flap is rotated from the skin side taking care not to sever any of the sphincters and rotate it to the fissure after freshening the edges and suture. The hygiene is of paramount importance here. Rotated flaps carries its own blood supply. (28)

Disadvantage of the method is that the failure rate may be high due to hindered drainage from the flap and also abscess formation below the flap leading to failure of the flap to take. Flap failure is attributed to the increased pressure that is not relieved. (28,29)

AIM OR MAIN OBJECTIVE

The aim of the study was to evaluate the outcome of lateral internal sphincterotomy as compared to manual anal dilatation as a basis for future practice.

SPECIFIC OBJECTIVES

1. To compare the outcomes of the procedures done (Manual Anal Dilatation and Lateral Internal Sphincterotomy).
2. To determine the length of hospital stay after lateral internal sphincterotomy and manual anal dilation.
3. To determine complication rates between the two methods.

JUSTIFICATION/ RATIONALE OF THE STUDY

Anal fissure is a common anorectal condition. It is associated with spasms of the internal anal sphincter. Several methods both surgical and pharmacological exist in the management of fissures. Comparison of the two surgical methods (lateral internal sphincterotomy and manual anal dilatation) will be of importance so as to show the better modality of treating this condition. The study in this region is an attempt to shift from manual anal dilatation to lateral internal sphincterotomy as a mode of fissure management. A similar study was done at Kenyatta National Hospital on retrospective basis 13 years ago and viewed fissures in ano treatment in general. Today there is need to update ourselves in the current status of fissure management.

METHODS AND PATIENTS

Study design and setting

This was a nine months (June 2004 – February 2005) hospital based prospective study comparing lateral internal sphincterotomy to manual anal dilatation. The research was conducted at KNH surgery department, the main outcomes of interest were duration of symptoms, complications, other previous interventions, duration of hospital stay, sex distributions, location of the fissure, distribution of the disease as per age group, recurrence of the disease post surgery. And lastly, complications that arose due to the surgery, not forgetting other local and systemic diseases that are associated with the disease. Patients were admitted through SOPC to either ward 5A, %B or 5D. Those who did not meet eligibility as per the criteria of inclusion were not recruited into the study, all patients were clerked, examined by the investigator and a diagnosis of fissure in ano was confirmed.

Consent was taken from them as per the questionnaire. They were all investigated and prepared for surgery by taking blood for urea and electrolytes and creatinine. Haemoglobin levels were also done. The procedure was explained to the patients before surgery. Choice of the procedure was selected by the consultants surgeon assisted by the investigator. The choice of the anaesthesia depended on the availability of the drugs. The scrub nurse set the instrument and brought them for the surgeon and the investigator to use depending on the procedure. Soap enemas were given on the morning of surgery including stool softeners (dulcolax) prescribed 48 – 72 hours prior to surgery.

After anaesthesia patients were put in lithotomy position, cleaned and draped. For MAD the goal being to reduce sphincter tone by controlled manual stretching of the internal sphincter. After insertion of the right index finger, the left index finger followed. Both the two were held in position for thirty seconds. These were followed by the right long finger and the then the left long finger. Eventually four fingers were in position and held for thirty seconds to one minute. This led to lateral destruction of the anal sphincter. In women this was done transversely. The anal canal was compressed with sufratule and a small piece of gauze. For LIS after draping the patient, the fissure was identified and a lateral incision was made. A halsted artery forcep was used to bring out the sphincter in the wound, which was subsequently divided either by use of diathermy or knife. One stitch was placed on the wound and one stitch applied. Compression was done with sufratule and gauze. Patient was reversed for further observation in the ward by the nurses and the investigator. After the procedure the anal opening was compressed with sufratule and a small piece of gauze. Post operatively all patients were started on pethedin 50mg and declofenac 50mg as start doses to

continue with oral analgesics. Prophylactic and biotics were prescribed for 48 hours. In lateral internal sphincterotomy the wound was closed with one stitch before compression. Saline sitz baths were commenced from day two, twice a day and after bowels. Surgical complications, visual analogue pain score (1-10) during hospital stay were recorded. At day one, two weeks and four weeks post treatment all patients were reviewed for fissure healing and complications. The pain was assessed by visual analogue without asking the patients then later asked how they were feeling. They were asked how they felt how much analgesic they were taking.

These was recorded against the procedures that were carried out on them as cured, better, same, pain and worse. Once stable with no complaints they were discharged off the clinic after four weeks. The decision to discharge them was done by the consultant surgeon of the unit and the investigator. The files were all marked and kept aside for further scrutiny.

Acute fissure was defined as a superficial ulcer in the anoderm with sharply demarcated edges with a duration of less than six weeks while chronic fissure was defined as benign indurated ulcer with undermined edges with visible internal sphincter fibres. The duration of symptoms was more than six weeks. Chronic fissures were associated with skin tag. Healing was defined as complete disappearance of clinical symptoms and re-epithelization of the anal canal mucosa.

Study area

Patients were sampled at random according to ward admissions. The study groups were patients who meet eligibility in wards 5A, 5B and 5D – Kenyatta National hospital.

To undergo lateral internal sphincterotomy or manual anal dilatation the eligibility criteria was:

- i) A patient diagnosed to have fissure in ano and granting an informed consent to participate in the study.
- ii) Free from complications such as haemorrhage or sepsis

CRITERIA OF EXCLUSION FROM THE STUDY

- Paediatric patients were excluded from the study
- Those seen earlier in our surgical outpatient clinic and surgical wards with a diagnosis of fissures in ano before the study began were left out.
- Patients without proper and convincing information about the diagnosis and had other anal diseases at the initial examination stages were left out of the study.
- Those with pre existing faecal incontinence were excluded.
- Those with previous anal sphincter surgery were left out.

CRITERIA OF INCLUSION INTO THE STUDY

- 1) All patients who were seen during the period of the study with fissures in ano
- 2) Had no other bowel diseases at the initial examination.

LIMITATIONS OF THE STUDY

The study was conducted at Kenyatta National Hospital over a period of nine months (June 2004 – February 2005). Procedures were carried out under spinal or general anaesthesia. Limiting factors were such as admission criteria where the patients had to pay a mandatory deposit which some felt was high. Long waiting queues before surgery was also a limiting factor. The study was carried out at the time when Mbagathi Hospital was opened as a District Hospital, so some patients were directed there making it difficult for us to reach them. During counselling, some patients felt that we could be investigating them for AIDS and so refused to enter into the study. This was a single centre study and so the catchments area was limited. Due to the economic hardships some patients did not come to the clinic for review as they were coming from outside Nairobi.

$$n = \frac{\left\{ Z_{1-\alpha/2} \sqrt{2P(1-P)} + Z_{1-\beta} \sqrt{P_1(1-P_1) + P_2(1-P_2)} \right\}^2}{P_1 - P_2}$$

Where; n = sample size to be determined

Z = standard errors from mean ($Z_{1-\alpha/2} = 1.96$)

α = level of significance ($Z_{1-\alpha} = 1.645$)

β = level of significance <80 = 0.842, >90 = 1.282

P_1 = proportion developing complications with method 1 (MAD)

P_2 = proportion developing complications with method 2 (Lateral internal

Sphincterectomy)

d = absolute precision (5% or 0.05 as a proportion)

Assumption value MAD = 50% } Complications arising as per previous studies.

Calculated value LIS = 20% }

Therefore complications of MAD are more than LIS.

ETHICAL CONSIDERATIONS:

1. Informed consent was granted by the patient to participate in the study. Nobody was otherwise included in the study
2. Strict confidentiality was ensured to safeguard the privacy of the patient.
3. The information gathered was used for the disclosed purpose of the study only and for no other reason.
4. Data entry was by code numbers and not by names
5. Approval to conduct the study was sought from the research and ethics committee of Kenyatta National Hospital and the study only begun once this was granted. This was on 31st May 2004.

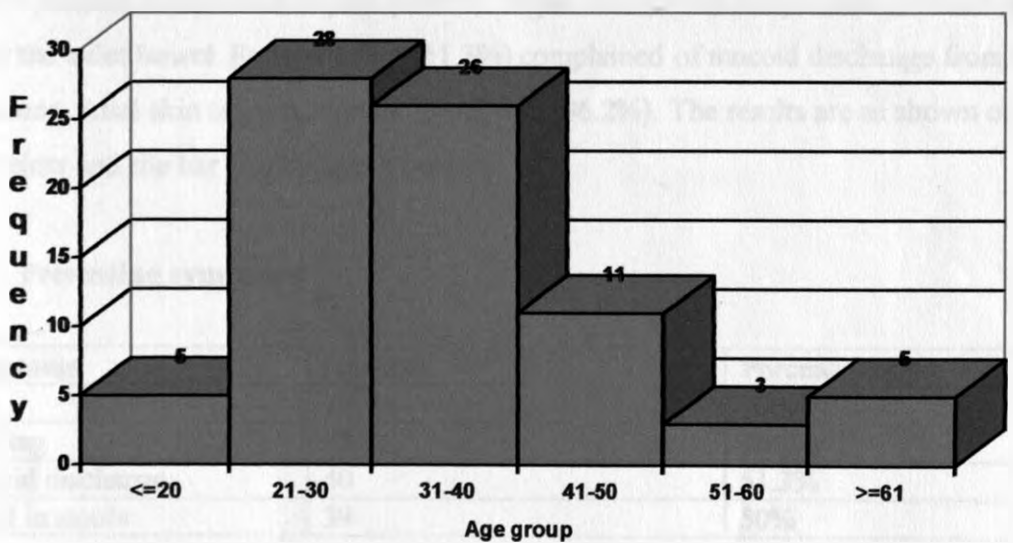
RESULTS

In this study a total of 78 patients were seen with a diagnosis of fissure in ano. Of these 78 patients 40 underwent lateral internal sphincterotomy while 38 underwent manual anal dilatation (Lords procedure). Patients were operated on by a consultant surgeon of the said unit assisted by the investigator

Age distribution

The age distribution of the patients who underwent the procedures are shown in figure 1 below. The youngest patient was 17 years old and the oldest was 82 years. The highest number of patients were in 21 – 40 age group (56 (68.2%)) The mode was 28 years found in ages 20 – 30 years.

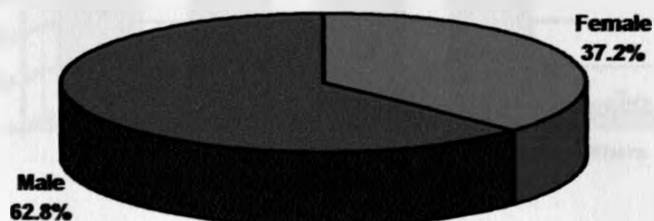
Figure 1: Age distribution



Sex distribution

Forty-nine patients were males (62.8%) while 29 were females (37.2%) as shown in figure 2 below. The ratio of male to female was 1.66:1

Figure 2: Sex distribution



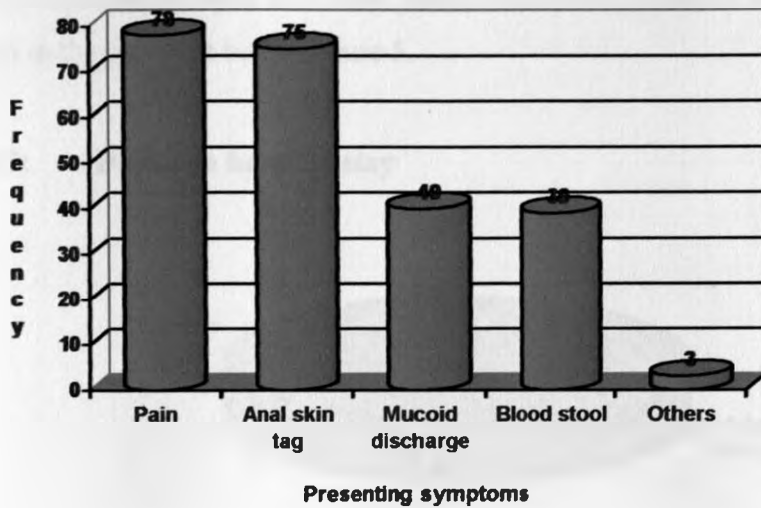
Presenting symptoms

All the 78 patients complained of pain (100%). 39 patients (50%) complained of blood on stools or the toilet bowl. Forty patients (51.3%) complained of mucoid discharge from the anal opening. Anal skin tag was seen in 75 patients (96.2%). The results are as shown on table 1 below and the bar chart figure 3 below.

Table 1. Presenting symptoms

| Symptoms | Number | Percentage % |
|------------------|--------|--------------|
| Pain | 78 | 100% |
| Anal tag | 75 | 96.2% |
| Mucoid discharge | 40 | 51.3% |
| Blood in stools | 39 | 50% |

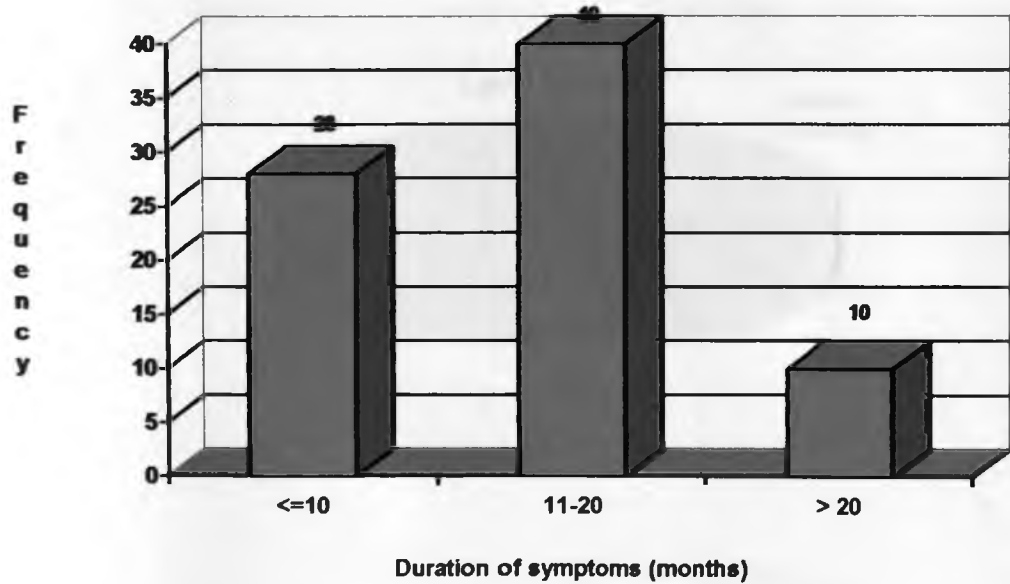
Figure 3: Presenting symptoms



Duration of symptoms prior to presentation to hospital

Patients took long before presenting themselves to hospital. The shortest duration was seen in one patient (2 months). The longest duration was seen in two patients (36 months) The mode was 40 patients seen between 11 and 20 months. The mean was 12.6 months. The results are as shown in figure 4 below

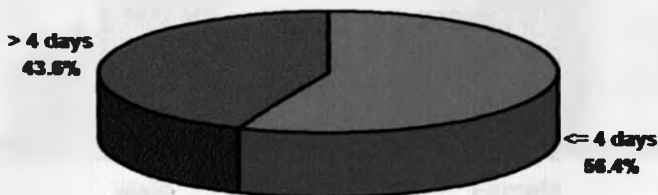
Figure 4: Duration of symptoms prior to presentation in hospital



Duration of hospital stay (grouped)

In the study group of 78 patients who underwent the procedures (MAD, LIS), 44 (56.4%) stayed for less than or equal to 4 days. Thirty-four (43.6%) stayed for more than 4 days. This is shown in the pie chart below figure 5.

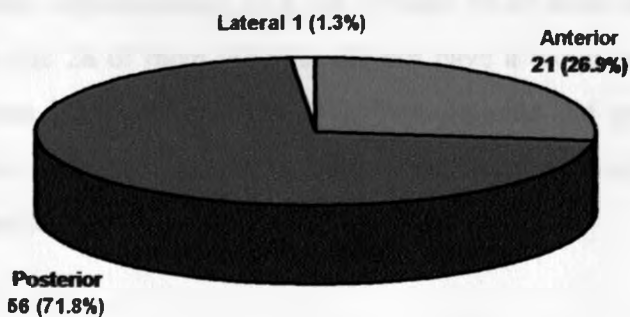
Figure 5: Duration hospital stay



Location of fissures

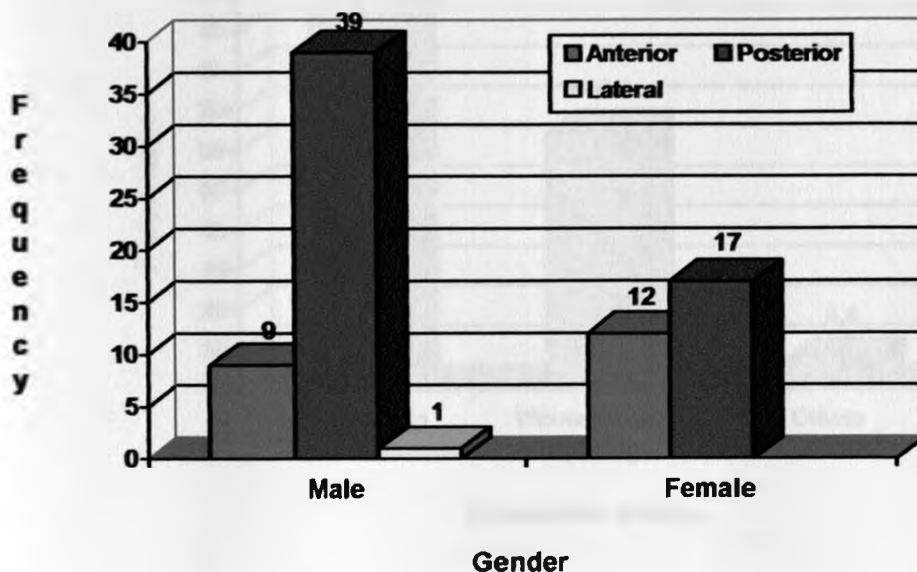
Fifty six patients were found to have fissures posteriorly placed (71.8%) while 21 patients (26.9%) had them anteriorly placed. One patient had fissure laterally placed (1.3%) the results are as shown below on the pie chart figure 6.

Figure 6: Location of the fissure



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Figure 7: Location of fissures by gender

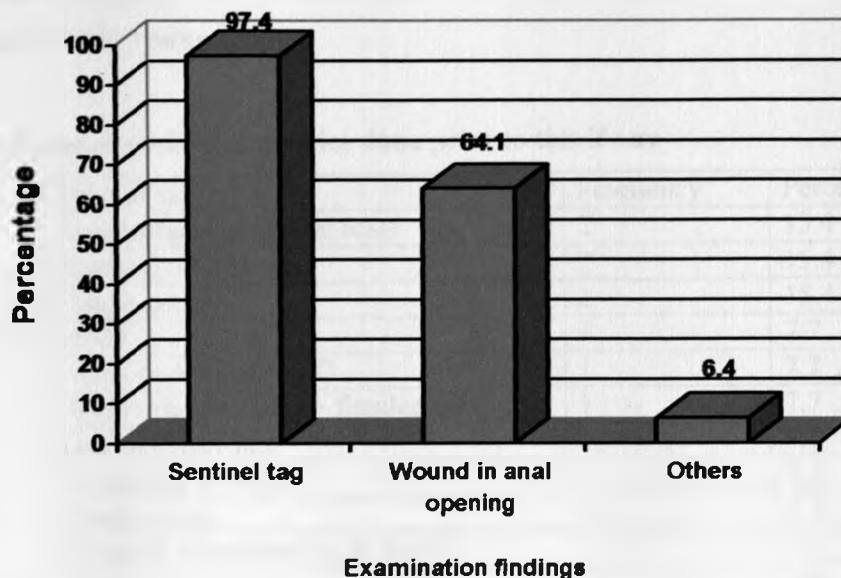


in the study group of the 78 patients, nine males had fissures anteriorly placed (11.5%) while 12 females had them anteriorly placed (15.4%). Thirty-nine males (50.0%) had them posteriorly placed while 17 females (21.8%) had them posteriorly placed. One male (1.3%) had the fissure laterally placed, the results are shown on the bar chart figure 7.

Examination findings

Seventy eight patients were reviewed, and out of these 50 of them (64.1%) had a wound in the anal opening, while 28 of them (35.9%) did not have a wound on the anal opening. A sentinel tag was seen in 76 patients (97.4%). Two patients did not have a sentinel tag. Induration was seen in one patient, haemorrhoids were found in two patient. Other swellings were found in one patient. This is shown on the bar chart figure 8.

Figure 8: Examination findings



Associated diseases to anal fissures

Seventy eight patients who were seen, of whom, 9 of them had haemorrhoids associated (11.6%). Fistula in ano was seen in one patient (1.3%), anal warts in one patient (1.3%), Vaginal tears at delivery in one patient (1.3%). These results are tabulated in table 2.

Table 2: Associated diseases to anal fissures

| Primary disease | Frequency | Percent |
|--------------------------|-----------|--------------|
| Haemorrhoids | 9 | 11.6% |
| Anal warts | 2 | 2.6% |
| Fistula in ano | 2 | 2.6% |
| Vaginal tear at delivery | 1 | 1.3% |
| Total | 14 | 18.1% |

Types of perineal surgeries done prior to this study

Fissure in ano may also follow some perineal surgeries as a complication. This has been shown on the table 3 below.

Table 3: Types of perineal surgeries done prior to this if any

| Type of surgery | Frequency | Percent |
|----------------------------------|-----------|---------|
| Accidental or perennial tears | 2 | 15.4 |
| Haemorrhoidectomy | 2 | 15.4 |
| Fistulectomy | 2 | 15.4 |
| MAD | 1 | 7.7 |
| Warts excision + MAD | 1 | 7.7 |
| Haemorrhoidectomy + fistulectomy | 1 | 7.7 |
| Tear of suture line | 1 | 7.7 |
| Ressection of warts | 1 | 7.7 |
| Fissurectomy | 1 | 7.7 |
| Removal of sentinal tag & MAD | 1 | 7.7 |
| Total | 13 | 100 |

TYPES OF TREATMENTS

Conservative methods used – Post Surgery

Of the patients seen seventy-one (91%) were treated on the W.A.S.H. regime. Forty-nine of them were treated with anaesthetic suppositories. The results are tabulated on table 4.

Table 4: Conservative methods used

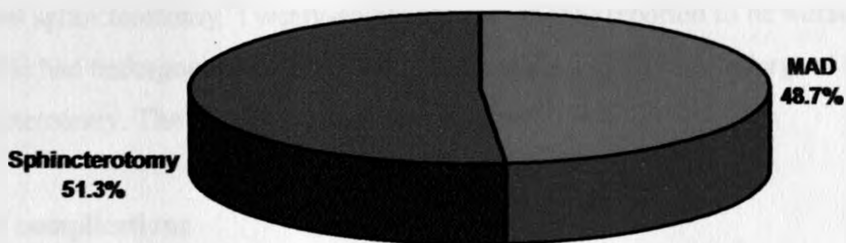
| Conservative methods used | Frequency | Percent |
|------------------------------|-----------|---------|
| WASH | 71 | 91.0 |
| Anaesthetics (Suppositories) | 49 | 62.8 |

Some of the patients underwent both conservative methods of WASH and Anaesthetics Suppositories simultaneously.

Operative methods used

The author reviewed 78 patients and out of this, 40 (51.3%) underwent lateral internal sphincterotomy while 38 patients (48.7) underwent manual anal dilatation. The results are as shown on the pie chart figure 9.

Figure 9: Operative modalities



Operative methods used versus duration of hospital stay

Seven patients (18.9%) who underwent manual anal dilatation stayed for less than four days while thirty-one (81.1%) stayed for more than four days. Thirty-seven (90%) underwent lateral internal sphincterotomy stayed for less than four days while three stayed for more than four days. The results are as shown below in table 5.

Table 5: Operative methods used versus duration of hospital stay

| | Duration of hospital stay | | Total |
|----------------|---------------------------|------------|-----------|
| | <= 4 days | > 4 days | |
| MAD | 7 (18.9%) | 30 (81.1%) | 37 (100%) |
| Sphincterotomy | 37 (90.0%) | 4 (10.0%) | 41 (100%) |
| Total | 44 (56.4%) | 34 (43.6%) | 78 (100%) |

Operative methods used versus outcome

Seventy eight patients were reviewed and of these, 41 (52.6%) reported cured or better. Five (13.2%) had undergone manual anal dilatation while 36 had undergone lateral internal sphincterotomy. Nine patients (11.5%) reported no change. Seven (18.4%) from the nine who reported no change had undergone manual anal dilatation while 2 (5%) had undergone lateral internal sphincterotomy. Twenty-eight patients (35.9%) reported to be worse. Of these, 26 (68.4%) had undergone manual anal dilatation while 2 (5%) had undergone lateral internal sphincterotomy. The results are as shown in table 10.

Early complications

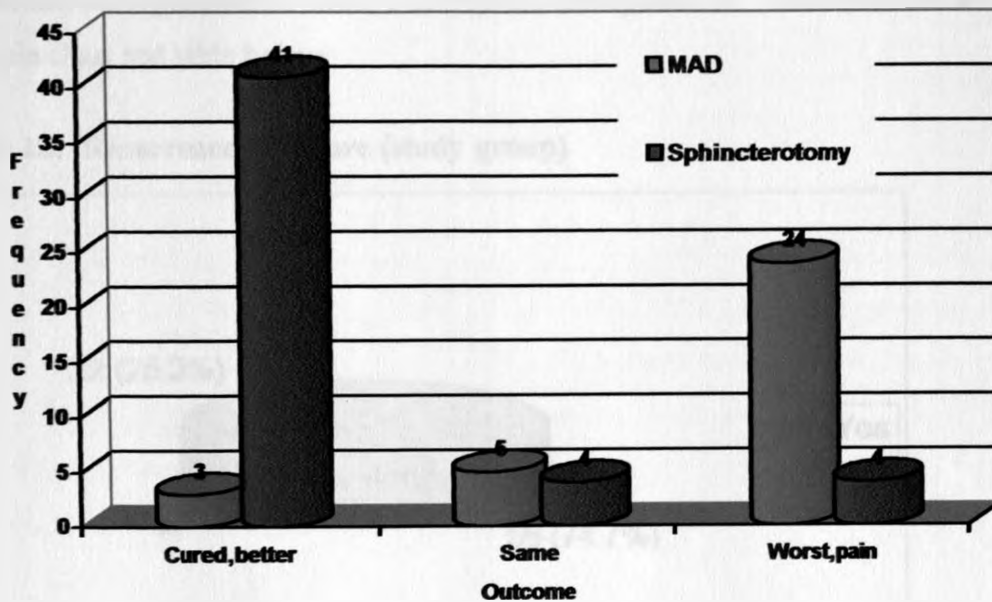
Eighteen patients from the 38 who underwent MAD had minimal bleeding to warrant change of the pack three times in 24 hours. At any one time the author used one rytec gauze. 20 did not bleed.

Forty patients underwent LIS, of this 3 bleed. Therefore the total number of patients who bleed were 21. Three patients who underwent MAD developed haematoma and five who underwent LIS developed haematoma totalling to eight. The results are as tabulated in table 6.

Table 6: Early complications

| Complication | MAD | LIS | Total |
|--------------|------------|----------|------------|
| Bleeding | 18 (23.1%) | 3 (3.8%) | 21 (26.9%) |
| Haematoma | 3 (3.9%) | 5 (6.4%) | 8 (10.3%) |

Figure 10: Operative method used versus outcome



Complications at four weeks follow up, patients with complications (n=46)

Forty-six patients developed complications such as incontinence of flatus, abscess formation, incontinence of stool and discharge (pruritus ani). In the study group that underwent manual anal dilatation had 28 patients (75.7%) with incontinence of flatus, 23 patients (62.2%) with abscess formation 21 patients (56.8%) with incontinence of stool and sixteen patients (32.2%) with discharge (pruritus ani). Complications which were seen in those patients who underwent lateral internal sphincterotomy were as mentioned above. Five patients (55.6%) had incontinence of flatus, two patients (22.2%) had abscess formation non had incontinence of stool but four patients (44.4%) had discharge. Thirty two patients out of the study group of 78 did not have complications.

The results are illustrated in table 7.

Table 7: Comparison of complications at four weeks follow up (n=46)

| Complication | MAD | LIS | Total |
|--------------------------|------------|-----------|------------|
| Incontinence of flatus | 28 (75.7%) | 5 (55.6%) | 33 (71.7%) |
| Abscess formation | 23 (62.2%) | 2 (22.2%) | 25 (54.3%) |
| Incontinence of stool | 21 (56.8%) | 0 (0.0%) | 21 (45.7%) |
| Discharge (Pruritus ani) | 16 (32.2%) | 4 (44.4%) | 20 (43.3%) |

Seventy-eight patients were seen, and out of these 22 (25.3%) had recurrences, 56 patients (74.7%) did not have recurrences. Twenty-two patients had recurrence and out of these, 14 (36.8%) had undergone MAD while 8 (20%) had undergone LIS. The results are as shown on the pie chart and table below.

Figure 12: Recurrence of fissure (study group)

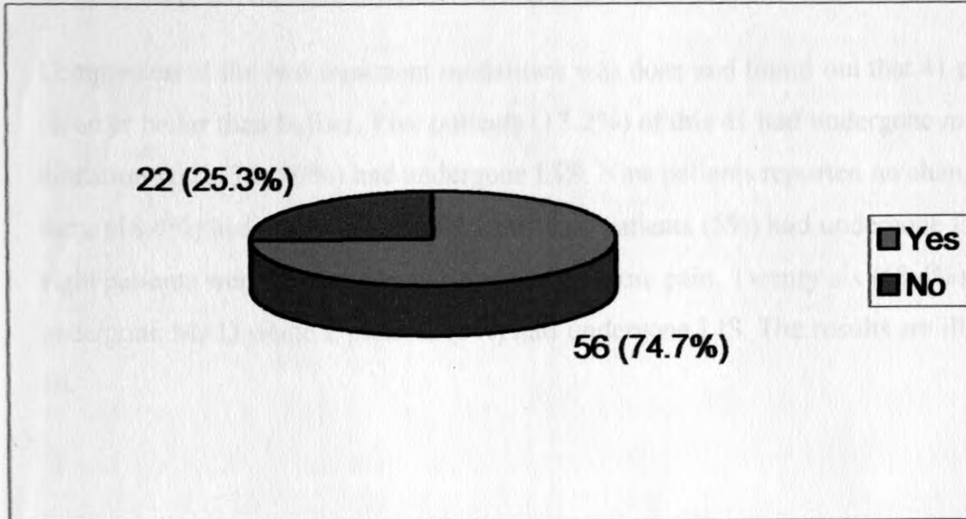


Table 8: Recurrence: MAD & LIS compared

| | MAD | LIS | Total |
|------------|------------|------------|--------------|
| YES | 14 (36.8%) | 8 (20%) | 22 (25.3%) |
| NO | 24 (63.2%) | 32 (80%) | 56 (74.7%) |
| | 38 (100%) | 40 (100%) | 78 (100%) |

OUTCOME

In the study group of 78 patients 25(32.1%) reported that they were cured. Sixteen (20.9%) reported that they were better than before, Nine patients did not notice any change while four (5.1%) felt a lot of pain. Twenty four patients (30.7%) indicated that they were worse. The results are illustrated in table 9.

Table 9: Outcome at four weeks follow up (n=78)

| Outcome | Frequency | Percent |
|---------|-----------|---------|
| Cured | 25 | 32.1 |
| Better | 16 | 20.9 |
| Same | 9 | 11.5 |
| Pain | 4 | 5.1 |
| Worse | 24 | 30.7 |
| Total | 78 | 100 |

Comparison of the two treatment modalities was done and found out that 41 patients were cured or better than before. Five patients (13.2%) of this 41 had undergone manual anal dilatation while 36 (90%) had undergone LIS. Nine patients reported no change, seven of these (18.4%) had undergone MAD while two patients (5%) had undergone LIS. Twenty eight patients were worse than before and felt more pain. Twenty six (68.4%) of them had undergone MAD while 2 patients (5%) had undergone LIS. The results are illustrated in table 10.

Table 10: Outcome at four weeks follow up versus operative methods

| | Operative methods used | | Total |
|--------------|------------------------|----------------|------------|
| | MAD | Sphincterotomy | |
| Cure, better | 5 (13.2%) | 36 (90%) | 41 (52.6%) |
| Same | 7 (18.4%) | 2 (5%) | 9 (11.5%) |
| Worst, pain | 26 (68.4%) | 2 (5%) | 28 (35.9%) |
| Total | 38 (100%) | 40 (100%) | 78 (100%) |

DISCUSSION

Anal fissure is one of the main proctological disorders encountered in general surgical practice. It may be wrongly diagnosed as haemorrhoids and perianal fistula. Despite the lesion's small size can cause great discomfort and pain which sometimes becomes incapacitating. Spasms of the anal sphincters have been noted in association with anal fissures, and for many years the aim of the treatment has been to reduce hypertonia of the sphincters. Operation techniques commonly used for fissure in ano include, anal stretch, open lateral internal sphincterotomy, closed lateral internal sphincterotomy, posterior midline sphincterotomy and to a lesser extent dermal flap coverage of fissure (27). In our study we compared two methods, which were gentle dilatation of the anal opening and open lateral internal sphincterotomy. Both methods work by reducing resting anal pressure to normal. As occurred in our series anal fissure is reported to affect more males than females. From studies done by Parklea in 1992 showed that the incidence of this disease in our SOP Clinic was 2.6: 1000 in Kenyatta National Hospital (14).

In our study we had 62.8% men and 37.2% women, this when calculated tells us that anal fissure affects more men than women in our country i.e. M: F 1.6887: 1, this contradicts the studies done in European clinics and Asian clinic where the ratio is 1: 1 (14)

Marvin L. Corman also noted that men and women are equally affected to the ratios of 1:1 (15). Studies done in Asia have also yielded the same results. The most recent studies done in Madrid (2004) tallies with our results well. They found that 65% were men while 35% were women (16). The condition is very common in the second and third decade, dropping slightly in the fourth decade. The mean age is 34.92 yrs of the patients who were seen, while the median is 32.00 yrs. The mode was found to be 24.00 years of age. According to studies done in our hospital in 1992 it was noticed that the average age was 29 years. During the study more males were seen than females. In our study, the predominating group are males of 34 years and females between 28-30 years. (15)

From our results, it is clear that the majority of patients are between 21 yrs of age and 40 yrs of age. It is less common below 24 yrs of age and above 50 yrs of age. From studies done in America, the age bracket was 20 yrs, but with our people, the age bracket was from 30 yrs onwards. (2)

The Asian community has an age bracket of 35 years. (15) With the condition being seen more in the 4th decade. It is rare in the elderly and children.

The possible explanations for the disease being rare in the young people and the aged is not very clear, but a few theories may be formulated.

The author may say that, in the old it is rare because of the muscular atony that develops with age.

Working in hot climatic conditions makes our people more dehydrated and so leaves the stools very hard to injure the anal mucosa.

Anal pain and anal skin tag are the commonest symptoms, others are such as mucus and blood in stools. This tallies well with other studies done by the Europeans. Anal pain in our study was seen in 100% of patients while anal tag was seen in 96% of patients. This tallies well with the European studies of 94% and 84% respectively. (15) Blood on stools was seen in 50% of patients while mucoïd stools were seen in 51.3% of patients. In the Caucasians population, these are only reported to occur. Pruritus ani, appetite loss and other discharges are less frequently seen. In our setup, patients come in chronic stages and so these are seen. (15)

The studies done in the west have yielded the same results like ours, pain being the main symptom seen in all the patients, anal tag seen in all of them as well. This is because of the healing process of epithelization. (16)

Our patients take too long before they are seen in our clinics. On average they take upto 12 months. The shortest duration seen in our clinic was 2 months while the longest was 36 months. The majority presented to the clinic after 12 months. This was found to be 33.3% of the patients. This long duration of symptoms makes the whole process chronic. Pain and bleeding are the main symptoms but pain is not as severe as that with acute fissure. Frequently the patients symptoms are attributed to secondary changes such as the presence of a lump in the anal opening (23). The mean duration of symptoms from other researches done in Spain was 17.6 months (range 1-70) The possible explanations why the patients presented late could be:

Many of them presented to other health institutions, treated for different anal diseases before they got to us.

The perineum is an area where people may not want to mention to be sick and so patients take long before coming to hospital. Before the onset of HIV/ Aids the condition was there but people had the reluctance of talking about ones own anus and this led to delay in diagnosis. (14)

The long appointments also extend the duration.

Many patients have known that perinial ulcers are a presentation of HIV/ AIDS and so many will stay away in the fear that they may be screened for the same since it is still a stigma in our community.

Majority of the patients who were seen had fissures posteriorly. Fifty Six of the patients (71.8%) (39 males and 17 females) had fissure posteriorly. These outcomes have also been seen in Europe and North America where studies have been done^(1,3,11). In studies done in Madrid, they found that 65% men and 35% women had fissures posteriorly while 60% women and 39% men had them anteriorly while 1-2% of the population had them laterally, both men and women alike (20).

For fissures to heal, the vicious cycle must be broken, this happens very rarely – only in acute cases.

The vicious cycle as above can only be broken if one is constantly on treatment, but this is not the case here. Some people do not follow the instructions. For the patients using suppositories, occasionally may have no money to buy the drugs. So the problem may persist. Comparison was made between two methods: manual anal dilatation and lateral internal sphincterotomy. The author looked at the outcome of the procedures, the advantages of lateral internal sphincterotomy and manual anal dilatation as well as the complication rates between the two methods. The length of stay of the patients in hospital after the procedure was also analysed by grouping them into two as per their duration of stay in hospital post surgery. We grouped our patients into two, those who stayed longer than four days after surgery and those who stayed for less than four days. Patients who underwent manual anal dilatation seven (18.3%) stayed for less than four days while 30(81.1%) stayed for more than four days. This is costly to the patients and a lot of time is wasted in a hospital bed. In lateral internal sphincterotomy, thirty-seven (90.0%) stayed for less than four days while four (10%) stayed for more than four days. Complication rates in manual anal dilatation were more than in lateral internal sphincterotomy. Incontinence of flatus and stool, abscess formation, discharge (pruritus ani) were some of the complications. Others were such as bleeding from the tears, fibrous formations and anal stenosis and haematoma formation post procedure.

This tallies well with the western literature. Those who underwent lateral internal sphincterotomy improved well, termed as epithalization of the fissure and absence of symptoms as before (20). Recurrence rate in manual anal dilatation was also found to be higher than in lateral internal sphincterotomy (20).

In manual anal dilatation the patients complicated as above and so they were to be observed longer and the complications treated. While in lateral internal sphincterotomy the complications were few. Others did not complicate.

Majority of the patients who were done manual anal dilatation complained of a haematoma formation and anal abscesses. This were sorted out and led to the long stay in hospital

Some patients who underwent manual anal dilatation developed severe bleeding. This was because of the atony of the sphincter due to the dilatation. In lateral internal Sphincterotomy there were none who bled. This has been shown in the Western literature (29).

Incontinence of stool was seen in 71% of the patients who underwent manual anal dilatation, as well as incontinence of flatus. This depressed the patients so they needed counselling and further treatment of the complications.

Review of the patient after four weeks follow up reflected that more complications were seen in patients who had undergone manual anal dilatation than lateral internal sphincterotomy. Results tally well with the Western Literature that more complications are seen in manual anal dilatation

Those Patients who developed anal stenosis had undergone manual anal dilatation and so the wounds created during the procedure healed with fibrosis leading to Stenosis. This is clear in the Western Literature (30,31).

CONCLUSION

- 1) The incidence of fissures in ano should be higher than what we have found in our study. This is because of the short duration of the study.
- 2) Our study shows that the number of men with fissures is higher than that of women. This is because more men were seen with the condition during our study than the females. The ratio of male to female was 1.66:1.
- 3) Our patients present themselves to a surgeon very late and in the chronic stage, which does not respond to conservative management.
- 4) Most of the patients are labelled to have haemorrhoids: this is because of the old belief that any pain and bleeding around the anal opening are due to haemorrhoids. Misdiagnosis has played a bigger role here. Some clinicians do not examine their patients properly especially when it is an acute fissure and with a lot of oedema and pain.
- 5) Some perineal surgeries lead to anal fissures especially haemorrhoidectomy when not well done and the wound heals leaving some areas bare then end up as fissures.
- 6) Complications are seen to be more rampant with manual anal dilatation than lateral internal sphincterotomy. These are such as incontinence of flatus and stool, Abscess formation and pruritis ani.
- 7) Recurrence was found to be more with manual anal dilatation than open lateral internal sphincterotomy.

- 8) Our operative management from the study is more superior now to the conservative management because many of them come in chronic stages. Lateral internal sphincterotomy then is the superior method.
- 9) Surgery can be done under spinal anaesthesia or local anaesthesia as per the western literature.
- 10) Conservative management cannot work in chronic anal fissures. It can only work in acute fissures because the vicious cycle can easily be broken in acute cases than chronic cases.

RECOMMENDATIONS

After a long struggle to find out the best operative method, I highly recommend that:-

- 1) Lateral internal sphincterotomy should be the method to be adopted in our set up and around the country as it gives the best results ie: shortest hospital stay, less complications, can be carried out in a clinic under local anaesthesia and as a day case.
- 2) To avoid recurrence, we recommend that before surgery, our patients are given a health education talk about their disease, the surgery to be done, the diet, post surgery care and when they should start taking their daily duties.
- 3) The author recommends that the simplest anaesthetic methods be used such as local anaesthesia and spinal anaesthesia. This will reduce the morbidity of the patients. This also reduces the expenses and time that the patient will stay in hospital occupying a hospital bed.
- 4) The anatomy of the anal region is of importance to understand fissures in ano, this should be taught at both district and provincial levels before one can start the procedure of lateral internal sphincterotomy. Doctors at internship should also be taught this method.
- 5) We advice that research on this painful condition be done at other levels countrywide. This will give us a proper picture of fissure in ano in the country. Our study only looked at fissures at Kenyatta National Hospital
- 6) Publications of literature about fissures in ano to the districts and provincial levels about the new modes of management.

QUESTIONNAIRE

1. Patients Personal details

- Name
- Age:
- Weight:
- Sex:
- DOA:
- DOD:

2. Presenting Symptoms

- Pain in the anal area
- Blood stool
- Mucoïd discharge
- Anal skin tag
- Others

3. Duration of symptoms prior to presentation in hospital

4. Examination findings

- Wound in the anal opening
- Sentinel tag
- Others

5. Location of the fissure

- Anterior
- Posterior
- Others

6. Any primary diseases associated with anal fissure:

.....
.....
.....

7. Types of perineal surgeries done prior to this if any:

.....
.....
.....

8. Complications at least four weeks follow up outcome (cured, better, same, worst, pain, ulceration)

- Incontinence of flatus

- Incontinence of stools
- Discharge (Pruritus ani)
- Abscess formation
- Others

9. Recurrence formation Yes No

10. Operative methods used

- MAD
- Sphincterotomy

11. Conservative methods used

- W.A.S.H
- Anaesthetics (Suppositories)

I do hereby acknowledge that I have been given adequate information about the purpose of the study. I have understood its importance and benefit to the medical personnel and the general public. I am also informed that the participation in or withdrawal from the study will not result in discrimination against me in terms of treatment and care. I am further informed that the information obtained shall be treated with confidentiality. Refusal to sign this consent will not interfere with your management.

Signed _____

Witness _____

Patient/ Guardian

Investigator/ Assistant

Date _____

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Date: 31 May 2004

Dr. Daniel O Alushula
Dept. of Surgery
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Dear Dr. Alushula

RESEARCH PROPOSAL "FISSURES IN ANO PROPOSAL FOR A PROSPECTIVE STUDY AT KENYATTA NATIONAL HOSPITAL" (P122/10/2003)

This is to inform you that the Kenyatta National Hospital Ethics and Research Committee has reviewed and **approved** the revised version of your above cited research proposal for the period 31 May 2004 – 30 May 2005. You will be required to request for a renewal of the approval if you intend to continue with the study beyond the deadline given.

On behalf of the Committee, I wish you fruitful research and look forward to receiving a summary of the research findings upon completion of the study.

This information will form part of database that will be consulted in future when processing related research study so as to minimize chances of study duplication.

Yours sincerely,

PROF. A N GUANTAI
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Cc Prof. K M Bhatt, Chairperson, KNH-ERC
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The Dean, Faculty of Medicine, UON
The Chairman, Dept. of Surgery, UON
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Supervisor: Prof. P G Jani, Dept. of Surgery, UON

IMPACT OF ADHERENCE ON SEVERITY OF ENDOGENOUS ECZEMA IN PATIENTS AT KENYATTA NATIONAL HOSPITAL.

Dr. David Ndonge Musyoka, MBChB

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DECLARATION

I certify that this is my own original work and has not been presented for a degree at any other university.



Dr David Ndonge Musyoka.MB,ChB(University of Nairobi)

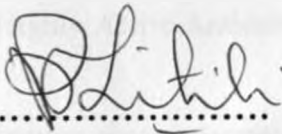
This dissertation has been submitted with our approval as supervisors:

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ABBREVIATIONS

ACD.....Allergic contact dermatitis

ADAtopic dermatitis

AZT.....Zidovudine.

EASI.....Eczema area severity index

ECRC..... Ethics committee and research committee

HAART.....Highly Active Antiretroviral Therapy.

ICD..... Irritant contact dermatitis

IGA..... Investigation Global Assessment

KNH.....Kenyatta National Hospital

MEMS..... Medication event monitoring system.

OSAAD Objective severity assessment of atopic dermatitis

PN..... Prurigo nodularis.

POEM..... Patient oriented eczema measure

QALY.....Quality of life

RRRelative risk

SASSAD..... Six area, six sign atopic dermatitis

SCORAD..... Scoring atopic dermatitis

U.O.N.....University of Nairobi

USA..... States of America.

3TC..... Lamivudine

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I dedicate this book to my parents.Nicodemus Katithi and Esther Nduku who have always reminded me that everything is possible with hard work.

ABSTRACT

Background

Eczema is a common dermatological condition and is associated with high morbidity, exposure to costly drugs with adverse side effects, and negative psychological impact on those affected. However, it is often inadequately controlled leading to symptomatic eczema, in clinical practice a prominent reason for this being poor patient adherence to prescribed therapy.

There is no published data on severity of endogenous eczema, impact of adherence on severity of eczema, and the underlying reasons for non-adherence in patients in KNH.

Objective:

The study was designed to investigate the impact of adherence on severity of endogenous eczema, and identify factors responsible for non-adherence in patients with eczema.

Methods.

This was a comparative cross-sectional descriptive study, patients with eczema were recruited then assessed for severity of eczema, adherence was also assessed. Quantitative methods were used to analyze severity of eczema and level of adherence to eczema therapy and qualitative methods were used to analyze the patient reasons for non-adherence. Eczema severity was assessed by use of the scorad index; adherence was assessed by use of adherence questionnaire and self reports. Qualitative analysis of in-depth interviews with non-adherent patients was carried out to find out the reasons for non-adherence.

Results

Ninety patients who participated in the study were found to have symptomatic eczema. Fifty-four (60%) patients had mild eczema, twenty-eight (31.1%) had moderate and eight (8.89%) had severe eczema. Severe endogenous eczema was significantly associated with non adherence ($r^2=8.5, p=0.010$).

Seventy-three (81.1%) patients were adherence to the prescribed medication. Male gender and low socioeconomic status were significantly associated with non-adherence ($p=0.04$) and ($p=0.01$) respectively, other socio-demographic factors did not significantly affect adherence.

The reasons for non-adherence were; worsening of symptoms, cost of drugs, drug burden and drug side effects.

Conclusion

There is inadequate eczema control in our population since majority of patients had symptomatic eczema, due to non-adherence and other factors. Reasons for non-adherence were identified and solutions to these problems should be adopted in programmes to improve adherence to enhance eczema control.

1.0 LITERATURE REVIEW

INTRODUCTION

ECZEMA

“Eczema” is derived from a Greek word which means “boiling” which implies that the skin can become acutely inflamed that fluid weeps out or vesicles form on the skin. Eczema is synonymous with dermatitis and the two words can be used interchangeably. Eczema is inflammation of the skin due to diverse etiologies, characterized histologically by spongiosis and clinically by exudation, crusting and thickening of the skin and often accompanied by itching or burning(1). The etiology of endogenous eczema is unknown but it has a polygenic mode of inheritance and is multifactorially determined particularly by environmental factors(2). The prevalence of atopic dermatitis(AD) has been increasing over the years in western countries and current estimates suggest that 9-12% of children have AD while the prevalence of AD general population is less than 10%(3)

In Africa few studies have been carried out prevalence's of 3.5%, 6.1% and even 17% have been recorded (4, 5). In the developed world eczema accounts for a large proportion of skin diseases both in hospital based populations and community-based populations. It is estimated that 10% of the population have eczema, 40% of the population experience an episode of eczema in their lifetime.

European study that was questionnaire based, prevalence of AD was 15.6% with some regional differences. Girls more often had flexural eczema and outnumbered boys in a ratio of 1.3:1 Boys more often had a personal history of asthma, whereas girls more often had a family history of asthma(6).

The international study of Asthma and Allergies in childhood (ISAAC) Steering Committee, described worldwide variation in prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and atopic eczema(6). In the U.S.A 17.2% of childhood population have AD. 15.6% prevalence in European children(7), 24% prevalence in 5-6 year old Japanese children and 6 –19 % in preschool children in Germany(8). The pathogenesis of AD is still not clearly

defined but its major diagnostic features include pruritus, typical morphology and distribution of the rash, chronicity and atopic personal or family history, while most of the minor criteria include generalized xerosis, ichthyosis, raised serum IgE, increased skin microbes, vasomotor instability, impaired cell mediated itch on sweating, intolerance to food, wool and stress, conjunctivitis, facial pallor and pityriasis alba.

1.1 TYPES OF ECZEMA

There are different types of eczema. AD will be described in more details being the commonest across all ages.

Wüthrich in 1989 proposed the division into two forms, namely; 'intrinsic' and 'extrinsic' atopic dermatitis: the former without any IgE-mediated reaction, the latter with the presence of an IgE-mediated reaction. According to Wüthrich,(9) the intrinsic form was present in about one-quarter of the cases.

Endogenous eczema(10).

1. Atopic (dermatitis occurring in a person with personal or family history of asthma, allergic rhinitis)
2. Seborrhoeic dermatitis (dermatitis seen in areas rich in sebaceous glands; usually after puberty)
3. Asteatotic eczema (eczema associated with decrease in skin surface oils; common in the elderly)
4. Gravitational eczema (eczema associated with varicose veins; seen around the ankles)
5. Hand eczema (most common eczema; causes can be many)
6. Forefoot eczema (common eczema seen in childhood, involving predominantly the forefoot)
7. Pityriasis alba (dry, white patches seen on the face and sometimes elsewhere; occurs during childhood)
8. Metabolic eczema (eczema due to nutritional deficiencies of , zinc, niacin, proteins, etc)
9. Drug eruptions (exfoliative dermatitis)

Exogenous eczema(11)

1. Irritant dermatitis.
2. Allergic dermatitis
3. Photo-sensitive dermatitis
3. Polymorphic light eruption.
5. Infective dermatitis

All types of eczema have the clinical spectrum of acute to chronic. In acute eczema, vesicles and bullae appear if inflammation is intense, in subacute eczema the skin is erythematous, dry and flaky, edematous and crusted (if secondarily infected). In chronic eczema it is characterized by thickening of the epidermis "acanthosis".

Eczema is nearly always itchy. Histological "eczematous change" refers to a collection of fluid between the epidermis keratinocytes "spongiosis" and the upper dermal perivascular infiltrate of lymphohistiocytic cells.

Treatment.

1. Education and explanation on eczema
2. Avoid known irritants.
3. Avoid getting too warm
4. Topical therapies, combination of steroid and emollients.
5. Adjunct therapies, oral antibiotics, sedating antihistamines, bandaging, systemic steroid in severe cases.
6. Phototherapy.
7. Immunosuppressants, cyclosporine, calcineurin inhibitors.

Unjustified fear about the dangers of topical steroids has often led to under treatment of eczema. Providing appropriate-strength steroid preparations are used for the right body site, these compounds can be used quite safely on a long-term intermittent basis(10).

Second-line agents

These may be considered in severe non-responsive cases, especially if the eczema is significantly interfering with an individual's life (e.g. growth, sleeping, schoolwork or job). Ultraviolet phototherapy, prednisolone, cyclosporine, methotrexate and azathioprine can all be effective treatments. However, they all have side effects and the risk/benefit ratio must be openly discussed with the patient before they are used(12)

1.2 ATOPIC ECZEMA

Atopic dermatitis (AD) (a type of eczema) is an inflammatory, chronically relapsing, non-contagious and pruritic skin disease(13)

AD is an itchy, inflammatory skin condition with a predilection for the skin flexures(14). It is characterized by poorly defined erythema with edema, vesicles, and weeping in the acute stage and skin thickening (lichenification) in the chronic stage. Atopic dermatitis, or eczema, is a common skin disease that is often associated with other atopic disorders, such as allergic rhinitis, hay fever and asthma. The clinical manifestations of atopic dermatitis vary with age; three stages can often be identified. In infancy, the first eczematous lesions usually emerge on the cheeks and the scalp. Scratching, which frequently starts a few weeks later, causes crusted erosions. During childhood, lesions involve flexures, the nape, and the dorsal aspects of the limbs. In adolescence and adulthood, lichenified plaques affect the flexures, head, and neck. In each stage, itching that continues throughout the day, worsens at night causes sleep loss, and substantially impairs the patient's quality of life.

The hallmarks of atopic dermatitis are a chronic, relapsing form of skin inflammation, a disturbance of epidermal-barrier function that culminates in dry skin, and IgE-mediated sensitization to food and environmental allergens. The histological features of acute eczematous patches and plaques are epidermal intercellular edema (spongiosis) and a prominent perivascular infiltrate of lymphocytes, monocyte macrophages, dendritic cells, and a few eosinophils in the dermis. In subacute, and chronic lichenified and excoriated plaques, the epidermis is thickened and its upper layer is hypertrophied. Two hypotheses concerning the mechanism of atopic dermatitis have been proposed. One holds that the primary defect resides in an immunologic disturbance that causes IgE-mediated sensitization, with epithelial-barrier dysfunction regarded as a consequence of the local inflammation. The other proposes that an intrinsic defect in the epithelial cells leads to the barrier dysfunction; the immunologic aspects are considered to be an epiphenomenon.

DIAGNOSTIC CRITERIA FOR AD

The United Kingdom working group on AD published criteria for diagnosing atopic dermatitis that include the following :

1. Evidence of itchy skin, including the report by a parent of a child rubbing or scratching.

In addition to itchy skin, three or more of the following are needed to make the diagnosis:

2. History of skin creases being involved. These include: antecubital fossae, popliteal fossae, neck, areas around eyes, fronts of ankles.
3. The presence of generally dry skin within the past year.
4. Symptoms beginning in a child before the age of two years. This criterion is not used to make the diagnosis in a child who is under four years old.
5. Visible evidence of dermatitis involving flexural surfaces. For children under four years old, this criterion is met by dermatitis affecting the cheeks or forehead and outer aspects of the extremities.

The working party Diagnostic criteria for AD has been validated, sensitivity of 85% and specificity of 95%, this is a simplified criteria which is easy to use and takes less than 2 minutes per patient to ascertain diagnosis and does not require subjects to undress (15).

Histology

The histologic features of acute eczematous patches and plaques are epidermal intercellular edema (spongiosis) and a prominent perivascular infiltrate of lymphocytes, monocyte macrophages, dendritic cells, and a few eosinophils in the dermis. In subacute and chronic lichenified and excoriated plaques, the epidermis is thickened and its upper layer is hypertrophied.

Prevalence, Cost, and Prognosis

According to the International Study of Asthma and Allergies in Childhood, the prevalence of symptoms of atopic dermatitis in children six or seven years of age during a one-year period varied from less than 2 percent in Iran and China to approximately 20 percent in Australia, England, and Scandinavia.

A high prevalence of 17% has also been found in the United States. (16). In the United Kingdom, one population survey of 1760 affected children from one to five years of age found that 84

percent of cases were mild, 14 percent were moderate, and 2 percent were severe. Studies suggest that atopic dermatitis imposes a high economic burden(17). Out-of-pocket expenses and overall costs that are similar to those for the treatment of asthma. Causes of family stress related to caring for children with moderate or severe atopic dermatitis (e.g. sleep deprivation, loss of employment, time-consuming treatment, and financial costs) may rival those related to caring for children with diabetes mellitus(18). Approximately 60 percent of patients with childhood atopic dermatitis are free of symptoms in early adolescence although up to 50 percent may have recurrences in adulthood. Early-onset disease, severe early disease, concomitant asthma and hay fever, and a family history of atopic dermatitis may predict a more persistent course(19). One cohort study of 1314 German children showed that the prognosis was related to disease severity and atopic sensitization, as evidenced by elevated serum levels of IgE antibodies to food and inhalant allergens at two years of age(20)

Epidemiology of Atopic Dermatitis

The prevalence of atopic dermatitis has doubled or tripled in industrialized countries during the past three decades; 15 to 30% of children and 2 to 10% of adults are affected(21). This disorder is often the prelude to an atopic diathesis that includes asthma and other allergic diseases. Atopic dermatitis frequently starts in early infancy (so-called early-onset atopic dermatitis). A total of 45% of all cases of atopic dermatitis begin within the first 6 months of life, 60% begin during the first year, and 85% begin before 5 years of age. More than 50% of children who are affected in the first 2 years of life do not have any sign of IgE sensitization, but they become sensitized during the course of atopic dermatitis(22). Up to 70% of these children have a spontaneous remission before adolescence. The disease can also start in adults (so-called late-onset atopic dermatitis), and in a substantial number of these patients there is no sign of IgE-mediated sensitization.

The lower prevalence of atopic dermatitis in rural as compared with urban areas suggests a link to the "hygiene hypothesis," which postulates that the absence of early childhood exposure to infectious agents increases susceptibility to allergic diseases(23).

Symptoms of atopic eczema exhibit wide variations in prevalence both within and between countries inhabited by similar ethnic groups, suggesting that environmental factors may be critical in determining disease expression(24).

The vast majority of atopic dermatitis has an onset before age five years, and prevalence data in children show a slight female to male preponderance (1.3 to 1).Vast majority of patients experience negative psychosocial consequences of eczema. A comparison of physician rated assessment of severity with patient rated ones demonstrated a poor relationship indicating that patients value several aspects of their eczema differently from their physicians. Most patients who have the clinical appearance of atopic dermatitis do not have raised level of allergen specific IgE.Skin penetration of certain chemicals is increased in atopic dermatitis(25).

Genetics of Atopic Dermatitis

The concordance rate for atopic dermatitis is higher among monozygotic twins (77%) than among dizygotic twins (15%)(23). Allergic asthma or allergic rhinitis in a parent appears to be a minor factor in the development of atopic dermatitis in the offspring, suggesting atopic dermatitis-specific genes.

Genomewide scan have highlighted several possible atopic dermatitis-related loci on chromosomes 3q21(26) 1q21, 16q, 17q25, 20p, and 3p26.

The region of highest linkage was identified on chromosome 1q21, which harbors a family of epithelium-related genes called the epidermal differentiation complex(27).Atopic dermatitis is a complex genetic disease that arises from gene-gene and gene-environment interactions. The disease emerges in the context of two major groups of genes: genes encoding epidermal or other epithelial structural proteins, and genes encoding major elements of the immune system

AD is probably a complex disease relying on the interplay of several factors. Several genes have been identified that may explain some cases. Genetics alone, however, cannot explain the results of studies of migrant populations that show, for example, that Jamaican children living in London are twice as likely to have atopic dermatitis as Jamaican children living in Jamaica: the increased risk of atopic dermatitis in smaller families and among higher social classes; and the

rising prevalence of atopic dermatitis in some countries. These observations suggest a key role for the environment in mediating disease expression(28). Whereas allergens such as house-dust mites and foods may be important in some cases, nonallergic factors such as rough clothing, *Staphylococcus aureus* infections, exposure to microbes during infancy, excessive heat, and exposure to irritants that disrupt the function of the skin barrier may also be important.

A Unifying Hypothesis

One classification distinguishes an IgE-associated form of atopic dermatitis (i.e., true atopic dermatitis, formerly called extrinsic atopic dermatitis) from a non-IgE-associated form ("nonatopic" dermatitis, formerly called intrinsic atopic dermatitis. This division implies that nonatopic dermatitis and atopic dermatitis are two different diseases. However, since dry skin is an important sign of both conditions, and the absence of IgE-mediated sensitization may be only a transient factor, there is a need to reconcile these divergent hypotheses. A new picture emerges from recent findings, in which the natural history of atopic C dermatitis has three phases:

The initial phase is the nonatopic form of dermatitis in early infancy, when sensitization has not yet occurred.

Next, in 60 to 80% of patients, genetic factors influence the induction of IgE-mediated sensitization to food, environmental allergens, or both — this is the transition to true atopic dermatitis.

Third, scratching damages skin cells, which release auto antigens that induce IgE auto antibodies in a substantial proportion of patients with atopic dermatitis.

Clinical implications

Since the barrier dysfunction of the skin and chronic inflammation are characteristic of atopic dermatitis, long-term clinical management should emphasize prevention, intensified and individually adapted skin care, reduction of bacterial colonization and — most important — the control of inflammation by the regular use of topical corticosteroids or topical calcineurin inhibitors. In children, before and after the diagnosis of IgE-mediated sensitization, measures that

prevent exposure to allergens should be beneficial. The current therapy of atopic dermatitis is reactive — treating the flares — but management should include early and proactive intervention with effective and continuous control of the skin inflammation and *S. aureus* colonization. This strategy has proved to be effective in reducing the number of flares(29). When applied early in infancy, it could potentially help to reduce later sensitization to environmental antigens and auto allergens.

Recent insights into the genetic and immunologic mechanisms that drive cutaneous inflammation in atopic dermatitis have led to a better understanding of the natural history of this disease and have highlighted the critical role of the epidermal-barrier function and the immune system. Both contribute to IgE-mediated sensitization and should be considered as major targets for therapy. New developments aimed specifically at the molecular defects in the stratum corneum could provide a customized way to improve the barrier function. Early and proactive management could improve the outcome and quality of life for patients with atopic dermatitis.

1.3 NUMMULAR (DISCOID) ECZEMA

Nummular (meaning "coin-shaped") dermatitis is a form of eczema. Nummular dermatitis is characterized by round-to-oval erythematous plaques most commonly found on the arms and legs(13). Lesions often start as papules, which then coalesce into plaques with scale. They are usually very pruritic

PATHOPHYSIOLOGY

Nummular dermatitis is a condition confined to the skin. It has recently been considered a form of adult onset AD.

Little is known about the pathophysiology of nummular dermatitis, but it is frequently accompanied by xerosis. Dryness of the skin results in dysfunction of the epidermal lipid barrier; this may allow permeation of environmental allergens, which induce an allergic or irritant response(30)This is supported by one study that showed that elderly patients with nummular dermatitis had increased sensitivity to environmental aeroallergens compared with age-matched controls.

Onset has been associated with medications. Onset of severe, generalized nummular lesions has been reported in association with interferon and ribavirin therapy for hepatitis C(31). Association with use of inhibitors of tumor necrosis factor has also been reported.

The etiology is unknown and likely multifactorial, dry skin, trauma, venous insufficiency, use of interferons, giardiasis and H.pylori have been implicated(32).

Increased numbers of mast cells have been observed in lesional compared with nonlesional samples in persons with nummular dermatitis.

Other research has demonstrated that mast cells present in the dermis of patients with nummular eczema may have decreased chymase activity, imparting reduced ability to degrade neuropeptides and protein. This dysregulation could lead to decreased capability of the enzyme to suppress inflammation.

The prevalence of nummular dermatitis is 2 cases per 1000 people(32), no racial predilection has been observed for nummular dermatitis and is more common in males than in females. Nummular dermatitis has 2 peaks of age distribution. The most common is in the sixth to seventh decade of life. This is most often seen in males. A smaller peak occurs in the second to third decade of life.

Patients present with a days-to-months' history of a pruritic eruption, usually starts on the legs. It may also burn or sting, often waxes and wanes with winter; cold or dry climates or swings in temperature may be exacerbating factors. New nummular dermatitis lesions often recur in the same locations as old lesions.

The diagnosis of nummular dermatitis is made on the basis of observing the characteristic round-to-oval erythematous plaques. They are most commonly located on the extremities, particularly the legs, but they may occur anywhere on the trunk, hands, or feet. Nummular dermatitis does not involve the face and scalp. Lesions are often symmetrically distributed. LSC often occurs on the lower legs, the neck, the scalp, or the scrotum; it is lichenified (thickened by chronic scratching), more violaceous, and, often, has no clear border.

1.4 DYSHYDROTIC (HAND, POMPHOLYX) ECZEMA

Is a skin condition that is characterized by small blisters on the hands or feet. It is an acute, chronic, or recurrent dermatosis of the fingers, palms, and soles, characterized by a sudden onset of many deep-seated pruritic, clear vesicles later, scaling, fissures and lichenification occur(13).

Dyshidrotic eczema is a type of eczema of unknown cause that is characterized by a pruritic vesicular eruption on the fingers, palms, and soles. The condition affects teenagers and adults and may be acute, recurrent, or chronic. A more appropriate term for this vesicular eruption is pompholyx, which means bubble. The cause of dyshidrotic eczema remains enigmatic. Some believe the terms pompholyx and dyshidrosis are obsolete and favor a new term, such as "acute and recurrent vesicular hand dermatitis"(33).

Epidemiology

The etiology of dyshidrotic eczema is unknown. The condition was inaccurately described in 1873 as dyshidrosis because of the clinical symptom of sweaty palms. The term dyshidrosis indicates a sweating abnormality, although histologic examination reveals no evidence of eccrine glandular involvement. Histologically, the vesicles are intraepidermal and spongiotic with little to no inflammatory changes. The more appropriate term for this vesicular eruption is pompholyx, which means bubble. A tiny percentage of individuals with the disorder note flares after ingesting metal salts, specifically chromium, cobalt, and nickel. Diets that eliminate these metal salts may rarely have some clinical benefit.

One causative study observed reactional pompholyx to interdigital-plantar intertriginous and endogenous reactions to metals or other allergens; however, an unexpected number of patients with so-called contact pompholyx, in which cosmetic and hygiene products play a preponderant role (compared with metals), were also reported(33).

A genetic component to the development of dyshidrotic eczema may be involved in some patients. Dyshidrotic eczema has been described in few large families; no gene or locus had been identified(34).A genome-wide search in a large Chinese family identified a locus at chromosome 18q22.1-18q22.3, with a maximum 2-point logarithm of the odds (LOD) score of 3.61 at marker

D18S1131 ($\theta = 0.00$). Haplotype analyses showed the gene to be located within 12.07 cM region between markers D18S465 and D18S1362, which corresponds to 8 Mb(34).

In USA dyshidrotic eczema accounts for 5% of all cases of eczema of the hand(35).A study in Turkey revealed a higher prevalence of dyshidrotic eczema in the summer months(36).No racial predilection is reported. The female-to-male ratio is 2:1.Peak incidence occurs in patients aged 20-40 years, although the disorder also occurs in teenagers and older patients.

Patients with dyshidrotic eczema first describe several hours of itching or burning sensations in their hands, feet, or both before the eruption develops. Tiny vesicles erupt first along lateral aspects of the fingers and then on the palms or soles. Palms and soles may be red and wet with perspiration. The vesicles usually persist for 3-4 weeks. Vesicle outbreaks may occur in waves. A photo-induced form of hand dermatitis resembling dyshidrotic eczema has been described(37).

Aetiology of dyshidrotic eczema remains undefined, suspected risk factors include stress, exposure to metal salts, allergic contact dermatitis, and female sex. Iannaccone et al cite exposure to intravenous immunoglobulin G (IVIG) as a possible risk factor.Exogenous factors said to trigger a flare include dermatophyte infections, contact irritants, and metal hypersensitivity. Although metal hypersensitivity does not play a role in all cases of dyshidrotic eczema, high oral ingestion of nickel and/or cobalt should be considered, regardless of patch test results.

1.5 SEBORRHEIC DERMATITIS

Seborrhoeic dermatitis a skin disorder affecting the scalp, face, and trunk causing scaly, flaky, itchy, red skin.it particularly affects the sebum-gland rich areas of skin(11).

Seborrheic dermatitis is a papulosquamous disorder patterned on the sebum-rich areas of the scalp, face, and trunk. In addition to sebum, this dermatitis is linked to *Malassezia*, immunologic abnormalities, and activation of complement. It is commonly aggravated by changes in humidity, changes in seasons, trauma (eg. scratching), or emotional stress. The severity varies from mild dandruff to exfoliative erythroderma. Seborrheic dermatitis may worsen in Parkinson disease and in AIDS(38).

Pathophysiology

Seborrheic dermatitis is associated with normal levels of *Malassezia* but an abnormal immune response. Helper T cells, phytohemagglutinin and concanavalin stimulation, and antibody titers are depressed compared with those of control subjects. The contribution of *Malassezia* species to seborrheic dermatitis may come from its lipase activity—releasing inflammatory free fatty acids—and from its ability to activate the alternative complement pathway(39).

Epidemiology

The prevalence rate of seborrheic dermatitis is 3-5%, with a worldwide distribution. Dandruff, the mildest form of this dermatitis, is probably far more common and is present in an estimated 15-20% of the population. The usual onset occurs with puberty. It peaks at age 40 years and is less severe. In infants, it occurs as cradle cap or, uncommonly, as a flexural eruption or erythroderma(34). Intermittent, active phases of seborrheic dermatitis manifest with burning, scaling, and itching, alternating with inactive periods. Activity is increased in winter and early spring, with remissions commonly occurring in summer.

Generalized seborrheic erythroderma is rare. It occurs more often in association with AIDS, congestive heart failure, Parkinson disease, and immunosuppression in premature infants(39).

Malassezia organisms are probably not the cause but are a cofactor linked to a T-cell depression, increased sebum levels, and an activation of the alternative complement pathway. Persons prone to this dermatitis also may have a skin-barrier dysfunction(40). Various medications may flare or induce seborrheic dermatitis. These medications include auranofin, aurothioglucose, buspirone, chlorpromazine, cimetidine, ethionamide, gold, griseofulvin, haloperidol, interferon alfa, lithium, methoxsalen, methyldopa, phenothiazines, psoralens, stanozolol, thiothixene, and trioxsalen.

1.6 STASIS (VENOUS, GRAVITATIONAL) ECZEMA

Stasis dermatitis is a common inflammatory skin disease that occurs on the lower extremities in patients with chronic venous insufficiency with venous hypertension. The condition typically affects middle-aged and elderly patients. It rarely occurs before the fifth decade of life, except in patients with acquired venous insufficiency due to surgery, trauma, or thrombosis. Stasis dermatitis is usually the earliest cutaneous sequela of venous insufficiency, and it may be a

precursor to more problematic conditions, such as venous leg ulceration and lipodermatosclerosis(41).

Pathophysiology

Stasis dermatitis occurs as a direct consequence of venous insufficiency. Disturbed function of the 1-way valvular system in the deep venous plexus of the legs results in backflow of blood from the deep venous system to the superficial venous system, with accompanying venous hypertension. This loss of valvular function can result from an age-related decrease in valve competency. Alternatively, specific events, such as deep venous thrombosis, surgery (eg. vein stripping, harvesting of saphenous veins for coronary bypass), or traumatic injury, can severely damage the function of the lower-extremity venous system.

Increased venous hydrostatic pressure is transmitted to the dermal microcirculation: this leads to increased permeability of dermal capillaries.increased permeability enables macromolecules, such as fibrinogen, to leak out into the pericapillary tissue; then, polymerization of fibrinogen to fibrin results in the formation of a fibrin cuff around dermal capillaries. It has been hypothesized that this fibrin cuff serves as a barrier to oxygen diffusion, with resulting tissue hypoxia and cell damage.The phenomenon of fibrin cuff formation is found in severe disease, such as venous ulceration. Fibrin cuffs are not found in ulcers due to causes other than venous hypertension. Decreased cutaneous fibrinolytic activity has been proposed to contribute to the formation of fibrin cuffs(42).

Formation of fibrin cuffs, coupled with decreased fibrinolysis, results in the dermal fibrosis that is the hallmark of advanced stasis dermatitis. Activated leukocytes become trapped in fibrin cuffs and the surrounding perivascular space, releasing inflammatory mediators that contribute to inflammation and fibrosis(43).These leukocytes release the growth factor transforming growth factor-beta1, an important mediator of dermal fibrosis. Furthermore, upregulation of vascular intercellular adhesion molecule-1 (ICAM-1) and vascular cell adhesion molecule-1 (VCAM-1), which are potent chemoattractants to keep leukocytes active in the perivascular environment, occurs. The finding of leukocyte-mediated cytokine production, aided by fibrin cuff formation, provides a direct link between dysfunctional venous circulation and cutaneous inflammation with fibrosis(44).

Herouy et al suggested that matrix metalloproteinases may be important in lesional skin remodeling in persons with stasis dermatitis.

Studies have estimated the prevalence of stasis dermatitis to be approximately 6-7% in patients older than 50 years. This finding makes stasis dermatitis twice as prevalent as psoriasis and only slightly less prevalent than seborrheic dermatitis(43).

Epidemiology

Prevalence of 6-7% would translate into approximately 15-20 million patients older than 50 years with stasis dermatitis in the United States. Much of the morbidity stems from the complications of chronic stasis dermatitis, including cellulitis and nonhealing venous ulcer(45).

A slight female preponderance has been reported in stasis dermatitis. This is most likely due to the fact that pregnancy results in significant stress on the lower-extremity venous system, with many women experiencing earlier and more severe derangement of lower-extremity valvular function.

The risk of developing stasis dermatitis steadily increases with each passing decade; when considering only adults older than 70 years, the prevalence of stasis dermatitis may be greater than 20%. Presents as an erythematous, scaling, eczematous patches affecting the lower extremity.

The medial ankle is most frequently and severely involved because of the fact that the medial ankle represents a watershed area with relatively poor blood flow compared with the rest of the leg. In advanced cases of stasis dermatitis, the inflammation may encircle the ankle and extend to just below the knee; this is sometimes referred to as stocking erythroderma(45).

1.7 ASTEATOTIC (WINTER, CRAQUELE, SENILE) ECZEMA

First described by Brocq in 1907,Chistina K. Anderson et al, using the term eczema craquele. asteatotic dermatitis is characterized by pruritic, dry, cracked, and polygonally fissured skin with irregular scaling. It most commonly occurs on the shins of elderly patients, but it may occur on the hands and the trunk.

Pathophysiology

Initially, excess water loss from the epidermis results in dehydration of the stratum corneum with upward curling of corneocytes. The outer keratin layers require 10-20% water concentration to maintain their integrity. A significant decrease in free fatty acids in the stratum corneum is present in people with asteatotic dermatitis. Stratum corneum lipids act as water modulators, and cutaneous loss of these lipids can increase transepidermal water loss to 75 times that of healthy skin(46).Elderly persons with decreased sebaceous and sweat gland activity, patients on antiandrogen therapy, people using degreasing agents, and people bathing without replacing natural skin emollients lost to bath water are at risk for asteatotic eczema.

When the stratum corneum loses water, the cells shrink. A significantly decreased cellular volume can stress the skin's elasticity, creating fissures. Edema in the dermis leads to additional stretch on the overlying epidermis. Fissures rupture dermal capillaries, causing clinical bleeding. The disruption of cutaneous integrity can result in inflammation with risk of infection. Transepidermal absorption of allergens and irritants is increased as the epidermis is damaged, increasing susceptibility to allergic contact dermatitis and irritant contact dermatitis.

Seasonality is prominent, and most patients present in the winter months, especially in areas where indoor humidity is decreased by heating. The frequency of asteatotic dermatitis is increased in the northern United States, particularly during the winter season(47).

Epidemiology

Although most cases resolve without ill effects, asteatotic dermatitis can be chronic with relapses frequent during the winter months and during times of low humidity.Men older than 60 years develop asteatotic dermatitis more commonly than women,the median patient age at presentation is 69 years. Asteatosis can also occur in young people.

Presents with pruritic and dry skin with dermatitis on the pretibial areas. Sometimes, the dysesthesia may be described as a pinprick or biting sensation.

Primary lesions: Slightly scaly, inflamed, linearly cracked and/or fissured skin most commonly involves the pretibial areas, but it may also occur on the thighs, on the hands, and on the trunk

Causes

Multiple etiologic factors may coexist to cause asteatotic dermatitis, frequent or prolonged bathing in hot water, decreased sebaceous and sweat gland activity in elderly persons, decreased keratin synthesis in elderly persons, low environmental humidity and cold winds that increase the loss of water by convection, radiation, long-term malabsorption of essential fatty acids, including linoleic acid and linolenic acid, Nutritional deficiencies, zinc deficiency(48), Essential fatty acid deficiency, such as linoleic acid deficiency or linolenic acid deficiency.

Myxedema and other thyroid diseases with diminished sweat and sebaceous gland activity(49), Neurologic disorders with decreased sweating in denervated areas.

Drugs like antiandrogen therapy, cimetidine and diuretic therapy(50), Malignant lymphoma(51), gastric adenocarcinoma, glucagonoma, angioimmunoblastic lymphadenopathy(52), breast cancer, large-cell lung carcinoma, and colorectal carcinoma.

Pityriasis alba, occurs in children within age group of 3 to 16 years and is characterized by white skin patches with delicate scales on the face, trunk and limbs.

Pityriasis alba is sometimes a manifestation of atopic dermatitis in children(53), but can occur as an independent eczema. The cause is not known, but many hypotheses prevail regarding the origin of this childhood eczema. A relative vitamin and calcium deficiency from inappropriate food habits in children seems to be the most plausible explanation, as pityriasis alba invariably occurs in the growing years in children.

Worm infestations, sun sensitivity, dry skin, and sensitization to various food preservatives have also been cited as the causes for this childhood disorder with loss of skin color(54).

2 ADHERENCE

Adherence to (or compliance with) a medication regimen is generally defined as the extent to which patients take medications as prescribed by their health care providers. The word "adherence" is preferred by many health care providers, because "compliance" suggests that the

patient is passively following the doctor's orders and that the treatment plan is not based on a therapeutic alliance or contract established between the patient and the physician(55).

Adherence rates are typically higher among patients with acute conditions, as compared with those with chronic conditions; persistence among patients with chronic conditions is disappointingly low, dropping most dramatically after the first six months of therapy(56).

The average rates of adherence in clinical trials can be remarkably high, owing to the attention, study patients receive and to selection of the patients, yet even clinical trials report average adherence rates of only 43 to 78 percent among patients receiving treatment for chronic conditions. There is no consensual standard for what constitutes adequate adherence. Some trials consider rates of greater than 80 percent to be acceptable, whereas others consider rates of greater than 95 percent to be mandatory for adequate adherence, particularly among patients with serious conditions such as infection with the human immunodeficiency virus (HIV).

Pharmionics, the study of what patients do with prescribed medications, has attracted much research interest in the era of rising healthcare costs and cost-effectiveness analysis. Medication adherence lies at the core of pharmionics, and the annual cost of non-adherence was estimated at \$US100 billion in 1993, or 70% of healthcare expenditure on drugs(57) Non-adherence refers to unfilled prescriptions, incorrect dosages, incorrect dosing intervals, skipped doses, or premature cessation of medication usage, and its correlation with health outcomes has been extensively studied with oral medications. Non-adherence has been causally related to treatment failure and adverse health outcomes in acute conditions such as bacterial infections, as well as in chronic conditions such as hypertension, hypercholesterolemia, diabetes mellitus, HIV, and epilepsy(58). Adherence to topical dermatological treatment may be as poor as 32-61% for different diseases(59).

Rates of adherence to medication regimens among children with chronic diseases are similar to those among adults with chronic diseases, averaging about 50 percent, with decrements in adherence occurring with time(60).

Race, sex, and socioeconomic status have not been consistently associated with levels of adherence(61).

Given the magnitude and importance of poor adherence to medication regimens, the World Health Organization has published an evidence-based guide for clinicians, health care managers, and policymakers to improve strategies of medication adherence(62).

In dermatology, topical administration is the primary tool since the medication is applied directly to the diseased site, thus localizing therapy and minimizing toxicity to other sites.

Thirty-seven children were given 0.1% triamcinolone ointment and were counseled to use it twice daily. They were told to return in 4 weeks, at which time they were told to continue treatment for another 4 weeks. Electronic monitors were used to measure adherence over the entire 8 week study. Patients were not informed of the compliance monitoring until the end of the study. The findings were that adherence to topical medications is very poor in a clinic population of children with atopic dermatitis. Office visits are one means to increase adherence. If adherence to topical treatment can be improved, exposure to more costly and potentially toxic systemic agents may be avoidable.

The efficacy of topical medications generally increases when they are used as directed. To help patients achieve optimal therapeutic outcomes, it is worthwhile for physicians to utilize strategies that may improve patient adherence. Effective strategies include positive patient-physician relationships, patient education, follow-up visits or calls, and patient participation in treatment decisions. Adherence to topical dermatological treatment may be as poor as 32-61% for different diseases(63).

The mini-questionnaires for oral and topical treatments has 89% specificity for detecting poor adherence. mini-questionnaires provide dermatologists with specific tools to rapidly identify poor compliers(64).

Adherence Assessment Methods

Since non-adherence to medication regimens has been correlated with adverse effects, accurate detection of non-adherence is needed to identify individuals at risk of suboptimal treatment outcomes.

The commonly used methods used to measure adherence include direct questioning, Drug diaries, Pill counts, Prescription renewals, and medication tube weights. These have been shown overestimate adherence (57).

More invasive methods such as serum marker tests do not offer greater accuracy as they reflect drug intake only during the past three half-lives of the drug.

Medication event monitoring system (MEMS), a microchip embedded in a bottle cap, was introduced in the 1980s. It records the opening and closing of medication bottles but does not evaluate whether the medication was actually consumed. MEMS has been shown to provide more accurate information on dosing intervals and missed doses than traditional measures do. Originally limited to research use, MEMS technology costs <\$US0.10 daily and is presumably cost-effective since it obviates the need for extra clinic visits and laboratory tests to evaluate medication adherence(58).

The use of MEMS with topical medications has been limited by the difficulty of adapting the technology to fit medication tubes, the stability of the vehicle and drug formulation in a MEMS container, and the inability of MEMS to measure the amount dispensed at each tube opening-closing event. Despite these difficulties, MEMS has proven to be more accurate in assessing topical medication adherence than surveys, diaries, and medication tube weights(65).

Adherence to Oral Medications

Medication non-adherence and predictors of this behavior have long been a focus of research.

In a study of 24 patients observed by MEMS over a period of 3428 days, adherence rates averaged 76% for the duration of the study, and adherence was significantly and inversely correlated with frequency of administration. Eighty-seven percent of subjects complied with once-daily administration, 81% with twice-daily administration, 77% with three-times-daily administration, and 39% with four-times-daily administration. Other studies have drawn similar conclusions, citing frequency of drug administration, medication cost, and number of total medications as risk factors for non-adherence(66). While frequency of administration has consistently been associated with non-adherence, the effect of the total number of medications on adherence remains inconclusive. A reflection of drug regimen complexity, the number of total

medications has been negatively associated with adherence in the elderly population(66). In other studies, the number of total medications has been correlated with an increase in adherence that hypothetically stems from familiarity with taking medications and incorporation of the drug regimen into daily life. Adherence to one medicine in a multimedicine regimen has also been proven to be highly predictive of adherence to all medications in the regimen and particularly to synchronously scheduled medicines(67). Thus far, the aforementioned studies have suggested that medication adherence is independent of disease, drug, prognosis, or symptoms. There is inconclusive evidence in these studies that age, sex, race, socioeconomic status, and physician-patient communication are predictors of adherence.

2.1 TABLE 1: METHODS OF MEASURING ADHERENCE (68)

| Test | Advantage | Disadvantage |
|---|--|---|
| Direct methods | | |
| Direct observed method | Most accurate | Patients can hid pills in the mouth and then discard them; impractical for routine use |
| Measurement of the level of medicine or metabolite in blood | Objective | Variations in metabolism and "white-coat adherence" can give a false impression of adherence; expensive. |
| Measurement of the biologic marker in blood | Objective; in clinical trials, can also be used to measure placebo | Requires expensive quantitative assays and collection of bodily fluids |
| Indirect methods | | |
| Patient questionnaire, patient self-reports | Simple, inexpensive; the most useful method in the clinical setting | Susceptible to error with increases in time between visits; results are easily distorted by the patients |
| Pill counts | Objective, quantifiable and easy to perform | Data easily altered by the patient (e.g., pill dumping) |
| Rates of prescription refills | Objective; easy to obtain data | A prescription refill is not equivalent to ingestion of medication; requires a closed pharmacy system |
| Assessment of patient's clinical response | Simple; generally easy to perform | Factors other than medication adherence can affect clinical response |
| Electronic medication monitors | Precise, results are easily quantified; tracks patterns of taking medication | Expensive; requires return visits and downloading data from medication vials |
| Measurement of physiologic markers(e.g., heart rate in patients taking beta-blockers) | Often easy to perform | Marker may be absent for other reasons(e.g. increased metabolism, poor absorption, lack of response) |
| Patients diaries | Help to correct for poor recall | Easily altered by the patient |
| When the patient is a child, questionnaire for caregiver or teacher | Simple; objective | Susceptible to distortion |

Although predictors of non-adherence are unclear, the undesirable consequences of non-adherence are apparent. Non-adherence is the most common cause of non-response to a medication (67). Lack of response to a medication is often interpreted as 'drug failure', and physicians commonly decide to switch medications or increase the drug dosage as a result. In a study (n = 5056) of oral hypoglycemic agents, there was a 45% greater likelihood of regimen intensification (increased dosage) among patients whose pharmacy dispensing data indicated that they had taken <80% of the prescribed doses(69). Changing medications or increasing dosage secondary to non-adherence may harm patients by eliminating potentially effective drugs from the patient's treatment options or increasing the risk of adverse effects and toxicity, respectively.

Despite the prevalence of the problem, clinical recognition of non-adherence is poor(67). Many physicians do not think of non-adherence as a potential cause of minimal response or lack of response to medication. One survey showed that only 25% of physicians discussed medication regimens with their patients(70). Studies have repeatedly shown that unresolved concerns, miscommunication, and regimen complexity are common barriers to adherence(66) Thus, the need for physicians to explain the medication regimen and ask patients whether they would realistically adopt the regimen is crucial.

Adherence in Dermatology. It has long been inferred that non-adherence to dermatologic medications is less common given the visible nature of the disease and the potential for more social and psychological consequences from dermatologic disease(71). In reality, topical medication non-adherence poses a significant problem for dermatologists and their patients.

Several barriers to adherence are unique to topical medications and the field of dermatology. Topical medications may be time-consuming to apply, non-esthetic, or irritating and are often less preferable than oral and even intramuscular administration of drugs. They may be difficult to apply for patients with physical handicaps. The inexactness of dosage/application terms such as 'sparingly' or 'liberally' offers much room for interpretation and self-titration, and the context of medication application such as 'damp or dried skin' may be difficult to remember or inconvenient to follow. Aggressive marketing efforts promoting cosmeceuticals, conflicting messages from non-healthcare professionals in the beauty industry, and easy access to quasi-medical articles and websites also encourage patients to question and challenge medical advice and treatment.

Atopic Dermatitis

Atopic dermatitis is often a chronic disease punctuated with intermittent flares. Prevention of flares relies heavily on emollient use during asymptomatic periods of disease. Only in times of active disease are corticosteroids or nonsteroidal anti-inflammatory medications employed and even then they are used with suboptimal adherence because of the prevalence (72%) of corticosteroid phobia(72).As in the case with oral antihypertensives, adherence to aggressive emollient use is poor when the disease is silent,(73) and patient education about the disease and consistent medication and emollient use are critical to disease management. In a 1-year study of 51 children with poorly controlled disease (Six Area, Six Sign Atopic Dermatitis [SASSAD] severity score of 42.9), 24% were not being treated with any emollient(73) Those who were using emollients were applying suboptimal amounts (mean 54 g/week). Furthermore, 25% of patients were being inappropriately treated with potent (class I-III) corticosteroids relative to their disease.

This study verified the importance of patient education in improving medication adherence and treatment outcomes. A specialist dermatology nurse provided verbal and written instructions on the nature of atopic dermatitis, explained how medications and emollients exert their effects, and discussed targets for the length of time that a medication tube should last. The parents of the subjects then filled prescriptions and returned with the products to the dermatology nurse. The nurse then demonstrated how to apply emollients and the use of fingertip units for topical corticosteroid application. Advice on how to make topical medications more acceptable to pediatric patients was also given. At 1-year follow-up, 77% of patients exhibited target emollient use. Emollient use increased by 800% to a mean of 426 g/wk. There was no increase in the potency or quantity of corticosteroids used. The increase in adherence was associated with an 89% decrease in mean disease severity, reducing the SASSAD severity score from 42.9 to 4.6(42) Such findings strongly support the link between topical medication adherence and favorable dermatologic outcomes, and stress the value of patient education as a method of bolstering adherence.

Non-adherence to topical medications poses a common and costly problem in the care of dermatologic patients. It accounts for poor patient response to medications and, subsequently, poor control of their skin disease or development of preventable disease.

Physician recognition of non-adherence and addressing common barriers to adherence are of paramount importance in minimizing non-adherence and its impact on health. The available evidence supports the need for physicians to establish a partnership with patients to develop a medication plan that is suitable for the patient and his/her lifestyle. Physicians or trained dermatology nurses who review medications, their purpose, application instructions, and adverse and desired effects have proven to be effective in improving patient understanding of the disease and medication adherence. Frequent physician assessment of medication adherence and scheduling of follow-up visits to re-evaluate drug response are also likely to improve patient adherence.

Better drug design, in terms of pharmacokinetics and pharmacodynamics, may be useful for complementing educational and counseling efforts to improve patient adherence. The ease of administration, cosmetic acceptability, and the 'forgiveness' of drug dosing (i.e. the duration of therapeutic action minus the recommended interval between doses) could be improved to facilitate patient adherence to topical medications(74). Future research and implementation of aggressive and creative interventions that address adherence issues could, in the end, prove to be of substantial value in patient care.

Quality-of-Life Assessment

When eczema is in the acute phase, the quality of life of the child is clearly reduced, particularly in patients with moderate or severe forms; active eczema also has an important impact on family life(75) of the family of the affected child also has a disturbed lifestyle: the chronic course of the disease with frequent relapses puts a special burden on both children and their parents.

Reasons for medication non adherence include forgetfulness, lack of efficacy, inconvenience, change in disease severity, and fear of medication side effects(76). In addition, patients may find topical treatments to be particularly unpleasant and time consuming relative to other types of medications . Prior studies suggest that more frequent patient contact(76) (e.g., office visits or

phone calls), less messy medication formulations, clear instructions, and education about the natural course of the disease and the rationales for different therapeutic strategies (77) could be viable means for increasing adherence.

3 ECZEMA CONTROL

Control of a disease is the goal for most clinicians in all chronic diseases.

Eczema is considered to be adequately controlled when the patient has no symptoms and no signs of eczema.

To assess eczema control validated scales are used, and it is recommended that one uses an objective scale. In this study SCORAD was used, it is validated and also widely used to assess control and severity of all types of eczema.

Control of a disease is affected by several factors:

1. Adherence to prescribed therapy.
2. Availability of correct medicine and right dosage.
3. Correct diagnosis.
4. Disease characteristics.
5. Presence psychiatric illness.

In management of eczema it is paramount that the correct diagnosis is made since eczema can be confused with other papulosquamous disorders.

3.1 SCALES FOR ASSESSING ECZEMA SEVERITY

In 1993, a consensus report of the European Task Force on Atopic Dermatitis defined a validated scale, the SCORAD (SCORing Atopic Dermatitis) in which the extent and the severity of the lesions and subjective symptoms, such as pruritus and loss of sleep, were evaluated (78). In 1997, the European Task Force on Atopic Dermatitis published a clinical validation and guidelines for the use of the SCORAD index.

When the SCORAD index is below 25 (out of a maximum score of 103), eczema severity is considered to be 'mild'; when it is between 25 and 50 to be 'moderate'; and when over 50 to be severe(79).in adequately controlled eczema SCORAD index score is zero.

Berth-Jones(80) in 1996, proposed a simple clinical severity score named SASSAD (Six Area, Six Sign Atopic Dermatitis), addressed to children and adults. The score was obtained by the grading of six signs (erythema, exudation, excoriation, dryness, cracking, and lichenification), each on a scale of 0 (absent), 1 (mild), 2 (moderate), or 3 (severe), at each of six sites: arms, hands, legs, feet, head/neck, and trunk. However, a limitation of the SASSAD is that it does not measure the extent of the lesions.

Another validated scoring system was proposed in 2001 by Hanifin et al(81) and named EASI (Eczema Area and Severity Index), addressed to both children and adult patients. Four anatomic areas of the body were evaluated: the head/neck, the trunk, and the upper and lower extremities. Each of the four body regions was separately assessed for erythema, induration/papulation/edema, excoriation, and lichenification. The average degree of severity of each sign in each of the four body regions was assigned a score ranging from 0 to 3. Symptoms (e.g. pruritus), along with secondary signs (e.g. xerosis, scaling) were excluded from the area assessments. This represents a simple system that can be used by practitioners and investigators to standardize the baseline evaluation of eczema and track changes in eczema over time. However, there are no clear cuts to define at which score the disease is mild, moderate, or severe.

An attempt to use an objective method to quantify severity in pediatric patients produced the OSAAD (Objective Severity Assessment of Atopic Dermatitis), a scale based on transepidermal water loss and skin hydration(82) however, this method is very difficult to use in clinical practice.

In 2004, Charman et al.(73) published a new, validated score called the POEM (Patient-Oriented Eczema Measure), which was based on patients' views of what constitutes disease severity. This tool can be used both for adults and children; the patients are supposed to answer simple questions about the frequency of seven symptoms: itch, sleep disturbance, skin bleeding, skin

weeping/oozing, skin cracking, skin flaking, and skin dryness/roughness. One major limitation of the POEM is that it is only subjective.

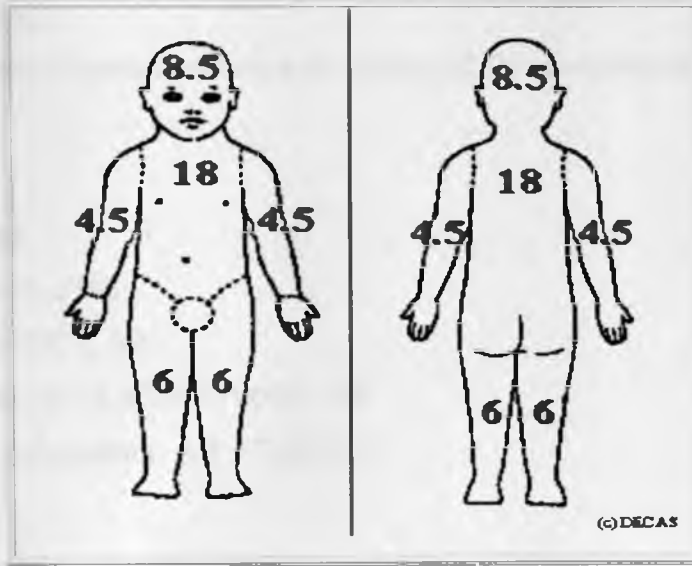
The IGA (Investigator Global Assessment) is another severity score tool that has been widely used in pediatric trials. It is a simple, 6-point scale ranging from 0 (clear) to 5 (very severe disease), representing an overall evaluation of dermatitis that can be performed by the investigator at every visit.

Many more severity scores have been published, most of which have not been validated. In 2003, Charman and colleagues (84) found that only 27% of the investigators using eczema severity scales assessed the clinical course of the disease with a tool that had been published before. In 2004, Eichenfield(85) considered the SCORAD index and the EASI system as the two methods that appear the most reliable and user friendly in clinical practice. These instruments are useful to identify different categories of severity in both everyday practice and clinical trials. A recent systematic review identified 20 different published outcome measures, but the authors state that only EASI, SCORAD, and POEM have been adequately validated, and recommend using EASI or SCORAD for an objective estimate of disease severity, plus the POEM as a measurement of eczema severity from the patient's perspective. It should not be forgotten that, in terms of bibliometry, SCORAD is the most widely used severity score in clinical research and should not be placed on the same level as EASI, which was validated *a posteriori* following industry-sponsored trials.

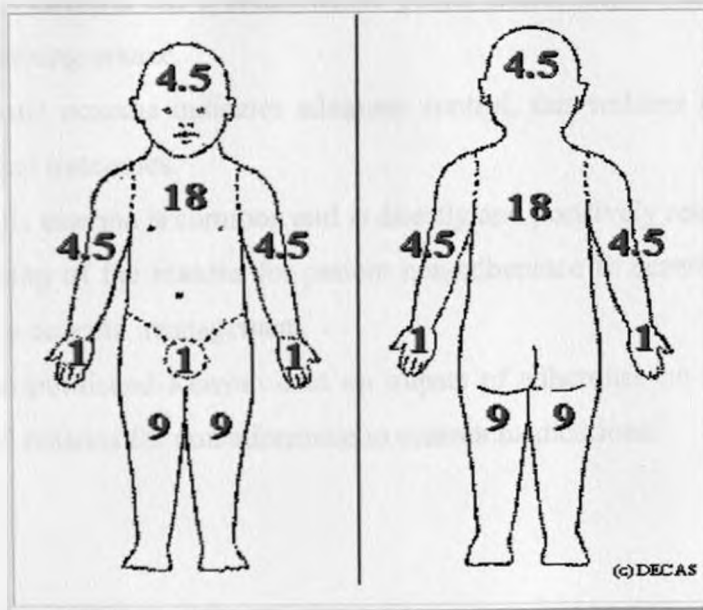
3.2 SCORAD

Extent criteria

1. The rule of 9 before the age of 2



The rule of 9 in adults and older children



2. Intensity criteria

- Erythematic : stage 1/ stage 2/ stage 3
- Edema / population : stage 1/ stage 2/ stage 3

| | |
|----------------------------|----------------------------|
| <u>Oozing / crusting</u> : | stage 1 / stage 2/ stage 3 |
| <u>Excoriation</u> : | stage 1 / stage 2/ stage 3 |
| <u>Lichenification</u> : | stage 1 / stage 2/ stage 3 |
| <u>xerosis</u> : | stage 1 /stage 2 / stage 3 |

3. Subjective symptoms

The two most representative items concerning the quality of life of patients are :

Pruritus

Insomnia

4. How is it calculated

A = SPREAD.../100

B = INTENSITY.../18

C = SUBJECTIVE SYMPTOMS.../20

SCORAD calculation: $A/5 + 7.B/2 + C$

4 STUDY JUSTIFICATION

- Endogenous Eczema has a considerable public health impact because it tends to run a chronic relapsing course.
- Asymptomatic eczema indicates adequate control, this reduces morbidity and negative psychological outcomes.
- Symptomatic eczema is common and is directly and positively related to morbidity.
- Understanding of the reasons for patient non-adherence to eczema medicine is essential for effective eczema management.
- There is no published Kenyan data on impact of adherence on severity of endogenous eczema and reasons for non adherence to eczema medications.

5 RESEARCH QUESTION

What is the impact of adherence to severity of endogenous eczema in patients attended at KNH?

6 NULL HYPOTHESIS

Adherence to medication does not affect severity of endogenous eczema in patients at KNH.

7 PRIMARY OBJECTIVE

The primary objective of this study is to determine the impact of adherence on severity of endogenous eczema in patients seen at KNH.

8 SPECIFIC OBJECTIVES

1. To determine the proportion of patients with asymptomatic and symptomatic endogenous eczema.
2. To determine the proportion of patients who are adherent and non-adherent to pharmacologic therapy.
3. To establish association between adherence and severity of endogenous eczema.
4. To establish relationship between socio-demographic characteristics and adherence.
5. To determine the reasons for non-adherence.

9 STUDY METHODOLOGY

9.1 Study design

A cross-sectional descriptive study.

9.2 Study site

Dermatology clinic, medical and paediatric wards.

9.3 Study population

Patients with eczema and on follow up for at least 3 months at KNH. All patients with no age limitation. The entry point was patients with eczema as documented in the patients file and ascertained by a dermatologist, at KNH.

9.4 Case definition

1. Endogenous eczema- any patient with diagnosis of endogenous eczema documented in the patient file and had been ascertained by a dermatologist, and was on treatment for at least three months.
2. Patients with adherence of 80% or more were classified as adherent while those with less than 80% were categorized as nonadherent, this was assessed by use of patient questionnaires and self reports.
3. Severity of endogenous eczema, this was based on scorad index: when the scorad index is below 25, eczema severity is considered to be 'mild'; when it is between 25 and 50 to be 'moderate'; and when over 50 to be 'severe'.
4. Patients were categorized as either asymptomatic or symptomatic depending on the scorad index: Asymptomatic; all patients known to have eczema who had scorad index of less than one. Symptomatic; all patients known to have eczema who have a scorad index of one or more.

9.5 Screening, recruitment and sampling.

Files of patients with eczema in the dermatology clinic, paediatric and medical wards were obtained and perused, those patients who met the case definition were identified, approached and an informed consent obtained after consent explanation.

Only those who consented were included in this study.

A study proforma was filled depending on history and clinical findings.

The primary doctors retained the overall responsibility for management of the patients recruited into this study.

All patients were seen after the primary doctor's review.

Sampling technique

Consecutive sampling was applied until the desired sample size was achieved.

9.6 Sample size

The sample size for the study was estimated using the following sample size formula:

$$n = (Z_{\alpha/2})^2 P(1-P)/d^2 = 84,$$

where: n=sample size,

$Z_{\alpha/2} = 1.96$ at 95% confidence interval,

P=prevalence .adherence level 32%

d=margin of precision of error of 0.1

9.7 Patient selection

9.7.1 Inclusion criteria

1. All patients who met the case definition.
2. All patients presenting to KNH with a documented diagnosis of eczema, and had been on follow up for at least 3 months.
3. A duly signed written informed consent.

9.7.2 Exclusion criteria

1. All patients who declined to consent.
2. All patients whose diagnosis had not been ascertained by consultant dermatologist.

3. All patients with documented history of psychiatric illness.
4. Patients who had not been put on eczema medication even though had eczema.
5. All patients with exogenous eczema like contact eczema, Photo-sensitive dermatitis and infective dermatitis.

10 DATA COLLECTION

Clinical methods and methodology

Once recruited each patient was evaluated by way of medical history and physical examination.

All the information was recorded in the study proforma administered by the PI or study assistant

The patient or caretaker was interviewed on the drug usage, this was compared with the drug prescribed in file or prescription. The actual usage and frequency was elicited by self reports, this was compared with the prescribed instructions. The self reports were compared with the prescription instructions, patients who were using the wrong drug in terms of composition or concentration were considered to be non adherent. The frequency of drug use was also compared with the prescribed frequency if this was less than eighty percent of the prescribed frequency then the patient was classified as non adherent.

Physical examination was then carried out, this included general examination and systemic examination, with bias towards skin. Looking for dry itchy scaly skin, erythema, edema, papulation (vesicles, bullae) oozing, crusting, xerosis and lichenification. The body surface area affected by eczema was calculated. Presence or absence of pruritus was ascertained from patient or care taker. Using the information obtained scorad index was calculated. The findings were recorded in the study proforma.

11 DATA MANAGEMENT AND STATISTICAL ANALYSIS

All data in this study was verified, cleaned and entered into data entry sheets.

Statistical analysis was performed data using the statistical package for social sciences (SPSS) version 15 for windows.

Analysis involved descriptive statistics such as means for continuous variables and frequency distributions for categorical variables. Comparisons for continuous data were made using the t-test and for categorical data using the chi-square test.

Proportions were obtained for categorical data:

- Proportion of study population adherent to therapy,
- proportion of study population with symptomatic eczema, mild, moderate and severe eczema.
- Adherence was analyzed as a categorical variable.

Differences were considered significant when the p-value is equal to or less than 0.05(95% CI)

The results have been presented in tables, pie charts and histograms.

12 ETHICAL CONSIDERATIONS

The study was conducted after the approval by the department of medicine and therapeutics, University of Nairobi, and the Kenyatta National Hospital Ethical and Research Committee.

A detailed written and verbal consent explanation was given to the study participants (Appendix VII).

All participants signed an informed consent form (Appendix VIII).

All patients were educated on eczema and counseled on adherence to prescribed therapy.

13 RESULTS

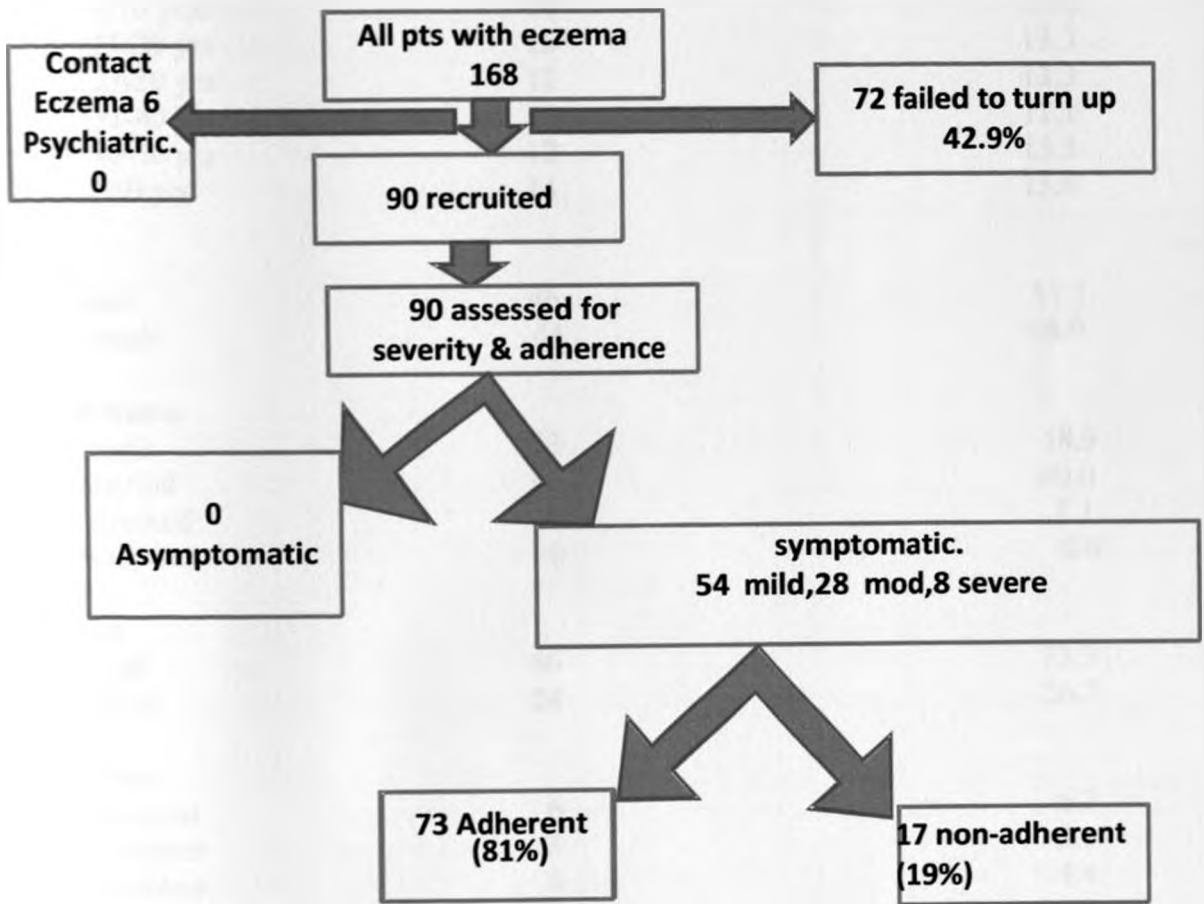
The study was conducted between April and August 2010. 168 patients who met the inclusion criteria were recruited into the study. 72 patients did not turn up for review.

Six(6) patients had contact eczema and were excluded. 90 patients were assessed for severity of eczema and level of adherence to prescribed eczema medication (figure 1).

The number of males and females was the same. The mean age was 26.9 years.

The demographic characteristics of the patient population are shown in table 1.

Figure 1: FLOW CHART ON PATIENT SCREENING AND RECRUITMENT



All patients were symptomatic implying poor control since adequately controlled eczema is usually asymptomatic.

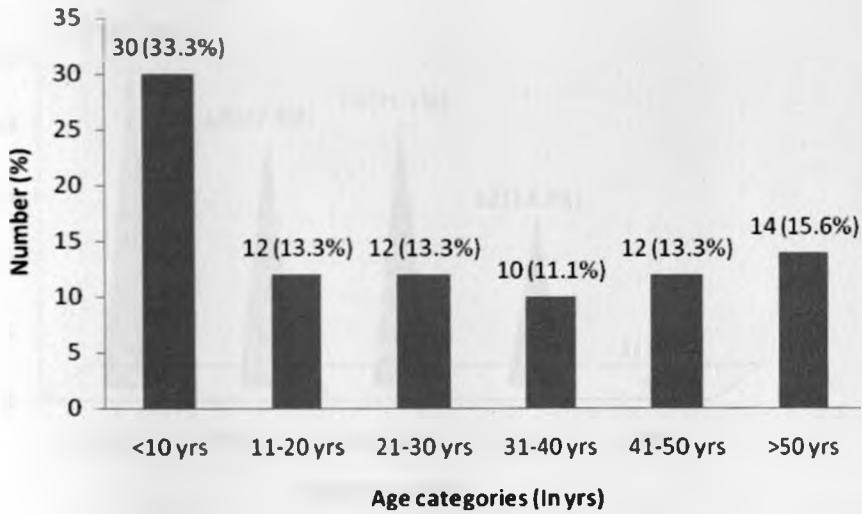
Table 1: The demographic characteristics of the patient population.

| Characteristic /Variable | Number | Percent (%) |
|----------------------------------|--------|-------------|
| 1.Age categories (In yrs) | | |
| < 10 years | 30 | 33.3 |
| 11-20 yrs | 12 | 13.3 |
| 21-30 yrs | 12 | 13.3 |
| 31-40 yrs | 10 | 11.1 |
| 41-50 yrs | 12 | 13.3 |
| >50 yrs | 14 | 15.6 |
| 2.Gender | | |
| Male | 46 | 51.1 |
| Female | 44 | 48.9 |
| 3.Marital Status | | |
| Single | 53 | 58.9 |
| Married | 36 | 40.0 |
| Divorced | 1 | 1.1 |
| Widowed | 0 | 0.0 |
| 4.Residence | | |
| Rural | 66 | 73.3 |
| Urban | 24 | 26.7 |
| 5.Occupation | | |
| Saloonist | 6 | 6.7 |
| Carpenter | 3 | 3.3 |
| Mechanic | 4 | 4.4 |
| Farmer | 20 | 22.2 |
| Shopkeeper | 4 | 4.4 |
| Teacher | 6 | 6.7 |
| Other (Child, Student etc) | 47 | 52.2 |
| 6.Education Level | | |
| None | 24 | 26.7 |
| Primary | 34 | 37.8 |
| Secondary | 19 | 21.1 |
| Tertiary | 12 | 13.3 |
| Other | 1 | 1.1 |

Table 1 continuation.

| Characteristic /Variable | Number 90 | Percent (%) |
|--|------------------|--------------------|
| 7.Average Income | | |
| < 30,000 Ksh | 11 | 12.2 |
| 30,000-200,000 Ksh | 56 | 62.2 |
| 200,001-1.8 m | 23 | 25.6 |
| >1.8m | 0 | 0.0 |
| 8.Age at which Eczema developed | | |
| Less <1yr | 32 | 35.6 |
| 1-5 yrs | 16 | 17.8 |
| 6-10 yrs | 5 | 5.6 |
| 11-20 yrs | 6 | 6.7 |
| >20 yrs | 31 | 34.4 |
| 9.Type of Eczema | | |
| Eczema (unclassified) | 23 | 25.6 |
| Atopic Eczema | 55 | 61.1 |
| Seborrheic Eczema | 3 | 3.3 |
| Stasis Eczema | 7 | 7.78 |
| Discoid Eczema | 2 | 2.22 |
| 10.Severity of Eczema | | |
| Asymptomatic | 0 | 0 |
| Mild < 25 | 54 | 60 |
| Moderate 25-50 | 28 | 31.1 |
| Severe > 50 | 8 | 8.89 |
| 11.Adherence to all prescribed drugs | | |
| Non adherence | 17 | 18.9 |
| Adherence | 73 | 81.1 |
| 12.Reasons for Non Adherence | | |
| Side effects | 2 | 2.2 |
| Cost | 2 | 2.2 |
| Worsening of Symptoms | 23 | 25.6 |
| Drug burden | 2 | 2.2 |
| Drugs do not help | 2 | 2.2 |
| 13.Mild_ None Mild Eczema (Moderate/Severe) | | |
| Mild | 36 | 40.0 |
| Mild | 54 | 60.0 |
| 14.Above_ Below 13 yrs | | |
| Above 13 yrs | 58 | 64.4 |
| Below 13 yrs | 32 | 35.6 |

Figure 2: Age



Mean Age 26.9 years (SD=21.6) Min 3 months and Max 71 yrs. Median is 24.5yrs.
This was basically a young population, the burden of eczema is more in the young people.

Figure 3: Marital Status

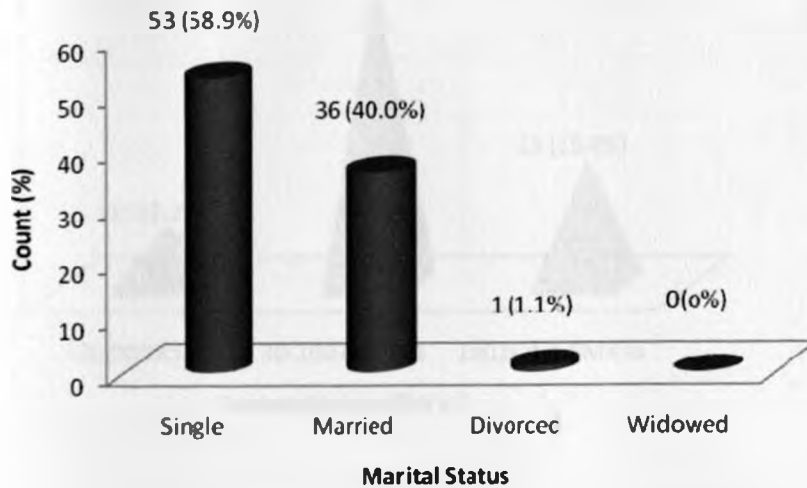


Figure 4: Education Level

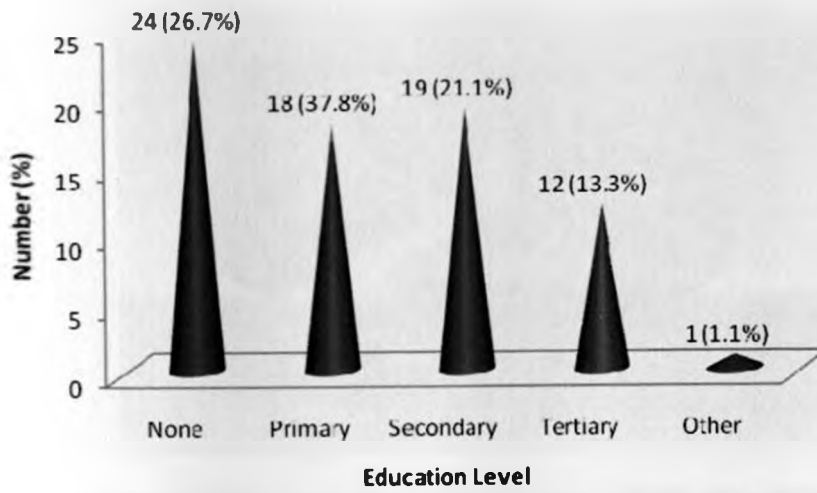


Figure 5: Average Income

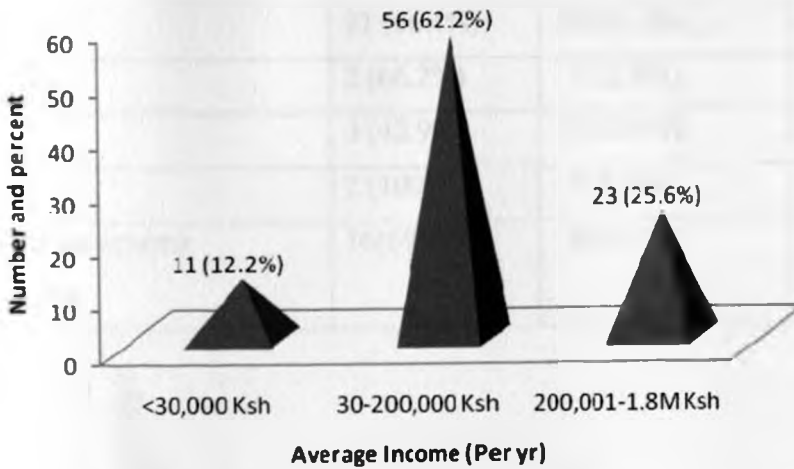


Figure 6: SEVERITY OF ECZEMA

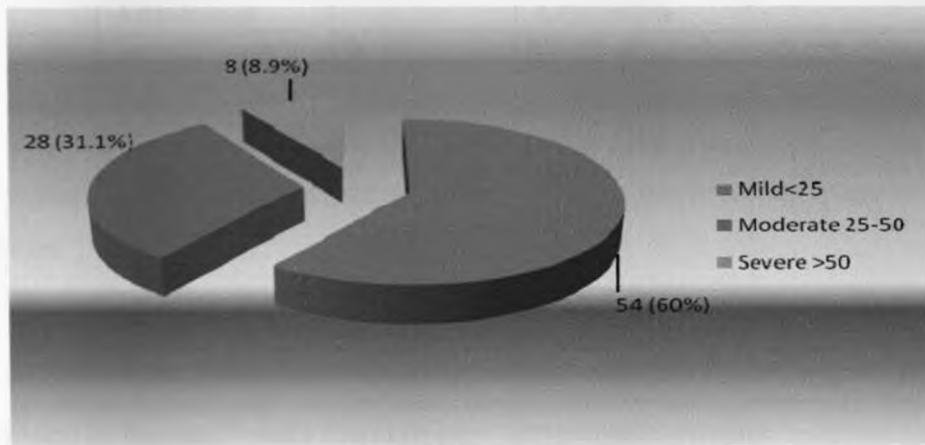


Table 1: ASSOCIATION BETWEEN ECZEMA TYPE AND SEVERITY

| Type of Eczema | SYMPTOMATIC | | |
|-------------------------------|-------------|------------|-----------|
| | Mild | Moderate | Severe |
| Atopic | 31 (56.4%) | 18 (32.7%) | 6 (10.9%) |
| Seborrheic | 2 (66.7%) | 1(33.3%) | 0 (0.0%) |
| Stasis | 3 (42.9%) | 3(42.9%) | 1(14.3%) |
| Discoid | 2 (100%) | 0 (0.0%) | 0(0.0%) |
| Endogenous eczema unspecified | 16(69.6%) | 6(26.1%) | 1 (4.3%) |

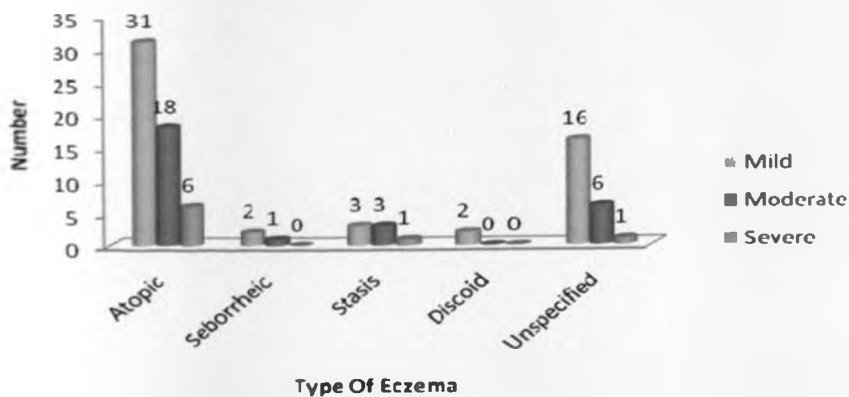


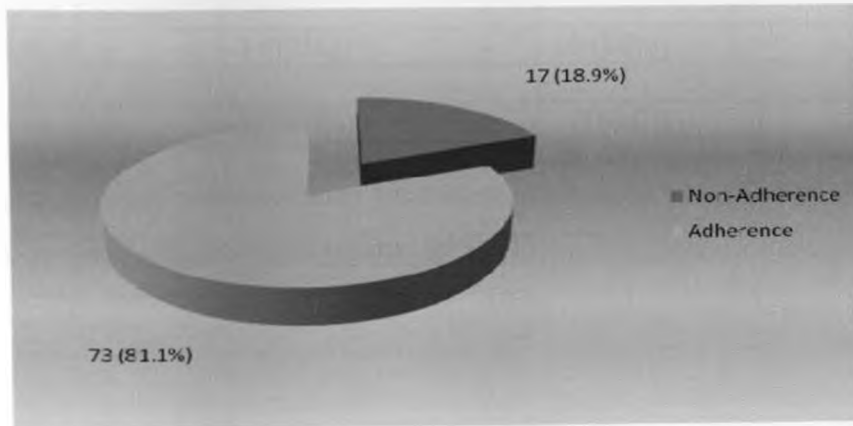
Table 2: TABLE SHOWING ECZEMA TYPES LEVEL OF ADHERENCE

| Type of Eczema | ASYMPTOMATIC | | SYMPTOMATIC | |
|--------------------------------|---------------|-----------|-------------------|-------------------|
| | Non Adherence | Adherence | Non Adherence | Adherence |
| Atopic | 55 | 0 | 12 (21.8%) | 43 (78.2%) |
| Seborrheic | 3 | 0 | 1(33.3%) | 2(66.7%) |
| Stasis | 7 | 0 | 1(14.3%) | 6(85.7%) |
| Discoid | 2 | 0 | 0(0%) | 2(100%) |
| Unspecified endogenous. eczema | 23 | 0 | 3(13%) | 20(87%) |
| TOTAL | 90 | 0 | 17 (18.9%) | 73 (81.1%) |

All patients evaluated in this study were found to have symptomatic eczema.

Fifty four patients (60%) had mild eczema, twenty eight (31.11%) had moderate eczema and eight(8.89%) had severe eczema.

Figure 7: Level of Adherence



Adherence was good across all eczema types studied with average of 81.3%(66.7-100%)

Figure 8: Eczema types versus Adherence level

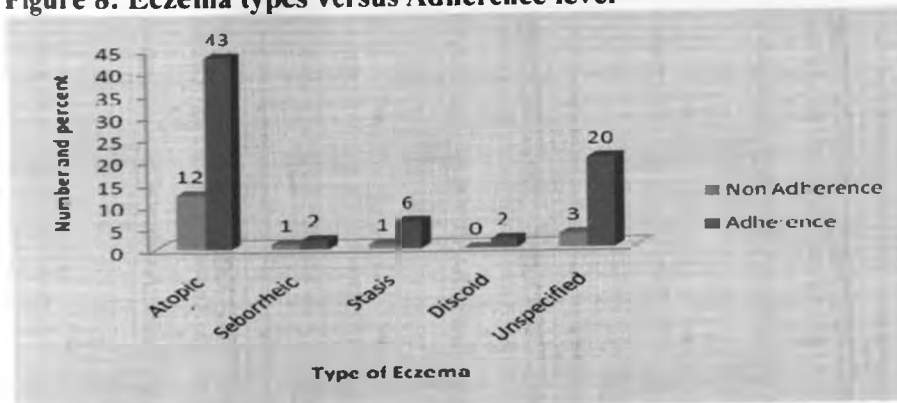


Table 3: Comparison of severity of Eczema among the Adherent and Non Adherent Groups

| Variables | Adherence to all of the prescribed drugs | | |
|---------------------------|--|-----------|--|
| | Non Adherence | Adherence | RR (95% CI,P-value) RR of non adherence |
| Severity of Eczema | | | |
| Mild <25 | 7 (12%) | 50 (88%) | 1.0 |
| Severe >50 | 2(50.0%) | 2(50.0%) | 7.14 (0.7-9.6), P=0.010 |

the patients who had severe eczema were more likely to be non adherent to medication compared to those who had mild eczema.(p=0.01)

ASSOCIATION BETWEEN SOCIO-DEMOGRAPHIC CHARACTERISTICS

ADHERENCE

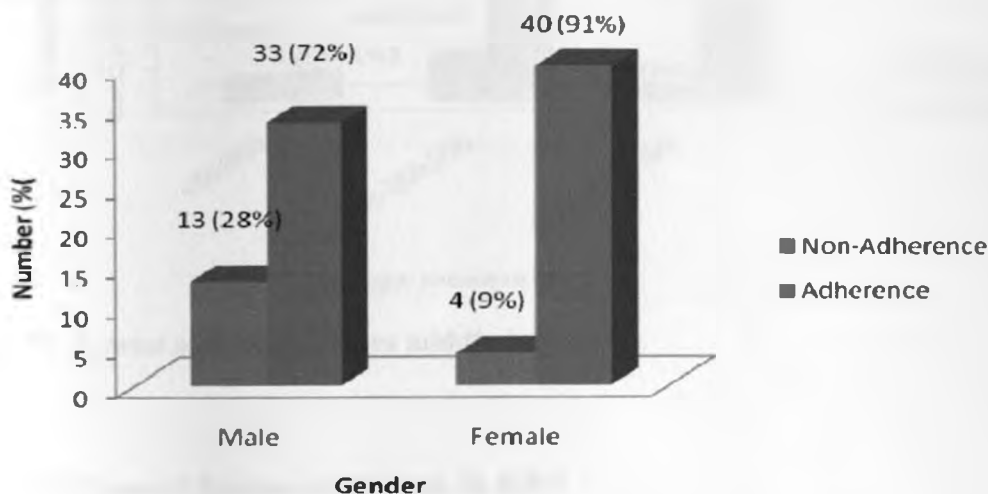
Association between characteristics and Adherence

| Variables | Adherence to all of the prescribed drugs | | |
|-----------------------------|--|-------------|--|
| | Non Adherence | Adherence | RR (95% CI,P-value) RR of non adherence |
| 1.Severity of Eczema | | | |
| Mild<25 | 7 (12%) | 50 (88%) | 1.0 |
| Moderate 25-50 | 8 (28%) | 21 (72%) | 2.7 (0.2-4.3) P=0.196 |
| Severe >50 | 2(50.0%) | 2(50.0%) | 7.14 (0.7-9.6), P=0.010 |
| 2.Gender | | | |
| Male | 13 (28%) | 33 (72%) | 1.0 |
| Female | 4 (9%) | 40 (91%) | 0.3(0.1-1.4), P=0.04 |
| 3.Marital Status | | | |
| Single | 10 (19%) | 42 (81%) | 1.0 |
| Married | 7 (19%) | 30 (81%) | 0.98(0.3-3.0), P=0.89 |
| Divorced | 0 (0.0%) | 1(100.0%) | 0.0 |
| Widowed | 0 (0.0%) | 0 (0%) | 0.0 |
| 4.Usual residence | | | |
| Urban | 3 (13%) | 21(87%) | 1.0 |
| Rural | 14 (21%) | 52 (79%) | 1.9 (1.2-5.2), P=0.47 |
| 5.Education Level | | | |
| None | 5(20.0%) | 20 (80.0%) | 1.0 |
| Primary | 8(26%) | 23 (74%) | 1.3(0.2-2.8), P=0.6 |
| Secondary | 4(19%) | 17(81%) | 0.94(0.2-6.9), P=0.81 |
| Tertiary | 0(0.0%) | 13 (100.0%) | 0.3(0.28-3.5), P=0.89 |
| 6.Age (In yrs) | | | |
| Below 13 yrs | 6(21%) | 23(79%) | 1.0 |
| Above 13 yrs | 11(18%) | 50(82%) | 0.84 (0.2-2.2), P=0.579 |
| 7.Duration of Eczema | | | |
| <6 months | 2 (0.0%) | 10 (100%) | 1.0 |
| >6 months | 15(19%) | 63(81%) | 1.2 ,(1.1-1.4), P=0.09 |
| 8.Average Income | | | |
| <30,000 Ksh | 5 (45.5%) | 6 (54.5%) | 1.0 |
| 30,000-200,000Ksh | 10 (18%) | 46 (82%) | 0.3 (0.2-1.75), P=0.05 |
| 200,001-1.8 m | 2(9%) | 21 (91.0%) | 0.11 (0.1-1.14.),P=0.01 |

Patients with lowest income were more likely to be non adherent in comparison to those with middle income($P=0.01$)

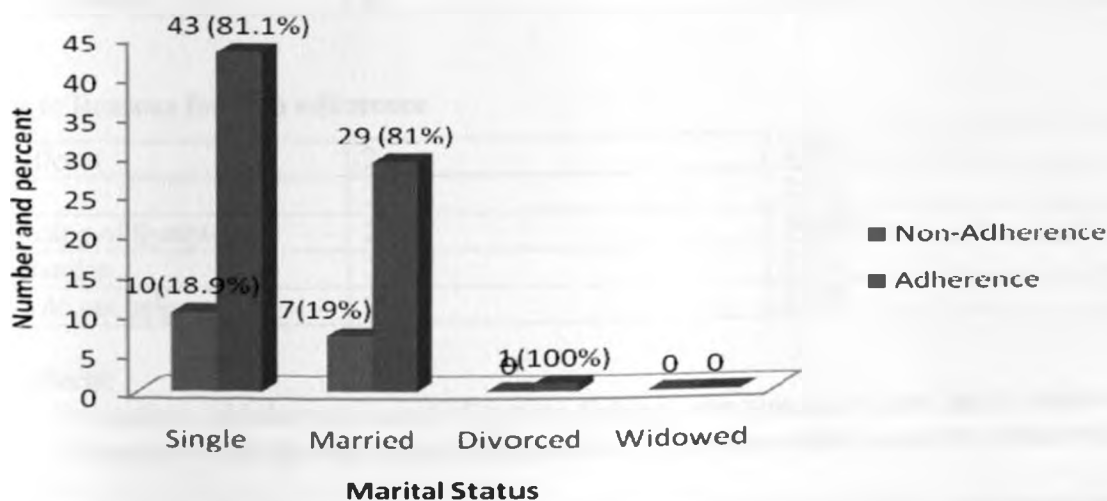
Male gender was associated with non adherence.($p=0.04$).

Table 4: Gender Vs level of Adherence



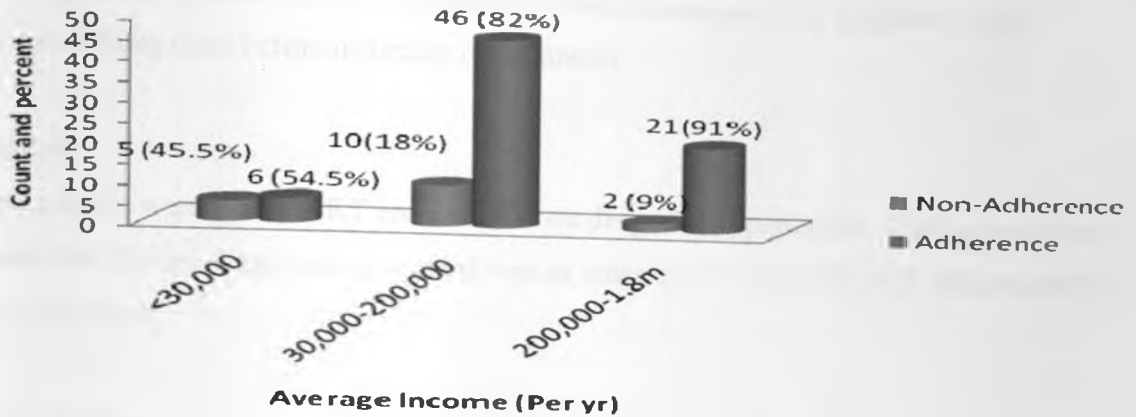
$P=0.04$

Figure 9: Marital Status Vs level of Adherence



$P=0.89$

Figure 10: Average Income Vs level of Adherence.



P=0.01. Lowest socioeconomic vs middle income

Table 5: Types of Eczema in Patients in KNH

| | | |
|----------------------------------|----|---------|
| Endogenous eczema (unclassified) | 23 | 25.6.0% |
| Atopic Eczema | 55 | 61.1% |
| Seborheic Eczema | 3 | 3.3% |
| Stasis Eczema | 7 | 7.78% |
| Discoïd Eczema | 2 | 2.22% |

Table 6: Reasons for Non adherence

| | | |
|-----------------------|----|-------|
| Side effects | 2 | 2.1% |
| Cost | 2 | 2.1% |
| Worsening of Symptoms | 23 | 24.0% |
| Drug burden | 2 | 2.1% |
| Drugs do not help | 2 | 2.1% |

Side effects:

1. One patient said she was scared of getting diabetes, she was told by her family doctor that the steroid cream she was using (betamethasone) can cause diabetes so she opted to stop it.
2. Another patient reported that her skin was becoming lighter and thinner, where she had been applying methylprednisolone cream, and for that reason she stopped using the medicine.

Worsening of symptoms

1. Ten patients reported no improvement after using the prescribed medication, although clinical assessment revealed improvement.
2. Seven patients reported that, the disease worsened on stopping the medication, they reported more itching than before initiation of treatment.

Drug burden

1. Two patients were on HAART and one was on drugs for hypertension. Both reported that, at times they felt the drugs were too many and at times opted to use HAART and omitted the drugs for eczema.

Drugs do not help

1. Two patients on treatment for atopic eczema with hydrocortine cream and Cetrizine felt there was no reduction in itchiness and skin rashes and hence stopped treatment.

Table 7: List of drugs used

| Drug | No. patients Using | Drug | No. patients using |
|------------------------------|--------------------|---------------------------|--------------------|
| Betamethasone cream | 18 | Piriton(chlorpheniramine) | 6 |
| Hydrocortisone cream | 14 | Methotrexate | 5 |
| Diprosalic | 13 | Dapsone | 3 |
| Mometasone | 12 | Flucloxacillin | 3 |
| Advantan(methylprednisolone) | 11 | Efavirenz | 3 |
| Fuscort, | 10 | Combivir(AZT/3TC) | 3 |
| Emulsifiers | 10 | Nifedipine | 2 |
| Aterax(hydroxyzine) | 10 | Enalapril | 2 |
| Cetrizine | 9 | Methyldopa | 1 |
| Prednisone tabs | 7 | Bromazepam(lexotanil) | 1 |
| Tacrovate | 6 | | |

4 DISCUSSION

This study was carried out at a tertiary referral hospital. The clinics and wards are run by consultant dermatologists and medical and pediatric residents.

The mean age of the study population was 26.9 years, this was a young population, this was expected as most of the patients presenting with eczema have atopic dermatitis, affects young people more. After the age of 20 years atopic dermatitis has a remission rate between 26 to 84 percent (87).

The male to female ratio was 1:1.

Majority of studies have reported higher prevalence in females up to 1:1.3(1). Edan Al-saimary et al in study done in Iraq reported female preponderance 1:13.

Fifty-five point two (55.2) percent of the patients were single this is because of higher prevalence of atopic eczema in the young (87).

Twenty patients were farmers(22.9) percent, this could be due high number of farmers in Kenya since no study locally nor globally which has found high incidence of endogenous eczema in farmers, however, higher incidence has been reported in urban dwellers(88).8.3 percent this can be due to high number of teachers in the country,since there is no published data on eczema in teachers.

Seven point three (7.3) percent were saloonists, high incidence of eczema has been reported in hair dressers and barbers in several studies(89,)this has been attributed to several chemical used in beauty industry eg *p*-benzenediamine, *o*-benzenediamine, *p*-phenylenediamine, *p*-toluenediamine, toluene-2,5-diamine. *p*-aminophenol, *m*-aminophenol, parabens, formaldehyde, methylisothiazolinones, ethanol, isopropanol, ammonia, phenols, alcohols, persulphates monoethanolamine, glyceryl monothioglycolate, ammonium thioglycolate, ammonium chloride or ammonium phosphate, hydrogen peroxide, formaldehyde, carbon dioxide, and carbon monoxide(90).

70.8 percent of patients were from rural areas this is in keeping with Kenyan population where upto 85 percent live in rural areas.

The high percentage of urban dwellers, 29.2 percent, is due to higher prevalence of eczema in urban areas (88) and possibly, the proximity to KNH.

ECZEMA CONTROL

All the patients evaluated in this study had symptomatic eczema, which means inadequately controlled eczema.

This may be attributed to the health seeking habits where patients who were free of eczema symptoms did not attend the clinic. Hence denied us the chance to capture the patients with asymptomatic eczema which implies, adequate eczema control. 72 patients which represents 42.86% of the patients scheduled for review did not turn up.

Eczema usually has a cycling relapsing and remitting course, most patients (56-92%) report a seasonal variation with 48-65% of patients worsening during winter and similar proportion reporting improvement during summer (86).

Most of the patients had mild eczema 54(60%), moderate 28(31.1%),severe 8(8.89%).

ADHERENCE

In this study we found high levels of adherence to eczema medication, with 81.1 percent of the study population adherent to medication.

This is similar to what Cramer and Rosenheck found , among patients with physical disorders, the mean rate of medication adherence was 76 percent (range 40-90 percent), whereas among patients with psychoses the mean rate was 58 percent(range 24-90 percent) and those with depression the mean rate was 65 percent range 58 to 90 percent.

In this study patients with psychiatric illness were excluded.

A study done in the U.S.A North Carolina reported adherence of 32 percent(78).

Other studies have reported adherence to topical dermatological treatment being as poor as 32-61 percent for different diseases(59). Comparing adherence in those under 13 years of age and those above 13 years it was found that there was no statistically significant difference in adherence between adults and children($p=0.58$).

Rates of adherence to medication regimens among children with chronic diseases are similar to those among adults with chronic diseases(60).

The clear predictors of non adherence in our population are low socio economic status($p= 0.01$) and male gender($p=0.04$).

Race, sex, and socioeconomic status have not been consistently associated with levels of adherence(61).this was based on a study done in ,USA where medication was provided free,

hence the impact of poverty may not have been evident unlike our set up where patients have to buy drugs.

COMPARISON OF LEVEL OF ECZEMA CONTROL AMONG THE ADHERENT AND NON ADHERENT GROUPS

The patients who had severe eczema were more likely to be non adherent to medication compared to those who had mild eczema.($p=0.01$).

This shows that adherence is an important determinant of disease severity and control.

The contribution of poor adherence to poor control has been reported in most chronic diseases, it has even lead to “drug failure”.

Non adherence is positively related to severity of disease(42) and hence low QALY and negative psychological effects, hence physicians should spend time and emphasize the importance of adherence to patients.

Witkowski JA et al reported that only 25% of doctors discussed adherence with patients, he also showed that a cordial relationship the physician and frequent reviews enhanced adherence.

REASONS FOR NON ADHERENCE

Reasons for non adherence in our population included side effects of drugs, cost of drugs, worsening of symptoms, drug burden and feeling that the drugs do not help. Many patients felt that, the medication they were taking was not helping, this can be attributed to the fact that most patients do not get the gold standard drugs for their eczema because the clinicians most often will prescribe what is available in the hospital or what the patient can afford hence, even with good adherence the eczema control may be poor and hence the feeling that the medication were not helping.

Many patients were on topical steroids and attributed skin color changes to the drugs, other patients had been informed of possible side effects, this was good information, but some patients got scared and hence non adherent and poor control, steroid phobia has been reported(72).

TYPES OF ECZEMA

Most patients had atopic eczema, contributing 60 percent of the study population, this is the commonest type of eczema world wide.

Approximately 10% to 20% of the world's population develops atopic dermatitis(79,80).The study population was young with mean age of 26.9 years and median of 24.5 years, most young people have atopic eczema.

Stasis eczema accounted for 7.78 percent two of the patients with stasis eczema had hypertension and two had lower limb varicose veins.

3.33percent had seborrheic eczema and were two of them were on HAART,discoid eczema accounted for 2.22 percent.

All types of eczema were poorly controlled and symptomatic.

15 CONCLUSIONS

1. All patients in this study population had poor eczema control,since they were all symptomatic.
2. Most of the patients were adherent to their medication.
3. The patients with severe eczema were more non-adherent to their medication.
4. Male gender was significantly associated with non-adherence
5. Low socioeconomic status was associated with poor adherence to medication.
6. Reasons for non adherent were,worsening of symptoms,cost of drugs,side effects to medications and drug burden.

LIMITATIONS

Patient self report may overestimate level of adherence; hence the high adherence in this study.

16 **RECOMMENDATIONS**

1. Patients need to be encouraged to come to clinic for review.
2. A further study needs to be carried out to find other causes of inadequate control of endogenous eczema.
3. This study revealed many issues that lead to non adherence, a further study needs to be carried out to find out the importance of these issues in our population.
4. Adherence to medication need to be emphasized in patients with severe endogenous eczema and male patients.
5. A study needs to be carried to find out the reasons for non adherence in male patients and patients with severe endogenous eczema.

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APPENDICES

PPENDIX 1: INTERNATIONAL CLASSIFICATION OF ECZEMA

L20. Atopic dermatitis (a type of eczema) is an inflammatory, chronically relapsing, non-contagious and pruritic skin disease. It has been given names like "prurigo Besnier," "neurodermitis," "endogenous eczema," "flexural eczema," "infantile eczema," and "prurigo diathsique.

L21. Seborrhoeic dermatitis is a skin disorder affecting the scalp, face, and trunk causing scaly, flaky, itchy, red skin. It particularly affects the sebum-gland rich areas of skin.

L22. Diaper rash or nappy rash, (also known as "Diaper dermatitis" and "Napkin dermatitis) is a generic term applied to skin rashes in the diaper area that are caused by various skin disorders and/or irritants.

Contact dermatitis or Irritant dermatitis is a term for a skin reaction resulting from exposure to allergens (**L23. allergic contact dermatitis**) or irritants (**L24. irritant contact dermatitis**). Phototoxic dermatitis occurs when the allergen or irritant is activated by sunlight.

L28. Lichen simplex chronicus (LSC) is a skin disorder characterized by chronic itching and scratching. The constant scratching causes thickening of the skin.

L28.1 Prurigo nodularis (PN) is a skin disease characterised by pruritic nodules which usually appear on the arms or legs. Patients often present with multiple excoriated lesions caused by scratching.

L30. Nummular dermatitis (also known as "Nummular eczema," and "Nummular neurodermatitis") is one of the many forms of dermatitis. Also known as discoid dermatitis, it is characterized by round or oval-shaped itchy lesions.

L30.0 Dyshidrotic eczema, Pompholyx, and "Podopompholyx" is a skin condition that is characterized by small blisters on the hands or feet. It is an acute, chronic, or recurrent dermatosis of the fingers, palms, and soles, characterized by a sudden onset of many deep-seated pruritic clear vesicles later, scaling, fissures and lichenification occur.

L83.1 Stasis (gravitational, venous eczema) Stasis dermatitis is a common inflammatory skin disease that occurs on the lower extremities in patients with chronic venous insufficiency with venous hypertension.

L30.8 Asteatotic (winter, craquele, senile) eczema is characterized by pruritic, dry, cracked, and polygonally fissured skin with irregular scaling. It most commonly occurs on the shins of elderly patients, but it may occur on the hands and the trunk.

APPENDIX II: WORLD BANK (1996) PER CAPITA INCOME

Kenya was ranked 17th poorest country in the world with per capita income of 250 USD.

Low income from 380-2520 USD(30,000-200,000)

Middle income from 2520-23520 USD

High income more than 23520 USD(KSH 1.84 million)

APPENDIX III: SCORAD

Extent criteria

Body surface area involved.

Intensity criteria

| | | |
|---------------------------|---|----------------------------|
| <u>Erythematic</u> | : | stage 1/ stage 2/ stage 3 |
| <u>Edema / population</u> | : | stage 1/ stage 2/ stage 3 |
| <u>Oozing / crusting</u> | : | stage 1 / stage 2/ stage 3 |
| <u>Excoriation</u> | : | stage 1 / stage 2/ stage 3 |
| <u>Lichenification</u> | : | stage 1 / stage 2/ stage 3 |
| <u>xerosis</u> | : | stage 1 /stage 2 / stage 3 |

C. Subjective, Symptoms

The two most representative items concerning the quality of life of patients are:

g. Pruritus

h. Insomnia

How is it calculated

a. **A = SPREAD.../100**

b. **B = INTENSITY.../18**

c. **C = SUBJECTIVE SYMPTOMS.../20**

d. **SCORAD calculation: $A/5 + 7.B/2 + C/10$**

APPENDIX IV: PHOTOGRAPHS OF PATIENTS WITH ECZEMA

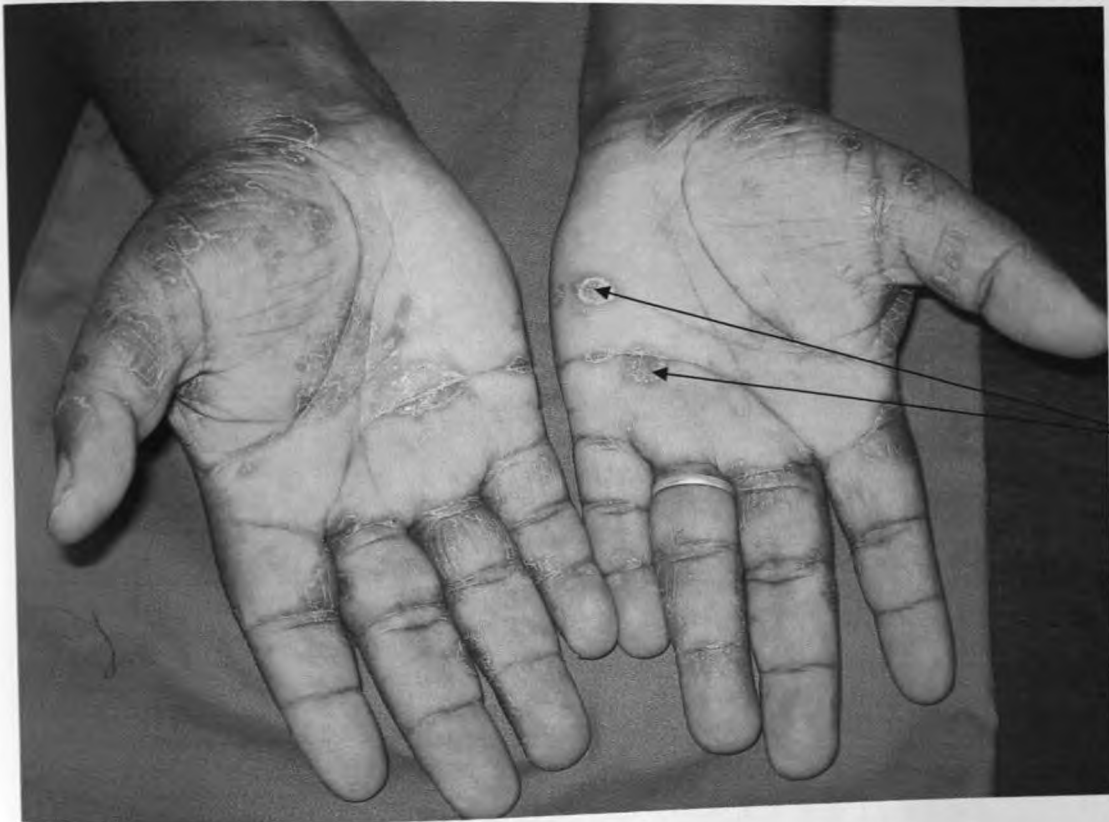
A. Acute eczema; characterized by; 1pupules,2erythema,3 edema 4 exudation.



B. Chronic eczema; characterized by; 1 excoriations, 2 lichenification



C. Sub acute, Dyshydrotic (pompholyx, palmoplantar) eczema characterized by
Scaling & fissuring and xerosis



D. Secondary infected eczema; 1.pus,exudation,2.edema.



1

2

1
1

APPENDIX V: STUDY PROFORMA

Date

Study number.....

Outpatient/inpatient number.....

Age.....

DEMOGRAPHICS

Gender 1...M/2...F

Marital status

1...single

2...married

3...divorced/separated

4...widowed

3 Usual residences

1...rural

2...urban(3 or more months stay in urban area)

4 What is your occupation? Specific occupations

Saloonist, carpenter, mechanic, farmer, shopkeeper, teacher, medical doctor, nurse etc.....

5 Level of formal education

1...none 2...primary 3...secondary. 4 tertiary 5...others specify

6 What is your average income per year? (A) below 30,000ksh,(B) 30-200,000ksh,(C) 200,001-1.8m ksh,(D) Above 1.8m.

7 Do you have any family member who has eczema? 1 Yes 2 No.

8 If yes what is the relationship?.....

9 At what age did you develop eczema? (A) Since birth,(B)1-5 yrs.(C)6-10yrs(D)11-20(E)Over 20yrs.

10 Do you have eczema? YES OR NO.....

11 Do you have allergic rhinitis? YES OR NO.....

ADHERENCE QUESTIONNAIRE

1 Have you ever been explained to what your disease is? Yes or no.....

2 If yes what is it?..... In addition, by whom?.....

3 Type of eczema as documented in patients file.....

5 Duration of eczema (months/years).....

6 Have ever used any non-prescribed medication for this illness? Yes or no.....

If yes which ones ?.....

7. Do you have any other disease for which you have take medication? Yes or no.....

If yes which condition/disease.....

8. Who buys your medication. 1 self 2 Employer/insurance. 3 parent. 4 child 5 other(specify).....

9 Where do you buy/get your drugs from? 1 KNH, 2 outside pharmacy, 3 others.

| prescribed drug | Drug used(a) | Instructed frequency | Frequency of actual use(b) | Prescribed amount & concentration | Used drug amount&conc (c) | Adherence % |
|-----------------|--------------|----------------------|----------------------------|-----------------------------------|---------------------------|-------------|
| | | | | | | |
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11 If any response in column a,b or c is not 100% adherence.
 We understand that there are times when one is no able to take their drugs for various reasons.
 What are some of the reasons for not having taken your drugs or given your child from time to time?

Probes

- Side effects.
- Cost
- Availability
- Worsening of symptoms
- Relief of symptoms
- Drug burden
- Drugs do not help.
- I did not understand the doctor.
- Others

12. Severity of eczema by use of SCORAD score.(1) mild <25 (2).moderate 25-50.(3) severe >50

13 Area of the body affected. 1 face, 2 limbs flexor areas, 3 limbs extensor areas.4 trunk .

14 what do you think makes your eczema worsen?1 stress 2 exposure to chemicals,3 I don't know 4 others

APPENDIX VI: A CONSENT EXPLANATION FORM

I am Dr Musyoka David Ndonge, a medical doctor in postgraduate training at the University of Nairobi. As part of my training, I am required to carry out a postgraduate research project. My project is on impact of adherence on control of eczema among patients attending KNH. I am required to recruit patients with this condition into this study, and I have identified you as a potential recruit.

However, before recruiting you to participate I request to give you the details of this research work. Eczema is a skin condition that affects the skin. Another name for eczema is dermatitis. The diagnosis of eczema is made from history and clinical findings, where the diagnosis is not clear a skin biopsy is done.

This will involve asking questions to get history of your condition, drug use, adherence and a physical examination to look for evidence of adequacy of control. If any question or examination causes distress it is your right to decline, these questions and examinations are however necessary to establish adequacy of control, drug adherence and reasons for non-adherence.

Except for discomfort during physical examination. No harm will be visited upon you.

Your participation will yield information that will increase knowledge and skill in management of patients with Eczema in future in our setting.

You are now free to ask any questions for clarification.

All information relating to your illness will be kept confidential. Your routine care provider, will be informed of my findings at all stages of this study.

You will not be paid to take part in this study.

If you decline to take part in this study, you will not be discriminated in any way, and will still get complete and quality care.

You will be free to withdraw from the study any time, and this will not influence your access to quality health care.

My contact is Tel 0722750037; and P.O. Box 424 00202, Nairobi.

You may also contact the Ethics Committee through: Prof.A.N.Guantai; Tel 020-2726300 ext 44355.

Thank you for your co-operation and participation.

B INVESTIGATOR'S STATEMENT:

As the principal investigator, I confirm that I have adequately explained to this patient/caretaker all the details of this study, and given them the opportunity to ask questions, which have been adequately answered.

Signed:.....

Date:.....

APPENDIX VII: CONSENT FORM

Name.....

Age.....

Serial number.....

I, named above, after a well-understood explanation from Dr Musyoka David Ndonge, and having sought and obtained all clarification, do willingly accept to take part in the study he is conducting on impact of adherence to medication on eczema control. I further accept to have all necessary examination done to me.

The benefits of my participation have been clearly explained to me, and I have understood well.

I understand that I am free to join or not to join this study, and will either way be accorded the best possible medical care. I know that I may withdraw from the study any time if do so wish without jeopardizing access to and quality of medical care accorded to me.

With this understanding, I on my own free will accept to take part in this study.

Sign

Relationship to patient.....

Dated

Contact: 1. Dr Musyoka David; Tel 0722750037; P.O. Box 424 00202 Nairobi

2.ECRC c/o Prof. A.N.Guantai; Tel. 020-2726300 ext 44355.

APPENDIX VIII: IDHINI YA KUSHIRIKISHWA KWA UTAFITI

Jina

Jmri.....

Nambari

Mimi,mwenye jina lililoko hapo juu,baada ya kuelezewa vyema na Dkt.Musyoka David Ndonge.kwa hiari yangu nakubali kuhusishwa katika utafiti anaofanya kuhusu maradhi ya ngozi.Nakubali kufanyiwa uchunguzi wowote ambao nimeelezewa kwa kina.

Nakubali kwamba nimefafanuliwa kikamilifu manufaa ya kushiriki kwangu.na kuelewa barabara.

Naelewa kwamba niko huru kushiriki au kutoshiriki katika utafiti huu,na sitanyimwa haki yangu ya huduma ya matibabu kwa misingi ya uamuzi wangu.

Niko huru kujiondoa kwa utafiti huu wakati wowote pasi adhabu yeyote.

Nikielewa haya yote,na kwa hiari yangu,najitolea kushirikishwa kwa utafiti huu.

Saini

Uhusiano na mgonjwa.....

Tarehe

Anwani : 1.Dkt Musyoka David.N. Simu :0722750037.S.L.P. 424 00202,Nairobi.

2.ECRC,c/o Prof.A.N:Guantai;Simu:020-2726300 ext 44355.

School Of Nursing Sciences

College Of Health Sciences

University Of Nairobi

**Topic: *Factors Affecting Provision Of Oral Care By Nurses In
The Intensive Care Unit At The Kenyatta National Hospital.***

**Thesis Submitted In Partial Fulfillment For The Award Of The
Degree Of Master of Science In Nursing (Critical Care)**

**By:
Dorcas W. Maina
H56/P/7933/05**

October 2007

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Declaration

I declare that this is my original work and has not been presented to any other training institution.

Dorcas Maina


H56/P/7933/05

Signature *Dorcas*

Date 22/10/07

Supervisors


This thesis has been submitted with the approval of my supervisors:

Signature  Date 22nd August 2007

Mrs. Theresa Odera

KRN, KRM, CCN, PG, MSc

Lecturer School of Nursing Sciences - University of Nairobi

Signature  Date 22/8/2007

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School of Nursing Sciences - University of Nairobi

Signature  Date 22nd AUGUST 2007.

Dr. David Misango

MB.ChB. M.Med (Anesthesia), Intensivist

Consultant Kenyatta National Hospital

Dedication

I dedicate this research work to my family for their constant encouragement, support and inspiration while undertaking this study.

Acknowledgements

I wish to acknowledge the help of my supervisors Mrs. T. Odero, Dr. D. Misango, and Dr. Anna Karani in development of this thesis.

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List of Abbreviations

BScN - Bachelor of Science in Nursing.

HDU - High Dependency Unit.

ICN¹ - International Council of Nurses.

ICN² – Intensive Care Nurse.

ICU - Intensive Care Unit.

KNH - Kenyatta National Hospital.

KMTC - Kenya Medical Training College

KRCHN - Kenya Registered Community Health Nurse.

MScN -Master of Science in Nursing.

VAP -Ventilator Associated pneumonia.

NB. ICN¹ - Appears first in the literature.

Operational Definitions.

Critical care nursing: That specialty within nursing that deals specifically with human responses to life-threatening problems. A critical care nurse is a licensed professional nurse who is responsible for ensuring that acutely and critically ill patients and their families receive optimal care.

Intensive care: To provide care for severely ill patients with potentially reversible conditions or to provide care for patients who require close observation and/or specialized treatments that cannot be provided in the general ward.

Intensive care unit (ICU): Refers to Intensive Care unit and High Dependency Unit at the Kenyatta National Hospital.

Oral care: Oral care/hygiene is the practice of keeping the mouth clean and healthy by brushing and flossing to prevent tooth decay and gum disease.

Nursing: Nursing is an applied science, which has a unique body of knowledge that utilizes principles from the physical, biological, and behavioral sciences. The central concern of nursing is the holistic person. The focus of nursing is health promotion, maintenance, curative, restorative, supportive and terminal care to individuals and groups of all ages, taking into consideration the factors that influence them in the total environment (The ICN¹ Definition of Nursing).

Quality of oral care: Based on each techniques relative ability to maintain a hygienic oral cavity and lower the risk of infection (Day 1993, Kite & Pearson 1995, Pearson 1996, Fitch et al; 1999; Stiefel et al. 2000; Evans 2001, Pearson & Hutton 2002, Schelder & Lloyd 2002).

Abstract

Background: Intensive care unit (ICU) patients have complex oral care needs. Inadequate oral care may predispose ICU patients to nosocomial infections. Recent initiatives have sought to improve the quality and evidence base of ICU oral care provision.

Objectives: To identify factors that affect the quality of oral care in the ICU at KNH.

Methods: This was a descriptive cross-sectional survey. A total of 100 nurses working in the ICU were asked to participate in the study. 80 agreed to participate with 75 returning properly filled questionnaires giving a response rate of 94%. The study took 6 months from the development of the proposal to final presentation.

Results: The Bivariate correlation shows that nurses' oral care education, having sufficient time to provide care and not viewing oral care as an unpleasant task had direct effects on the quality of care provided.

Conclusion: Improving the quality of oral care in intensive care unit is a multi-layered task. Reinforcing proper oral care in education programmes, desensitizing nurses to the often perceived unpleasantness of cleaning oral cavity, and working with hospital managers to allow sufficient time to attend to oral care are recommended.

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Chapter One: Introduction

1.1 Background information

Patients in the Intensive Care Unit (ICU) have very specific care needs, demanding the highest standard of professional care. Frequently, the life saving nature of the ICU means the patient's oral care takes low priority. A patient's individual requirements for oral care should be taken into consideration as part of the admission assessment (Jenkins1989). Usually nursing care for critically ill patients includes some type of oral care, as the nurse has the responsibility for assessing, planning, implementing, and evaluating each patient's oral care needs. However, this does not always occur. Oral hygiene practices performed by nurses have been reported to be inconsistent and highly variable (Treloar and Stechmiller1995).

ICU patients may require oral intubation to maintain a patent airway. However, it has been reported that an endotracheal tube can induce potential complications for a patient (Barnason, Graham and Wild 1998). Patients in our critical care units are unable to maintain their normal oral hygiene routine. After a patient has been intubated for several days without an adequate oral hygiene routine, the bacterial flora in the mouth can change resulting in predisposition to oral infections and gum diseases

1.2 Problem statement

Care of the mouth is an important nursing procedure and should be performed as part of the routine general hygiene of a patient. Nurses play an important role in providing effective oral care and promoting oral hygiene. However, oral hygiene has often been overlooked and performed on an ad hoc basis. In some instances, it has become a ritualistic and banal activity. Sporadic research has generated conflicting advice. Furthermore, it was reported that the delivery of oral care within institutional settings is fragmented (Roberts, 2001).

Hixson, Sole & King. 2000 noted that even though oral hygiene is considered standard nursing it is often neglected in critically ill patients or performed quickly by swabbing the mouth. The lack of published protocols for oral care in intubated patients has been noted in the nursing literature.

Comprehensive oral care is an evidence-based prevention strategy to reduce the risk of ventilator-associated pneumonia in patients receiving mechanical ventilation. Until recently, no comprehensive guidelines or standards existed to define necessary tasks, methods, and frequency of oral care to provide patients with optimal results.

Despite the presence of essential tools that can be used for oral assessment in ICUs, they are often not used. This may be due to lack of time or knowledge on the part of the bedside nurse as well as the lack of assistance for nurses in identifying particular problems such as Candida or herpes simplex infections (Porter 1994).

No research has been done in Kenya regarding oral care in intubated patients. The purpose of the study was therefore to describe factors associated with providing oral care in ICU at the Kenyatta National Hospital (KNH).

1.3 Justification

The study of Fitch, Munro and Glass (1999) provides some weak evidence that a mouth care protocol incorporating brushing of the teeth, gums and tongue confers some benefit in reducing inflammation of the oral mucosa. Such practices may also promote greater patient comfort and reduce the risk of VAP, but these outcomes have not been studied. Unfortunately, although the importance of oral hygiene has been stressed (McNeil 2000; Howarth 1977) little has been done about the effects of oral care interventions in critically ill patients. The lack of published protocols ensures that there is no uniformity in the way oral care is given to patients. It can, therefore, be suggested that oral care is an important component of intensive care nursing that is often ignored due to various factors.

Though little has been done in Kenya, research has been done elsewhere and shows the usefulness of oral care (Allen, Binkley, McCurren & Carrico 2004, Abidia 2007) and the multifaceted nature of factors affecting its outcome. This study sought to identify factors that affect the provision of oral care. The results can form a basis for further research to address these factors and help to standardize the care given to all patients.

1.4 Research questions

1. Is the quality of oral care affected by nurse factors?
2. Does the level of education of the nurse affect the quality of oral care provided?

1.5 Objectives

1.5.1 Broad objective

To investigate factors that affect the provision of quality oral care by nurses in the ICU at KNH.

1.5.2 Specific objectives

1. To determine the nurses' education and professional background.
2. To document the frequency of oral nursing care provision in the ICU at KNH.
3. To identify the resources available for oral care in the ICU at the KNH.
4. To determine the nurses' perception of the hospital's support in provision of oral care.

1.6 Hypotheses

Six research hypotheses in line with the objectives were postulated and tested:

1. Experience of nurses has no influence on the quality of oral care.
2. The level of education has no influence on quality of oral care
3. Availability of supplies does not affect the quality of oral care.
4. Availability of time has no effect on the outcome of oral care.
5. The priority given to oral care does not affect its quality.
6. The level of unpleasantness of providing oral care has no effect on quality of oral care.

Chapter Two: Literature Review

2.1 Introduction

Oral health is influenced by oral microbial floras, which are concentrated in dental plaque (Mojon 2002). Dental plaque provides a microhabitat for organisms and an opportunity for adherence of the organisms to either the tooth surface or other microorganisms. In critically ill patients, potential pathogens can be cultured from the oral cavity. These microorganisms in the mouth can translocate and colonize the lung, resulting in ventilator-associated pneumonia (Mehta & Niederman 2002). The importance of oral care in the intensive care unit has been noted in the literature, but little research is available on mechanical or pharmacological approaches to reducing oral microbial flora via oral care in critically ill adults. Most research in oral care has been directed toward patients' comfort; the microbiological and physiological effects of tooth brushing in the intensive care unit have not been reported. In addition, no evaluation of the effectiveness of pharmacological and mechanical interventions relative to each other or in combination has been published. Additional studies are needed to develop and test best practices for oral care for critically ill patients.

2.2 Problems Associated with Oral Intubation

With severe illnesses the bacterial strains naturally present in the mouth can shift from being predominately gram-positive normal flora to anaerobic gram-negative strains. (Treola and Stechmiller 1995, Abele Hom, Dauber & Baunerfeind 1997). Because organisms that colonize in the mouths of critically ill patients are virulent compared

colonize in the mouths of critically ill patients are virulent compared with organisms the mouths of healthy individuals, the potential for infection is increased (Jenkins 1989). Critically ill patients also have impaired immunological deficiencies and may be unable to respond to bacterial invasion of the lungs (Treola and Stechmiller 1995). Pathogens commonly responsible for nosocomial pneumonia in ICU patients were found to colonize in the dental plaque and oral mucosa of these patients (Scannapieco and Stewart 1992). Therefore, good oral hygiene measures may prevent the spread of infection from the oral cavity to the lower respiratory tract (McNeil 2000).

Assessment of the oropharynx and maintaining a favorable level of hygiene are difficult tasks to perform in both critically ill and intubated patients due to lack of access to the oral cavity (Liwu 1990). The orally intubated patient is at an even greater risk of colonization of organisms because mouth care is often hampered by the presence of tape, tubes, and bite blocks (Treola and Stechmiller 1995). The oral endotracheal tube is required for ventilation and airway protection, but the position of the tube and any anchoring devices may obscure the view of the oral cavity and limit access, which hinders the actual process of cleaning (McNeil 2000). Dislodging or displacing the endotracheal tube may be life threatening (Treola and Stechmiller 1995). As a result, nurses are often reluctant to manipulate the tube for oral assessment and hygiene measures. In turn, fixation tapes quickly become heavily contaminated with pathogens in the presence of salivary disturbances and the difficulties associated with cleansing the mouth (Hayes and Jones, 1995). Oral

assessment and care of the mouth is even more difficult in patients requiring prolonged intubation (Jenkins 1989).

Another aspect of maintaining the oral health of intubated patients is the impact of the use of drugs or procedures required to treat their medical condition that may have a detrimental effect on the oral cavity (McNeil, 2000). An example is drugs that cause xerostomia (Horwood, 1990). Intubated patients are forced to keep their mouths open and this may lead to dryness of the oral mucosa (Hayes and Jones 1995; Kite and Pearson, 1995 and Buglass, 1995). It is also common practice in ICUs to keep patients dehydrated in order to improve respiratory and cardiac function (Kite and Pearson, 1995). However, this may also exacerbate xerostomia and increase the potential for oral infections (McNeil, 2000).

2.3 Oral care and its relationship to nosocomial infections

Pneumonia is the most common nosocomial infection in ICU's that significantly contributes to morbidity and mortality patterns (Torres, Aznar and Gatell 1990) ICU patients. The risk is as much as 21 times greater than among non-ventilated patients and the mortality rate in these patients may exceed 50% (Torres et al. 1990). It is generally accepted that micro-aspiration of respiratory pathogens that colonize in the mouth of both older and critically ill patients can be a contributor to the development of nosocomial infections, particularly pneumonia (Scannapieco, Stewart and Myolette 1992; Fourrier, Duvivier and Boutigny 1998). Bacteria responsible for nosocomial pneumonia colonize the oral habitat of ICU patients (Scannapieco et al. 1992).

Mechanical ventilation involves the placement of an endotracheal tube into the lower airway. The bacteria that cause disease colonize the tube surface, which facilitates the transit of bacteria to the lung (Safdar, Cornish & Maki 2005). This is an important problem, not only because of increased mortality, but also because of the resulting extended length of hospital stay and the significant expansion of costs. Indeed, the onset of pneumonia can easily double the length of the patient's hospital stay.

The connection between oral health and VAP is rather straightforward. For pneumonia to develop, the pathogen must be aspirated from a proximal site (for example, the oropharyngeal cavity) into the lower airway. A person with teeth or dentures has non-shedding surfaces on which oral biofilms form. These biofilms are susceptible to colonization by respiratory pathogens (Scannapieco et al. 1992). Poor oral hygiene may predispose high-risk patients to oral colonization by respiratory pathogens. Subsequent aspiration would deposit these bacteria into the lower airway, thereby increasing the risk of infection. In addition, the host response to oral biofilms results in inflammation of the periodontal tissues. Thus, inflammatory products from the gingival tissues, as well as pathogenic bacteria shed from oral biofilms into the secretions, can be aspirated into the lower airway to promote lung infection (Scannapieco 1999).

Scannapieco and colleagues (1992) compared the colonization of dental plaque by respiratory pathogens in patients receiving treatment in medical intensive care units (ICUs) with that in matched, untreated control subjects. They examined the association between oral hygiene status together with other variables (for example,

antibiotic exposure) and the prevalence of oral colonization by potential respiratory pathogens. The results showed that patients treated in the ICU harbored greater levels of dental plaque than did the control subjects. Importantly, the authors found that bacterial pathogens known to cause pneumonia were prevalent only in the dental plaque of patients treated in the ICU. In some cases, up to 100 percent of the aerobic flora was *Staphylococcus aureus*, *P. aeruginosa* or one of several enteric species. In contrast, the dental plaque of control subjects rarely was colonized by respiratory pathogens. This finding suggests that the oral surfaces, especially dental plaque, could be a major reservoir of infection by respiratory pathogens in patients treated in the ICU.

2.4 Oral care practices:

Several oral care protocols have proved effective in reducing oropharyngeal colonization and nosocomial pneumonia risks. Chlorhexidine rinses and gels administration either two or three times daily have resulted in significant reductions in pneumonia in ICU patients (Fourrier et al. 2000). Generally, nurses have not been formally trained in assessing the oral status of patients in ICUs, and oral care protocols for these patients are not usually available (Fitch, Munro & Glass 1999). It has been recommended dental hygienists be involved in nursing education programs (Miller and Rubinstein, 1987) in order to improve the nurses' knowledge and ultimately their ability to provide better oral care. Fitch et al., 1999, recommended implementation of

a well-developed oral care protocol by bedside nurses to improve oral health of patients in the ICU (Table 1).

Table 1: Oral care protocol (Fitch et al 1999).

Preparation

1. Wash hands and don examination gloves.
2. Explain to the patient his/her mouth will be cleaned with toothpaste and mouthwash and then petroleum jelly will be applied to the lips.

Technique

1. Using a soft, pediatric-size toothbrush, brush the patient's teeth, gums, and tongue using Biotene[®] antibacterial dry mouth toothpaste. If the patient has no teeth, brush the gums and tongue gently.
2. If an airway (i.e., bite block) is present, remove, clean, and replace it after mouth care is completed.
3. If the patient is unresponsive and/or has clenched the mouth shut, use a mouth prop to gently open the mouth.
4. Rinse the toothpaste from the patient's mouth with an alcohol-free mouth rinse using an irrigation syringe or swab and suction as needed.
5. Apply Oral Balance moisturizing gel to a gloved finger and gently massage into the mucosal membranes of the patient's mouth.
6. With a gloved finger, apply petroleum jelly to the patient's lips.

Recently evidence based oral care protocols for ICU patients have been published; the protocols include oral assessment, tooth brushing with a child size brush, oral rinses and moisturizers every 2-6 hours (Schelder and Lloyd, 2002).

Some solutions and types of equipment used by nurses for oral care are not optimal (Kite and Pearson, 1995) Hydrogen peroxide and sodium bicarbonate effectively remove debris, but if not diluted carefully, may cause superficial burns. In a study conducted by Tombes and Galluci (1993) using hydrogen peroxide, significant mucosal abnormalities were reported and numerous subjective complaints were made. Foam swabs, which are commonly used to provide mouth care to patients who cannot provide self-care, are effective for stimulation of mucosal tissues but are ineffective in removing plaque (Nesley 1996, Buglass 1995, Moore 1995, Adams 1996, Holmes 1996 and Dewalt 1975).

It has also been stated 0.9% saline or water are just as effective as mouthwashes (Jenkins 1989, Liwu 1990 and Horwood 1990). Disposable cotton swabs are often used for cleaning and moistening the patients' mouths and teeth in critical care units. Examples of these are lemons and glycerine swabs that stimulate production of saliva initially but are acidic, causing irritation and decalcification of teeth (Adams 1996, Holmes 1996, Meurman and Cate 1996) therefore, choosing less erosive products is important.

2.5 Oral Care Protocol for ICU Patients

Fitch et al (1999) in their study used a mouth protocol (Table 1). The oral care provided in their study was performed by nurses and differed from routine oral care in several ways. First, a pediatric toothbrush was used which had the advantage of being small enough to remove plaque yet not disturb oral tubes. Its soft bristles reduce the

potential for trauma and bleeding. In addition, the care products selected were alcohol-free and antibacterial to enhance the mechanical effects of oral cleansing without drying the mucous membranes.

In the Fitch et al. study Oral Balance moisturizing gel was applied to the mucous membranes and then petroleum jelly was applied to the lips to reduce tissue drying. The nurses were able to complete the entire protocol in less than five minutes and preferred this mouth care protocol to previous methods in use in the unit. The experimental protocol was effective in reducing inflammation, whereas routine oral care had a minimal effect on inflammation.

They concluded the provision of a well-developed oral care protocol by bedside nurses could improve the oral health of patients in the ICU. There were also positive correlations between scores for salivary flow, plaque, inflammation, bleeding, and purulence obtained by the nurses and scores obtained by the dental hygienist indicating nurses can appropriately assess the oral status of patients in the ICU in the study after proper training.

2.6 Factors influencing oral care in ICU

The literature reports that although sound oral care is efficacious in reducing infection, oral care may be under-used in ICU's. Nurse education in oral practice has remained relatively unchanged over the past 120 years (Turner and Lawler, 1999). Turner and Lawler concluded that oral care practices do not reflect the influence of more recent

conceptual or rhetorical standpoints on oral care in nursing. Researchers have found indicators of this dissonance. Observing a sample of English nurses, Adams (1996) concluded that nurses, including those fully qualified, lacked adequate knowledge about oral health.

The hospital environment has been demonstrated to promote (by the provision of support for health promotion) and hinder (via time limitations and lack of continuity of care) nursing care (Berland, Whyte & Maxwell 1995). Similarly has factors such as availability of supplies; equipments and allocation of time affect the type and quality of oral care given by the nurse (Kite, 1995; Moore, 1995).

The availability of appropriate toothbrushes influences their use in ICU's (Kite, 1995); however, many units stock mouthwashes and foam swabs rather than toothbrushes, or the toothbrushes provided are of poor quality, large and not readily accessible (Moore, 1995).

Having sufficient time to provide oral care is also an important factor. The current shortage of nurses (in most developing countries) may be responsible for many nurses reporting that they feel overworked- a critical barrier in providing quality patient care (Alken, Clarke & Sloane 2002). When nurses are overworked and their time is rationed, oral care is often the first practice to be deferred. Archibald, Maaning & Bell (1997) investigated the relationship between nursing staffing, overcrowding and nosocomial infections rates and found that factors affecting nurse staffing had detrimental effects on patients' outcomes.

Nurses' attitudes towards particular health and treatment issues have been shown to affect nursing care (Roman, Sombes & Ezquerro 2001). Nurses' attitudes to oral hygiene have been associated with oral care practices as well. Wallace, Koepfel, Senko & Stawiaz 1997 studied the effects of nurses' attitudes and subjective norms on the intention to give oral care and found that they were important predictors of actual provision of care. Many nurses believe that oral health care carries low nursing priority (Wardh et al. 2000) and others recognize the importance of oral care yet believe that they lack adequate preparation and feel inadequate in their abilities to perform the procedure (Moore, 1995).

It can, therefore, be suggested that oral care is an important component of intensive care nursing that is often ignored due to various factors. The lack of published protocols ensures that there is no uniformity on the way oral care is given to patients. There is need, therefore, for research to be carried out to establish the current practice in oral care and look into ways of improving it.

Chapter Three: Methodology:

3.1 Study design

This was a descriptive cross sectional survey where a two-step analytical process was undertaken. First, several hypothesis were tested to determine the relationship between nurses' backgrounds, attitudes and perception of hospital factors and the quality of care in ICU. The level of significance was set at 0.05 (5%).

The second step of the analysis involved constructing regression equations to test the model proposed in Fig. 1.

3.2 Study area

The study was carried out in the Intensive Care Unit (ICU) at the Kenyatta National Hospital (KNH). KNH is a 1,800-bed referral and tertiary-care hospital, which is also the Teaching University Hospital. It is located in the heart of Nairobi about 3km from the city center. The ICU has 20 beds. (A preview of the study area is to be found in the appendix). This area was chosen because of its proximity, the availability of time and budget restrictions.

3.3 Study population

The study populations were the nurses working at the ICU KNH. There are a total of 100 nurses in working in the ICU. They are all registered nurses with a minimum of a diploma in Nursing.

3.4 Sample size determination

The following formula by Fisher et al 1999 was used to determine the sample size:

$$n = \frac{Z^2 pq}{d^2}$$

Where n= the desired sample size (if the target population is greater than 10,000)

z = the standard normal deviate at 95% confidence level (=1.96).

p = the proportion in the target population estimated to provide required oral care. (Since no studies have been done on these subjects 50% was used to determine the minimum sample size).

$$q = 1 - p$$

$$d = \text{level of precision (set at } \pm 5 \% \text{ or } 0.05)$$

Substituting these figures in the above formula:

$$\begin{aligned} n &= \frac{(1.96)^2 (.50) (.50)}{(0.05)^2} \\ &= 384 \end{aligned}$$

Since the target population is less than 10,000 the sample size was adjusted using the following formula:

$$n_f = \frac{n}{(1 + n) / N}$$

Where n_f = the desired sample size (where the population is less than 10,000)

n = the desired sample size (when the population is more than 10,000)

N = the estimate of the population size

$$\text{Hence } n_f = \frac{384}{(1 + 384) / 100} = \frac{384}{385 / 100} \\ = 99.74026$$

Since the calculated sample size is the same as the number of nurses working in the unit, all the nurses were included in the sample. The sample size was hence 100.

3.5 Selection of study subjects

All nurses working in the ICU at the time of the study were recruited for the study. Those on annual leave during the period of study (16 in total) were excluded while 4 declined to give consent even after explanations. The available sample was hence 80 nurses. To these, research assistants gave the questionnaires. Five questionnaires were returned incomplete and 75 were completed with a response rate of 94%.

The majority of the respondents were aged between 30-39 years. Most respondents were female (61) and ICN trained (81.3%). All the respondents were registered nurses. 72 (96%) of the respondents had diploma in nursing, 2 had bachelor's degree while one had a masters degree in intensive care nursing.

3.6 Inclusion criteria

1. Nurses who at the time of study were working in the ICU.
2. Nurses who gave consent to participate in the study.

3.7 Exclusion criteria

1. All nurses not deployed to work in the ICU at the time of study.
2. Nurses deployed to work in the ICU but who were on leave during the period of data collection.
3. Nurses deployed to work in the ICU but didn't give consent to participate.

3.8 Variables

3.8.1 Dependent variable:

Quality of oral care.

3.8.2 Independent variables:

1. Oral care education.
2. Years of ICU experience.
3. Time available.
4. Supplies provided by the hospital.
5. Value/importance of oral care.
6. Perceived unpleasantness of providing oral care.

3.9 Proposed model for ICU oral care

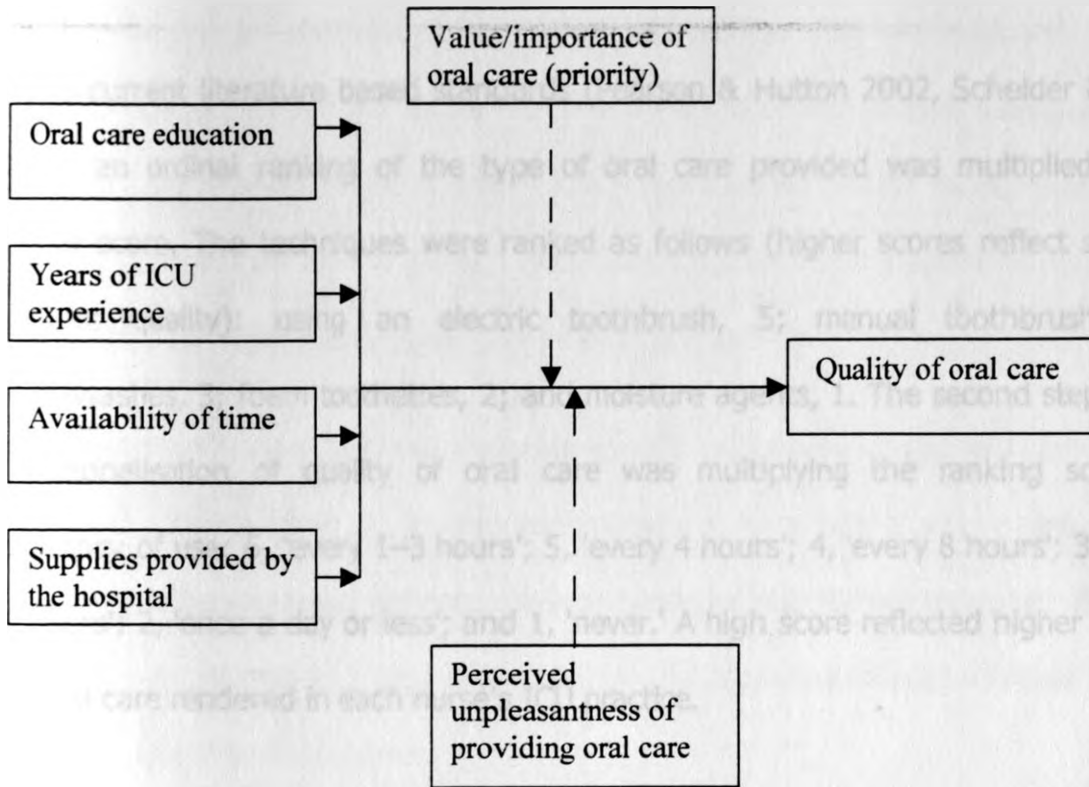
The following model was tested in the study. It suggested that provision of oral care in ICU is more than a function of education and individual experience of nurses, and includes both the organizational effects of the hospital environment and subjective attitudes nurses may hold about oral care.

It was expected that oral care education and years of ICU experience would have a positive impact on the quality of oral care provided. However, time and supplies were expected to hinder the provision of quality oral care. The value a nurse puts on oral care and its perceived unpleasantness were expected to interfere or facilitate provision of oral care.

Fig. 1: Proposed model for ICU oral care

Independent variables

Outcome



3.10 Operationalisation of variables

The variables in this study were operationalised as follows.

Dependent variable

Using current literature based standards (Pearson & Hutton 2002, Schelder & Lloyd 2002), an ordinal ranking of the type of oral care provided was multiplied by an ordinal score. The techniques were ranked as follows (higher scores reflect superior hygienic quality): using an electric toothbrush, 5; manual toothbrushes, 4; mouthwashes, 3; foam toothettes, 2; and moisture agents, 1. The second step of the operationalisation of quality of oral care was multiplying the ranking score by frequency of use: 6, 'every 1–3 hours'; 5, 'every 4 hours'; 4, 'every 8 hours'; 3, 'every 12 hours'; 2, 'once a day or less'; and 1, 'never.' A high score reflected higher level of the oral care rendered in each nurse's ICU practice.

Independent variables

The six variables were clustered into three conceptual groups: experience and education, nurses' perception of their hospital's facilities and support for providing oral care, and nurses' attitudes toward oral care practices. Nurses' professional backgrounds were represented by two variables: years of ICU experience and oral care education. Education was a measure of nurses' sources of learning about oral care for intubated patients. Responses were ranked so that formal sources received

higher scores (nursing school, 4 points; continuing education, 3 points; in-service, 2 points; and self-taught, 1 point) and each nurse checked all that applied.

Nurses' perceptions of hospital support for oral care were measured by responses to three 5-point Likert-scaled statements. A high score on the first two measures –'There are supplies readily available to provide oral care in our unit' and 'I have adequate time to provide oral care at least once a day'– represented greater facility support for oral care. A high score on the third, 'I need better supplies and equipment to perform oral care in ICU,' represented lower institutional support.

Two Likert-scaled items indicated attitudes specific to the provision of oral care: 'I find cleaning the oral cavity to be an unpleasant task' and 'Oral care is a very high priority for mechanically ventilated patients'. A high score on the former indicated a higher degree of unpleasantness while a high score on the latter represented a higher priority to the management of oral care.

3.11 Data collection.

Data was collected by means of a self-administered questionnaire.

3.11.1 Study instrument

A self-administered questionnaire was developed by the principal investigator and used to collect data (appendix 1). It had three sections: nurses' experience and education; current practice in oral care and nurses attitudes and perception towards

oral care. Nurse' demographic information was also included. An interview schedule/guide was used to get information from the in charge.

3.11.2 Pre-testing of research tool

A biostatistician reviewed the tool, which was then pre-tested for completeness and clarity at the intensive care unit. 5 nurses working in the ICU were requested to fill in the questionnaire. These nurses were excluded in the final survey. The results were checked against completeness, clarity and comprehension and the necessary amendments were made.

3.11.3 Selection and training of research assistants

Two research assistants among the BScN interns working at KNH at the time of data collection were recruited. They were trained on the purpose of the research, the objectives, how to use the research tool and interview techniques before commencement of the study. They were also trained on how to approach potential subjects, how to check the tool for completeness and entering of data in the computer.

3.12 Data analysis and presentation

Data collected was checked for completeness and unclear responses were substantiated. It was then coded and entered into the computer for easy access. It was analyzed using SPSS software. Descriptive statistics were used to summarize

quantitative data. The results were then presented in terms of tables and graphs and in percentages for qualitative data.

Correlation and regression analysis were applied to assess the relationship between independent variables and the provision of oral care.

3.13 Ethical consideration

Permission to carry out the research was sought from the Kenyatta National hospital-University of Nairobi Research standards and Ethics committee. Permission was also sought from the Permanent Secretary ministry of science and technology.

Each potential respondent had the research purpose explained to him/her, its benefits and the procedures. The respondents then signed an informed consent and any respondent seeking further clarification was assisted.

Any person unwilling to participate was not forced to do so and any person wishing to withdraw at any time during the study was free to do so. Anonymity, confidentiality and privacy were strictly maintained. Only the principal investigator and the research assistants had access to the data.

Chapter Four: Results

This chapter presents the results of the study under: demographic factors, nurses' experience and education, current practice in oral care and nurses' attitudes and perception towards hospital support.

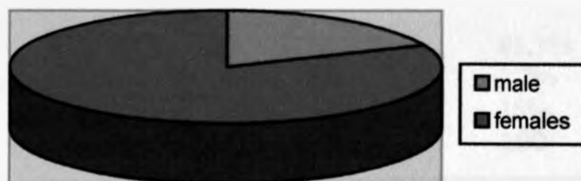
4.1 Demographic factors.

Table 2: Demographic factors

| | Frequency | Percentage |
|---------------------|-----------|------------|
| Gender | | |
| Females | 61 | 81.3% |
| Males | 14 | 18.6% |
| Total | 75 | 100 |
| Age in years | | |
| 21-29 years | 6 | 8% |
| 30-39 years | 59 | 78.7% |
| 40-49 years | 10 | 13.3% |
| Total | 75 | 100 |

61 (81.3%) of the respondents were females while 14 (18.7%) were males. Majority of the respondents (78.7%) were aged between 30-39 years while only 6 (8%) were aged below 30 years.

Figure 2: Gender of respondents



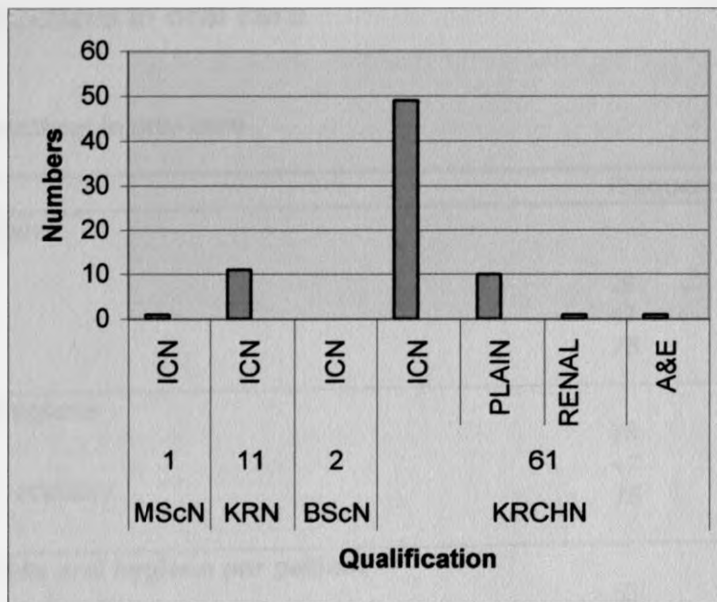
4.2 Nurses' experience and education

Table 3: Nurses' education and experience

| | Frequency | Percentages |
|---|-----------|-------------|
| Level of training | | |
| MScN | 1 | 1.3% |
| BScN | 2 | 2.7% |
| KRCHN | 61 | 81.3% |
| KRN/M | 11 | 14.7% |
| Total | 75 | 100 |
| ICU training | | |
| ICN training | 61 | 81.3% |
| No ICU training | 14 | 18.7% |
| Total | 75 | 100 |
| Length of basic training | | |
| 4 years and above | 2 | 2.7% |
| 2-3 years | 73 | 97.3% |
| Total | 75 | 100 |
| Number of years practiced as a nurse | | |
| Over 10 years | 28 | 37.3% |
| 5-9 years | 45 | 60% |
| 1-4 years | 2 | 2.7% |
| Total | 75 | 100 |
| Number of years worked in ICU | | |
| 5-9 years | 18 | 24% |
| 1-4 years | 54 | 72% |
| Less than one year | 3 | 4% |
| Total | 75 | 100 |
| Training on oral care | | |
| Nursing school | 61 | 81.3% |
| Continuous medical education | 2 | 2.7% |
| Self taught | 12 | 16% |
| Total | 75 | 100 |

61(81.3%) were ICU trained. The ICU training is one a one-year post basic course offered at the Kenya Medical Training College (KMTTC) as well as at the Kenyatta National Hospital. Upon completion the nurse becomes a holder of a post basic diploma.

Figure 3: Professional qualifications



73(97.3%) nurses took three and half years in their basic training. These are nurses who are trained at the diploma level and are registered nurses. They work in various capacities ranging from ward in charges to bedside nurses.

2(2.7%) nurses took 4 years for their basic degree in nursing (BScN). This course is undertaken at the university level.

Over half of the respondents (60%) have practiced as nurses for between 5-9 years while 72% have worked in the ICU for between 1-4 years. 81.3%(61) of the respondents got their training on oral care as part of the ICU training in the nursing school. 12(16%) of the respondents learnt how to perform oral care on the job through personal initiatives.

4.3 Current practices in oral care

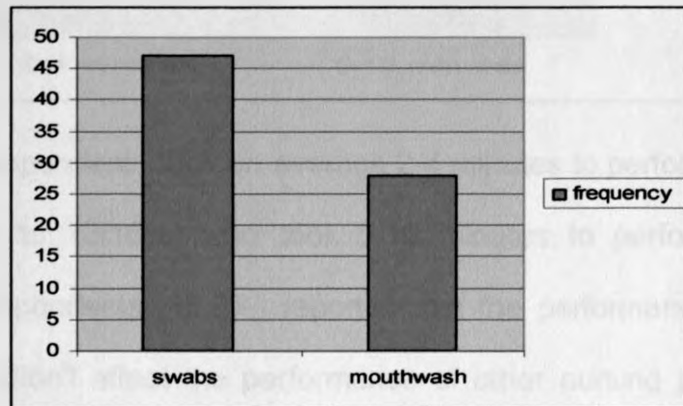
Table 4: Current practices in oral care

| | Frequency | Percentages |
|---|------------------|--------------------|
| Tool used for oral care | | |
| Mouthwash | 28 | 37.3% |
| Swabs | 47 | 62.7% |
| Total | 75 | 100 |
| Frequency of oral hygiene | | |
| Every 12 hours | 28 | 37.3% |
| Once a day or when necessary | 47 | 62.7% |
| Total | 75 | 100 |
| Time taken to provide oral hygiene per patient | | |
| 2-4 minutes | 63 | 84% |
| 5-10 minutes | 12 | 16% |
| Total | 75 | 100 |
| Time taken to perform oral hygiene affect other nursing procedures | | |
| Yes | 8 | 10.7% |
| No | 67 | 89.3% |
| Total | 75 | 100 |

28 (37.3%) respondents used mouthwash as the agent when providing oral care while the rest used swabs soaked in normal saline. All the respondents reported that they used the two an agent interchangeably depending on which was available. They all

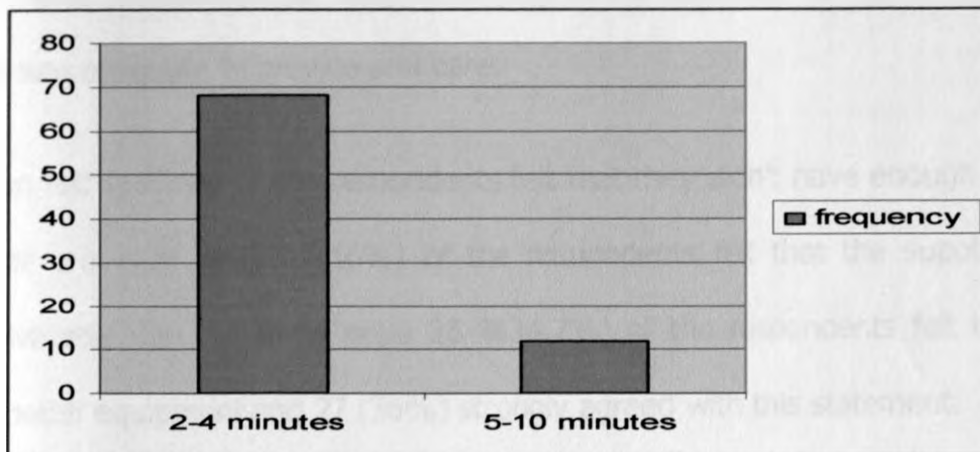
reported that they performed oral assessment of the oral cavity before carrying out oral care. They checked on the state of the oral mucosa, loose teeth, bad odor, sores and the position of the orotracheal tube.

Figure 4: Tool used for carrying out oral care



Above half of the respondents (62.7%) carried out oral care once a day or whenever necessary. They cited having to carry out oral care when necessary for patients with a lot of oral secretions or a patient with bad breath.

Figure 5: Time taken to provide oral care.



84% (63) of the respondents took on average 2-4 minutes to perform oral hygiene per patient compared to 12(16%) who took 5-10 minutes to perform the procedure. Majority of the respondents (89.3%) reported that the performance of the oral care procedure hence didn't affect the performance of other nursing procedures since it didn't take long to perform.

4.4 Nurses' attitudes and perception towards hospital support

Table 5: Nurses' attitudes and perception towards hospital support

| | | Strongly disagree | Disagree | Neutral | Agree | Strongly agree | Total |
|--|-----------|-------------------|----------|---------|-------|----------------|-------|
| Adequacy of time to provide oral care | Frequency | 3 | 26 | 11 | 21 | 14 | 75 |
| | % | 3.9% | 34.7% | 14.7% | 28% | 18.7% | 100 |
| Availability of supplies | Frequency | 0 | 49 | 2 | 12 | 12 | 75 |
| | % | 0% | 65.3% | 2.7% | 16% | 16% | 100 |
| Need for better equipment | Frequency | 7 | 9 | 6 | 26 | 27 | 75 |
| | % | 9.3% | 12% | 8% | 34.7% | 36% | 100 |
| Unpleasantness of cleaning oral cavity | Frequency | 9 | 27 | 39 | 0 | 0 | 75 |
| | % | 12% | 36% | 48% | 0% | 0% | 100 |
| Priority of oral care | Frequency | 0 | 0 | 0 | 24 | 51 | 75 |
| | % | 0% | 0% | 0% | 32% | 68% | 100 |

While 34.7% (26) of the respondents disagreed with the statement that they have adequate time to provide oral care, 28% (21) of the respondents felt that the time available was adequate to provide oral care.

More than half (65.3%) of the respondents felt that they didn't have enough supplies to provide oral care while 12(16%) of the respondents felt that the supplies were readily available. On the same issue 26 %34.7%) of the respondents felt that they needed better equipment and 27 (36%) strongly agreed with this statement.

36(48%) of the respondents disagreed with the statement that cleaning the patient's oral cavity is an unpleasant task with 39(48%) of the respondents taking a neutral stand.

All the respondents agreed that oral care is a high priority for mechanically ventilated patients with 51(68%) strongly agreeing with the statement.

On average the nurses in the ICU work on three shifts. The morning shift and afternoon shifts are five and half hours each while the night shift is fourteen and half hours long. 16-17 nurse work per shift in an attempt to achieve the one to one ratio (nurse: patient). Allocation to certain shifts depends on the nurse's seniority, training and personal characteristics like temperament. Oral care is almost always carried out during the morning shift for most of the patients.

Table 6: Bivariate Correlations (Spearman's rho).

| | | Quality of care | Experience | Education | Time | Supplies | Needs better equipment | Priority | Unpleasant |
|-------------------------------|-------------------------|-------------------------|------------|-----------|-------|----------|------------------------|----------|------------|
| Quality of care | Correlation Coefficient | 1.000 | | | | | | | |
| | Experience | Correlation Coefficient | -.200 | | | | | | |
| Sig. (2-tailed) | | .085 | | | | | | | |
| Education | Correlation Coefficient | .288* | .038 | | | | | | |
| | Sig. (2-tailed) | .012 | .746 | | | | | | |
| Time | Correlation Coefficient | .313** | .037 | .124 | | | | | |
| | Sig. (2-tailed) | .006 | .756 | .288 | | | | | |
| Supplies | Correlation Coefficient | .158 | .043 | .220 | -.072 | | | | |
| | Sig. (2-tailed) | .177 | .715 | .058 | .542 | | | | |
| Needs better equipment | Correlation Coefficient | -.126 | -.008 | -.139 | .019 | -.759** | | | |
| | Sig. (2-tailed) | .283 | .944 | .234 | .871 | .000 | | | |
| Priority | Correlation Coefficient | -.002 | .109 | .038 | .106 | -.026 | .161 | | |
| | Sig. (2-tailed) | .984 | .353 | .749 | .367 | .826 | .167 | | |
| Unpleasant | Correlation Coefficient | -.488** | -.069 | -.234* | -.223 | -.068 | -.036 | -.242* | |
| | Sig. (2-tailed) | .000 | .555 | .044 | .055 | .564 | .760 | .037 | |

* P < .05 ** P < .01 n = 75

Results of bivariate analyses indicated that education, having sufficient time and not viewing oral care as unpleasant were associated with higher quality care. Experience in ICU, seeing oral care as a priority and the hospital factors concerning supplies and equipment were not related to care contrary to expectations.

Table 5 shows the correlation coefficients for variables used in the analysis. Three of the seven variables had the predicted relationship with quality of care. Education, which was a measure of the source of training in oral care, had the predicted

relationship with a p value .012. It was expected that time would have a positive correlation to quality of care and this was found to be so with a p value of 0.006. Perceiving oral care to be unpleasant was predicted to affect the quality of care negatively. This was found to be so with a p value of 0.000.

Quality of care as measured here was not associated with experience in ICU, which was expected to positively affect the quality of care. The correlation coefficient was -.200 with a p value of .085. Quality of care was also not associated with priority given to oral care, p value of .984. The variable concerning equipment was also not associated with quality of care although the available supplies approached significance in the predicted direction.

Therefore hypothesis 2, 4 and 6 were rejected.

As shown in table 6 education, but not experience correlated with reporting oral care to be less unpleasant (correlation coefficient of -.234 and a p value of 0.044). Neither education nor experience was associated with priority, however. Priority and viewing of oral care as unpleasant task correlated with each other (correlation coefficient of -.242 and a p value of 0.037). Having time to provide oral care didn't correlate with both attitude variables.

Table 7: Quality of care regressed on all independent variables

| | Std. Error | Beta | t | Sig. |
|------------------------|-------------------|-------------|----------|-------------|
| Education | .190 | .226 | 2.346 | .022 |
| Experience | .066 | -.149 | -1.461 | .149 |
| Time | .082 | .216 | 2.094 | .040 |
| Supplies | .147 | .070 | .389 | .698 |
| Needs better equipment | .132 | -.041 | -.230 | .818 |
| Priority | .208 | -.108 | -1.078 | .285 |
| Unpleasant | .086 | -.442 | -4.252 | .000 |

Dependent Variable: quality of care

When quality of care was regressed on all independent variables simultaneously (table 7), education on oral care, availability of time and finding oral care unpleasant produced the highest betas. Viewing oral care as unpleasant task produced a significance of 0.000 strongly proving the predicted relationship as correct. The more the nurse views the oral care as unpleasant the lower the quality of oral care provided. On the other hand time had a p value of 0.040 while education had a p value of 0.022 both showing a high correlation to quality of oral care.

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Chapter five: Discussion

Oral hygiene in the ICU is a commonly performed nursing procedure in which the aim is to ensure that patients' mouths are cared for. Pritchard and David indicate mouth care is required to:

1. Achieve and maintain oral cleanliness
2. Prevent infection/stomatitis
3. Keep the oral mucosa moist
4. Promote patient comfort

Although a study done elsewhere (Adams 1996) show that nurse including those fully qualified lacked adequate knowledge about oral health, education in this study had the predicted relationship with a p value .012. This could be attributed to the fact that 61(81.3%) of the respondents had ICU training in which oral care education was incorporated. Also all the nurses working in the ICU were registered nurses.

ICU nurses may be hesitant to provide oral care to patients who are intubated because endotracheal tubes may limit access to the oral cavity. The fear of dislodging or displacing the tube is also a deterrent (Treloar DM, Stechmiller JK 1995). Provision of oral care may be affected by the perception that oral care contributes less to patients' health and well-being (or has lower priority) than other nursing interventions for critically ill patients. The respondents however took oral care as a high priority nursing procedures that didn't affect their performance of other nursing procedures.

The frequency of oral hygiene for intubated patients is an area of controversy. Day and Jenkins suggest the frequency is based upon the scores from an "at risk" calculator, whereas Trenter et al. (1986) recommend anywhere between two and four hours, depending on the patient's condition. The oral hygiene protocol suggested by Barnason et al. (1998) of brushing every 12 hours and oral moistening at least every two hours while the patient remained intubated appeared effective but suggested more detailed research in this area is needed.

Although current critical care nursing manuals advocate oral care ranging from every 2 hours to every shift (8-12 hours), more than half of the respondents (62.7%) reported carrying out oral hygiene on the patient once a day during the morning shift. This is despite that 67(89.3%) of the respondents reported that the time needed to carry out oral care didn't interfere with other nursing procedures, but still carried out oral care just once a day and in a few instances when necessary. This could be attributed to convenience on the part of the nurse.

According to existing nursing literature, toothbrushes are not the tools of choice for oral care by nurses (Howarth, 1977; Harris M, 1980). To the contrary, there is strong evidence to support the use of a toothbrush for the effective control of plaque and its associated complications; the toothbrush is recommended to be the tool of choice for mouth care (Kite and Pearson, 1995). However as noted just below half of the respondents (37.3%) used betadine mouthwash while 67.3% used saline swabs depending on which was readily available. None of the respondents reported using a

toothbrush. On further inquiry, it was reported that in fact toothbrushes are not found in the ICU because they are associated with able patients unlike the ones in the ICU. The practice of using a toothbrush for the maintenance of the oral hygiene of orally intubated patients is hence not widespread.

Generally, though nurses have been formally trained in assessing the oral status of patients in ICUs, and oral care protocols for these patients are usually available, the quality is still low. This could be attributed to individual differences and the fact that the nurses feel they need more supplies and equipment to carry out this procedure. It has been recommended dental hygienists be involved in nursing education programs in order to improve the nurses' knowledge and ultimately their ability to provide better oral care. Fitch et al. recommended implementation of a well-developed oral care protocol by bedside nurses to improve oral health of patients in the ICU (Table 1).

The hospital environment has been demonstrated to promote (by the provision of support for health promotion) and hinder (via time limitations and lack of continuity of care) nursing care (Berland et al.1995). Similarly has factors such as availability of supplies; equipments and allocation of time affect the type and quality of oral care given by the nurse (Kite, 1995; Moore, 1995). This can be supported by the findings of this research, which showed that more than half of the respondents (65.3%) reporting that they needed more supplies and equipments in order to carry out oral care effectively. The institutional support can therefore be rated as low.

These findings suggest that oral care among intubated patients in ICU is a multi-tiered process. Experience, though perhaps necessary, is not sufficient to improve quality of oral care in ICU. The intersection between nurse' experience and attitudes and institutional support in terms of providing adequate time is clear from these data and further suggests that attempts to change oral care nursing practices will require intervention that involve hospital managers and peer leaders.

Studies (Atkinson JC, 1994; Abele Horn et al, 1997; Johanson et al, 1988) indicate that multifaceted implementation strategies are more likely than single-faceted strategies to be effective at changing behavior and thus moving best practices into action. Hence, outlining specific care protocols or procedures facilitates consistency and quality care through standardization.

Study limitations

The results may not be generalized to other hospitals such as private hospitals, faith based organizations and even other public hospitals due to the differences in organizational factors. KNH was selected as a study site owing to its proximity, budget and its size, which makes it more representative of other institutions.

The interrelationship between the factors was not considered and this could explain some of the correlation between the dependent and independent variables.

Conclusion and Recommendations

Conclusion

This study revealed that the oral care hygiene is an important component of intensive care nursing. It was found that education, availability of time and viewing of the oral care as an unpleasant task directly influenced the outcome of care. On the other hand, experience, availability of supplies and equipment as well as the priority given to oral care didn't have significant effects on the outcome of oral care.

The results of this study hence suggest that oral care provision for mechanically ventilated patients can be improved by providing oral care education, providing nursing staff with adequate time and reducing the perception that oral care is unpleasant. Multifaceted interventions to improve oral care nursing practices are required to reduce the incidence of pneumonia in mechanically ventilated patients, thereby improving patient safety.

Recommendations

The following are recommended for the oral care of ICU patients:

1. ICU patient's individual requirements for oral care should be considered as part of the admission assessment.
2. Education of nurses to provide skills in oral assessment and oral care is essential. A dental hygienist can train bedside nurses to improve the oral assessment and enhancement of oral care for ICU patients.
3. The use of an assessment model such as the "BRUSHED" Assessment Model (appendix 7) is recommended for the immediate identification of oral problems for every patient and should be carried out daily.
4. The use of a comprehensive protocol such as the Mouth Care Protocol presented in Table 1 has been shown to be effective and is recommended.
5. The frequency of oral care is an area of controversy and may depend more on the patient's condition. However, brushing every 12 hours and oral moistening at least every two hours while the patient remains intubated is recommended until further research is done in this area.
6. Some solutions and types of equipment used by nurses for oral care are not optimal and, therefore, caution must be applied if they are used. Swabs are ineffective in removing plaque, whereas the use of a soft pediatric toothbrush is recommended instead.

7. Further research is needed to determine the most effective way to perform oral hygiene care in critically ill patients as well as deciding on the most appropriate frequency of oral care. Research is also needed to determine the impact of oral health and improved oral health status on patients' outcome.

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Appendices

Appendix 1: Questionnaire

Q. No.....

Questionnaire for the research on '*Factors affecting provision of oral care by nurses in the intensive care unit at the KNH*'.

Instructions:

- Read and sign the attached consent form before filling this questionnaire. Do not write your name.
- This questionnaire has sections A to D. Fill all the sections.
- Indicate by circling the number against the most appropriate response for sections A to C.
- For section D circle the number that best describes your feelings about hospital support for oral care.

Section A: Demographic factors

1. Please indicate your gender.

- 1) Female
- 2) Male

2. What is your age in completed years?

- 1) 21-29 years
- 2) 30-39 years
- 3) 40-49 years
- 4) Above 50 years

Section B. Nurse's experience and education

3. What is the level of your training? (NB: If all apply please indicate).

- 1) MscN
- 2) BScN
- 3) KRCHN
- 4) KRN/M
- 5) ICN

4. How long did your basic training take?

- 1) 4 years and above
- 2) 2- 3¹/₂years
- 3) 1 year

5. For how many years have you practiced as a nurse?

- 1) Over 10 years
- 2) 5-9 years
- 3) 1-4 years
- 4) Below 1 year

6. How long have you worked in the intensive care unit?

- 1) Over 10 years
- 2) 5-9 years
- 3) 1-4 years
- 4) Less than one year

7. How did you start working in the ICU?

- 1) Personal choice
- 2) Deployment
- 3) Other (specify).....

8. Do you have any formal training on oral care of intubated patients?

- 1) Yes
- 2) No

9. If yes how did you get your training?

- 1) Nursing school
- 2) In service
- 3) Continuous medical education
- 4) Self taught
- 5) Others (specify).....

10. How long did this training (on oral care) take?

- 1) 1 year
- 2) 6 months
- 3) 3 months
- 4) Others (specify).....

Section C. Current practice in oral care

11. Which of the following do you use when carrying out mouth care on an intubated patient?

- 1) Electric toothbrush
- 2) Manual toothbrush
- 3) Mouthwashes (specify which).....
- 4) Swabs (specify).....
- 5) Moisture agents

12. How often do you carry out oral care?

- 1) Every 1-3 hours
- 2) Every 4 hours
- 3) Every 8 hours
- 4) Every 12 hours
- 5) Once a day or less
- 6) Never

13. Do you carry out any assessment before providing oral care?

- 1) No
- 2) Yes (Explain)

14. On average how long does it take you to perform oral hygiene per patient?

- 1) Under one minute
- 2) 2-4 minutes
- 3) 5-10 minutes

4) Over 10 minutes

15. Does the time it takes you to perform oral hygiene affect the performance of other nursing procedures?

1) Yes

2) No

If yes please specify

Section D. Nurses,' attitude and perception towards hospital support

For questions in this section, please circle the number the best describes your feelings about hospital support for oral care and its provision using the following key.

KEY:

1. Strongly disagree 2. Disagree 3. Neutral 4. Agree 5. Strongly agree

16. I have adequate time to provide oral care at least once a day

1 2 3 4 5

17. There are supplies readily available in our unit to provide oral care.

1 2 3 4 5

18. I need better supplies and equipment to perform oral care in ICU.

1 2 3 4 5

19. I find cleaning the oral cavity to be an unpleasant task

1 2 3 4 5

20. Oral care is a very high priority for mechanically ventilated patients

1 2 3 4 5

Appendix 2: Interview guide for ward in charge (interviewer administered).

1. How many shifts do the nurses work?.....
.....
.....

2. How long is each shift?.....

3. How many nurse work per shift?.....
.....

4. Which criteria do you use to allocate nurses to certain shifts?.....
.....
.....

5. Do you have any protocol on how to carry out oral care?

Yes

No

6. If yes, who developed the protocol?.....
.....
.....

7. Is it followed by the nurses?

All the nurses.....

Some of the nurses.....

None of the nurses.....

If not followed by all, why is this so?

.....

.....

.....

.....

8. If no (to Q.5) why isn't there one?.....

.....

.....

Appendix 3: Consent form:

School of Nursing Sciences - University of Nairobi

Investigator: Dorcas Maina

I am a student in the University of Nairobi undertaking research in fulfillment of the requirement of the award of Masters Degree in Nursing. I am carrying out research on *'factors affecting provision of oral care by nurses in the ICU at the KNH'*.

Procedures:

This study involves interviewing nurses working in the ICU by filling a self-administered questionnaire. The results of this study may aid in development of an oral care protocol, which will ease your burden of patient care, and also in improving it. There are no risks associated with participating in this study although some questions may be personal in nature.

You are at liberty to participate or to withdraw at any time you wish without prejudice or coercion. You will not be required to write your name and the results will be treated with confidentiality.

Research committee contact:

If in doubt please contact the research committee:

Professor Bhatt - the chairman Kenyatta Ethics and Research Board,

P.O Box 20723,

Nairobi.

Or the investigator at 0721716514.

I have read and understood the purpose and benefits of this study and hence agree/decline to participate.

Respondents Signature.....date

Investigator's signaturedate.....

Appendix 5: Letter of Approval from the Permanent Secretary Ministry of Science and Technology



REPUBLIC OF KENYA
MINISTRY OF SCIENCE & TECHNOLOGY

Telegrams: "SCIENCE TEC", Nairobi
Telephone: 02-318581
E-Mail: ps@scienceandtechnology.go.ke

JOGOO HOUSE "B"
HARAMBEE AVENUE,
P.O. Box 9583-00200
NAIROBI

When Replying please quote
Ref. MOST 13/001/ 37C 491/2

3rd August 2007

Dorcas W. Maina
University of Nairobi
P.O. Box 30197
NAIROBI

Dear Madam

RE: RESEARCH AUTHORIZATION

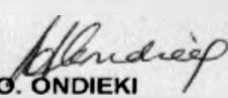
Following your application for authority to carry out research on, '*Factors Affecting Provision of Oral Care by Nurses in the Intensive Care Unit at the Kenyatta National Hospital*'

I am pleased to inform you that you have been authorized to carry out research the Kenyatta National Hospital for a period ending 30th June 2008.

You are advised to report to the Director Kenyatta National Hospital before embarking on your research project.

On completion of your research, you are expected to submit two copies of your research report to this office.

Yours faithfully


M. O. ONDIEKI
FOR: PERMANENT SECRETARY

Copy to:

The Director
Kenyatta National Hospital
Nairobi

Appendix 6: Research Clearance Permit

PAGE 2

PAGE 3

THIS IS TO CERTIFY THAT:

Prof./Dr./Mr./Mrs./Miss DORCAS W. MATINA

of (Address) UNIVERSITY OF NAIROBI
P.O. BOX 30197 NAIROBI

has been permitted to conduct research in PERMANENT SECRETARY OF EDUCATION
KENYATTA NATIONAL HOSPITAL Location, NAIROBI District,
NAIROBI Province.

on the topic FACTORS AFFECTING PROVISION OF ORAL CARE BY NURSES IN THE INTENSIVE CARE UNIT AT THE KENYATTA NATIONAL HOSPITAL.

for a period ending 30TH JUNE 2008

Research Permit No. MOST 13/001/37C 492
Date of issue 3.8.2007
Fee received SHS. 500.00



M.G. ONDIEKI
Applicant's Signature FOR Permanent Secretary
Ministry of Science and Technology

Appendix 7: Preview of Kenyatta National Hospital

KNH is the largest Teaching and Referral hospital in Kenya. It is located in Nairobi province about 3 kilometers from the Nairobi central business district. The hospital was set up during colonial times when it was referred to as King George's hospital. The hospital also receives patients from the other regions in Africa.

It has now a capacity of 2000 beds. Its many clinical departments include: department of medicine, department of surgery, department of obstetrics and gynecology, department of pediatrics, laboratory department, and radiology department. Other departments/wards fall under these; that are renal unit, intensive care unit, trauma and emergency department and operating theaters.

The hospital has an ICU divided into the intensive and high dependency unit. The total bed capacity is 20.

It has about 1600 nurses with the ICU/HDU having 100 nurses.

The hospital hosts the University of Nairobi medical school and other students from various training institutions who come for training and experience in the hospital.

Appendix 8: 'BRUSHED' assessment model

Hayes and Jones (1995) recommended the use of the BRUSHED Assessment Model.

This model was made to prompt nurses to check for particular clinical signs during oral assessment.

B Bleeding

(Gums, mucosa and coagulation status)

R- Redness

(Gum margins, tongue, and antibiotics stomatitis)

U- Ulceration

(Size, shape, herpetic, infected)

S- Saliva

(Xerostomia, hyper salivation, characteristics)

H- Halitosis

(Character, acidotic, infected)

E- External factors

(Angular cheilitis, endotracheal tapes)

D- Debris

(Visible foreign particles)

Appendix 9: Budget projections

| Item | Units | Cost Per Unit (Shs) | Amount (Shs) |
|--|-------------|---------------------|-------------------|
| A. Computer services | | | |
| Computer (lap top) and software | 1 | 68,000.00 | 68,000.00 |
| Printing proposal | 40 pages | 5.00 | 200.00 |
| Photocopying proposal | 3x40 pages | 3.00 | 360.00 |
| Binding | 3 copies | 70.00 | 210.00 |
| Photocopying questionnaires | 110 copies | 3.00 | 330.00 |
| Printing final report | 70 pages | 3.00 | 210.00 |
| Photocopying final report | 7x 70 pages | 3.00 | 1,470.00 |
| Binding final report | 4 copies | 70.00 | 280.00 |
| Binding the book | 1 copy | 500.00 | 500.00 |
| B. Stationery | | | |
| Printing paper | 3 reams | 450.00 | 1,350.00 |
| Notebooks | 2 | 50.00 | 100.00 |
| Calculator | 1 | 1,200.00 | 1,200.00 |
| Flash disks | 2 | 2,000.00 | 4,000.00 |
| Staples and stapler | 1 | 300.00 | 300.00 |
| Paper punch | 1 | 300.00 | 300.00 |
| Ball pens | 1 dozen | 180.00 | 180.00 |
| Erasers | 5 | 10.00 | 50.00 |
| 4C. Personnel/Human resources | | | |
| Wages- pretesting (research assistants) | 2x1 day | @400.00 | 800.00 |
| Wages (research Execution) | | | |
| i) Research assistants | 2 x 5 days | @700.00 | 7,000.00 |
| ii) Principal investigator | 1 x 5 days | @1,400.00 | 7,000.00 |
| D. Ethical committee fee | | | |
| | | Submitted once | 500.00 |
| E. Ministry of Science and technology | | | |
| | | Submitted once | 500.00 |
| Sub Total | | | 94,840.00 |
| 15% contingencies | | | 14,226.00 |
| TOTAL | | | 109,066.00 |

✓
**OUTCOME OF LATERAL INTERNAL
SPHINCTEROTOMY AS COMPARED TO MANUAL
ANAL DILATATION**

**A PROSPECTIVE STUDY AT
KENYATTA NATIONAL HOSPITAL** //

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**A DISSERTATION SUBMITTED AS PART FULFILMENT FOR THE
DEGREE OF MASTER OF MEDICINE IN SURGERY
UNIVERSITY OF NAIROBI**

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DATE:..... *11/07/06*

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SIGNED: 

DATE:..... *11/9/06*

DECLARATION

I certify that this dissertation is my original work and that it has not been submitted in any other university.

INVESTIGATOR:

DR. DANIEL O. ALUSHULA

Signed: *D. Alushula*

Date: *11.07.06*

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LIST OF ABBREVIATIONS

MAD – Manual anal dilatation

LIS – Lateral internal sphincterotomy

HIV – Human immuno deficiency

AIDS – Acquired immuno deficiency syndrome

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SUMMARY

Background

Fissure in ano (anal fissure) is a common anorectal condition. Concern is drawn here because if acute, the degree of patient discomfort and disability far exceeds that which might be expected for any otherwise rather trivial lesion.

Objectives

The study evaluated the outcome of lateral internal sphincterotomy as compared to manual anal dilatation as a basis for future practice.

Anal fissure can be classified into two: acute and chronic fissure. It can occur at any age, but is usually a condition of young adults. Sex distribution is males to females is 1:1, however, women are much more likely to develop this condition than men. Fissures in ano can occur anteriorly at 12 o'clock or posterior at 6 O'clock. They are commonly found in the midline, this is because of the elliptical arrangement of the external sphincter, which is felt to provide less support to the anal canal in the antero-posterior axis thus rendering this location most susceptible to trauma. Blood supply, which is already tenuous, may be further compromised by compression and contusion as the branch of the inferior rectal artery passes through the internal anal sphincters.

Clinical diagnosis of fissure in ano is made from history and physical examination.

Setting

A prospective study carried out at Kenyatta National Hospital wards 5A, 5B and 5D.

Methods and patients

In the study period of nine months, from June 2004 to February 2005, both months inclusive, we sampled seventy-eight patients of whom forty underwent lateral internal sphincterotomy and thirty-eight underwent manual anal dilatation (Lords procedure).

Procedures were carried out under spinal or general anaesthesia. Early complications noted were such as bleeding and haematoma formation. These were noted immediately after surgery and up to 24 hrs post surgery. Late complications were noted from the third day, and these were such as incontinence of flatus, incontinence of stool, pruritus ani, and abscess formation.

Results

Fifty six percent of the patients who underwent manual anal dilatation developed stool incontinence while none developed this with lateral internal sphincterotomy. Eighty four point eight percent of those who underwent manual anal dilatation developed incontinence of flatus. While 15.2% of those who underwent lateral internal sphincterotomy developed incontinence of flatus. Eighty percent developed pruritus ani in manual anal dilatation while 20% developed this in lateral internal sphincterotomy. Abscess formation was seen in 92% of those who underwent manual anal dilatation while 8% of this was seen in lateral internal sphincterotomy. On discharge the patients were reviewed and the early complications resolved. Late complications were seen up to four weeks post surgery these resolved with time and conservative management. This tells us that complications were temporary.

Those who underwent lateral internal sphincterotomy had a short duration of stay in hospital while those who underwent manual anal dilatation had a longer duration of stay for the complications to be treated. Recurrence was found to be more in manual anal dilatation than lateral internal internal sphincterotomy whereby of those who underwent MAD, 14 (36.8%) had recurrence whereas in LIS 8 (20%) had recurrence.

The study showed that more men suffered than women to the ratio of 1.66:1. More complications were seen with manual anal dilatation than lateral internal sphincterotomy therefore patients were forced to stay longer in hospital for the complications to be treated. Since our patients came to us after two months (chronic fissures) conservative management had no role.

Conclusion

Therefore, lateral internal sphincterotomy remains the attractive option for many patients suffering from this painful condition.

INTRODUCTION

Chronic anal fissure is characterised by severe pain after defecation and often fresh anal bleeding. Physical examination reveals a tear in the anal canal. Most patients with chronic anal fissures have a high resting anal pressure, which impairs blood flow through the anal sphincters to the anal mucosa and prevents healing.

The primary aim of treatment of anal fissure is to reduce pressure generated by the anal sphincter mechanism to improve blood flow and allow mucosal healing. Due to the complications seen in our SOPC clinic following manual anal dilatation, done from the periphery and some units in KNH we were prompted to study the two modalities.

Lateral internal sphincterotomy being the conventional method for the treatment of anal fissure as compared to manual anal dilatation, the aim of the study was to evaluate the outcome of the two modalities as a basis for future practice.

LITERATURE REVIEW

According to Antropoli, the oldest proctologist, pathologies of the anal canal are extremely common. About 30 – 40% of the population suffers from proctological pathologies at least once in their lives. Anal fissure was recognised as a clinical entity in 1934. It is a longitudinal defect of the anal canal mucosa and anoderm extending usually from the dentate line to external verge of the anal canal. This defect exposes the lower half or even most of the fibres of internal anal sphincter. Anal fissure is almost always accompanied by extensive tension of these muscles. Anal fissure affects all age groups but predominantly occur in the third and fourth decades of life (1). The longitudinal tear in the squamous epithelium of the anal canal, frequently precipitated by the passage of hard stools, though in small proportions of patients, it may follow an episode of diarrhoea (2). Majority of patients have been mis-diagnosed to have haemorrhoids and in the real sense have fissure in ano. HIV/AIDS presents with wounds around the anal region and the stigma of the disease has made many keep away due to the embarrassment that may be caused on them, therefore they present late. (2)

AETIOLOGY

Fissures can be classified as primary or secondary depending on the cause.

The cause of idiopathic fissures is not known, however several theories have been postulated and some of these are: -

- 1) The **mechanical forces** imposed on the anal canal during the passage of stool. Hard stools are most commonly implicated, but forceful liquid stool can also produce the same results. (1,3)
- 2) **Tears** in the anus caused by foreign bodies such as instrumentation or undigested bone spicules or any other hard foreign body. (1,3)
- 3) **Laxatives** – habitual users of this may also develop fissures. There is fairly liquid stool for a long time leading to tightly fibrosed anal canal. If this patient passes hard stool the skin splits leading to fissures in ano. This is also due to the spasms of the internal sphincter muscles. (1,3)
- 4) **Childbirth** – A number of women end up with fissures especially anterior fissures following complicated childbirth. (1,3)
- 5) **Fissures** in ano may be secondary to other anal diseases such as fistulas in ano, proctitis as well as fibrous anal polyps. (1,3)
These diseases may also complicate surgery such as laying out of a fistula or haemorrhoidectomy. It may also follow pruritis ani. Not forgetting worm infestations such as Trichuris, Enterobius vermicularis that lay ova around the anal opening (4).
- 6) The **anatomy** of the anal canal plays a role in the aetiology of fissures in ano. This depends on the arrangement of the anal muscles. Lockhart –Mummary felt that the external sphincter structure plays a role in causing fissures. The lower portion of this muscle fibres is not truly circular, but consists of a band of muscles fibres that pass from posterior to anterior and split around the anus. He postulated that the anal mucosa is best supported laterally and is weakest posteriorly and therefore more fissures occur posteriorly (5).
- 7) **Blood supply.**
Klosterhalfen and colleagues did angiographies on cadavers to visualise the inferior rectal artery using vascular injection, they showed that the posterior commissure is less perfused than the other areas of the anal canal, hence, ischaemia may be an important aetiological factor in causing anal fissures in the posterior location. (5)

- 8) **Secondary causes** of fissures in ano are seen in patients with syphilis and other sexually transmitted diseases, tuberculosis, leukaemia, inflammatory bowel disease such as Crohn's disease, and HIV. (6)

In this secondary fissures, treatment or management will be directed to the primary causes as above. (6)

Physiology of regulation of internal sphincter tone

There are three main influences of internal sphincter tone and function reflecting the sphincter specialization of this muscle (7). The first is intrinsic myogenic tone. This depends on the extra cellular calcium levels entering via L-type calcium channels (7).

The second influence is the enteric nervous system also known as the third division of the autonomic nervous system. This pathway is located in the Auerbach and Meissner's plexi in the wall of the gut and responsible for peristalsis as well as local reflexes such as the rectoanal inhibitory reflex. The nerves are known to be non-adrenergic, non-cholinergic because neither guanethidine nor atropine block their activity, yet tetrodotoxin does. The neurotransmitter has been identified as nitric oxide. This relaxes the internal sphincter, an action blocked by N-nitro-L-arginine.

The third influence is the autonomic nervous system which affects contraction and relaxation of the internal sphincter via sympathetic and parasympathetic post ganglionic fibres respectively. They act directly on the smooth muscles and indirectly on the nerves of the enteric nervous system or both. The sympathetic neurotransmitter noradrenaline contracts the internal sphincter via its action on alpha-receptors, an action blocked by the alpha antagonist phentolamine. (8)

Physiological studies

Researchers have been interested in anorectal pressure studies in patients with anal fissures.

- a) Duthies and Benneth Measured sphincter pressure, with an open-ended tube connected to a recording device by a sterile gauze (9).
- b) Miles and Stewart have also shown that the posterior angulation of the rectum leads to uneven straining at the margins of the anal opening during defecation. (9)

Therefore during defecation the pressure of the hard faecal mass is mainly on the posterior anorectal angle in which event the overlying epithelium is greatly stretched, and being relatively unsupported by muscles, remains an area of fissures formation.

All patients demonstrated spasms of the sphincter on the digital rectal examination, but no increase in resting pressure in the control subjects. Following sphincter stretch, a moderate fall in pressure was noted but it returned virtually to normal by the eighth post-operative day (9).

- c) Gibbons and Read employed perfusion probes of varying diameters in patients with chronic anal fissures. Resting pressure was elevated in all subjects when compared to the controls. He therefore concluded that resting pressure was elevated in patients with fissures in ano (8).
- d) Nothmann and Schuster performed balloon rectosphincter manometry on patients with anal fissure. Resting pressures were twice as high as those measured in control subjects.

Following distention of the rectum by the balloon, there is the expected internal sphincter relaxation, but this is followed by a marked and prolonged contraction above the initial baseline termed the “overshoot” phenomenon. Therefore they concluded that this reflexly stimulated sphincter spasm and so involved in the aetiology of the condition (9).

ANAL ANATOMY

The anal canal commences at the level where the rectum passes through the pelvic diaphragm and ends at the anal verge. The muscular junction between the rectum and anal canal can be felt with the finger as a thickened ridge – the anorectal bundle or “ring”. The internal sphincter is a thickened continuation of the circular muscle coat of the rectum. (1,2) The involuntary muscle commences where the rectum passes through the pelvic diaphragm and ends at the anal orifice where its border can be felt. The internal anal sphincter is 2.5 cm long and 2-5mm thick. When exposed during life it is pearly white in colour and its individual transversely placed fibres can be seen clearly. Spasm and contracture of this muscle play a major part in fissure and other anal affections. (1,2)

The longitudinal muscle is a continuation of the longitudinal muscle coat of the rectum, intermingled with fibres from the pubo rectalis. Its fibres fan out through the lowest part of the external sphincter, to be inserted into the true anal and peri-anal skin. The longitudinal muscle that are attached to the epithelium provides pathway for the spread of perianal infections, and mark out tight compartment that are responsible for the intense pressure and pain that accompany many localised perianal lesions. Beneath the anal skin lie the space of the corrugator cutti ani muscle.

The external sphincter has its fibres attached posteriorly to the coccyx, while anteriorly they are inserted into the mid perineal point in the male, whereas in the female they fuse with the sphincter vaginae. (1,2)

Between the internal (involuntary) sphincter and the external (voluntary) sphincter muscle mass is found a potential space, the inter-sphincteric plane. The plane contains the eight to twelve apocrine glands which can cause infection and it is also a route for the spread of pus. It can also be opened by a surgeon to provide access for operation of the sphincter muscle. (1,2)

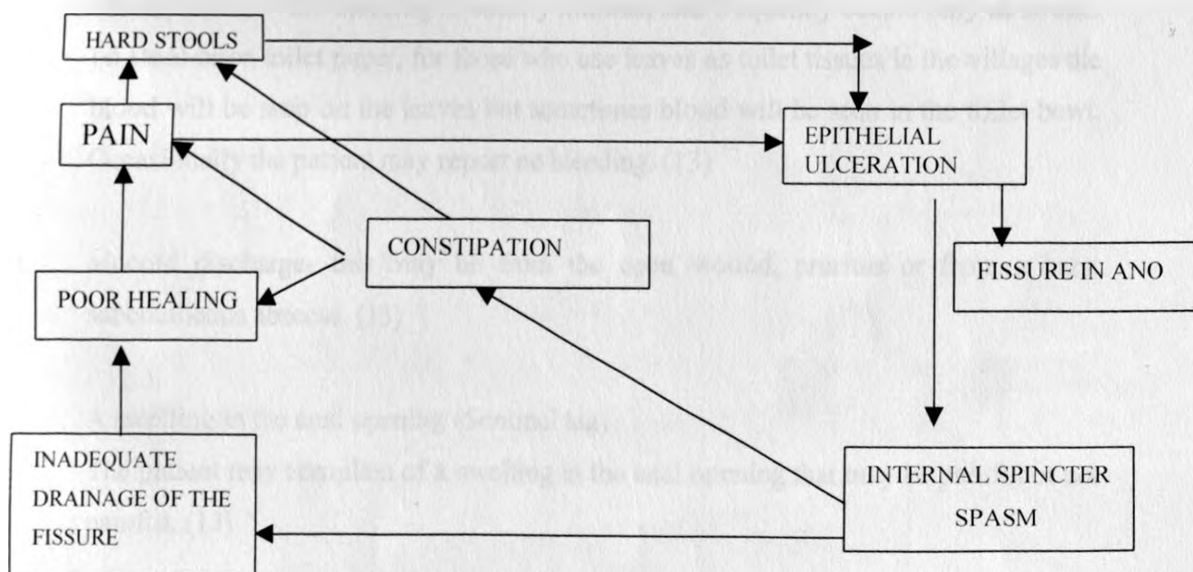
The mucous membrane – the pink epithelium lining the rectum extends through the anorectal ring into the surgical anal canal (1, 2).

Pathology

Acute anal fissures are not normally associated with skin tag formation. Chronic anal fissures are associated with the development of anal tags as a result of inflammatory oedema. The ulcer in chronic anal fissure is cone shaped and at the inferior extreme there is a tag of skin, usually oedematous. This tag is known as a sentinel pile. Sentinel because it guards the fissure. A vicious cycle ensues which in the sub-epithelial inflammation causes spasms of the internal sphincter inhibiting free drainage of the infected fissure and permitting continued inflammation, resulting in a small, chronic inadequately drained abscess. The reflex relaxation of the internal sphincter that normally precedes defecation is lost in patients with fissures in ano. and instead contraction of the internal sphincter occurs. (3).

Diagram as illustrated by the flow chart

Vicious cycle as seen in fissures in ano (3)



Due to the contraction of the internal sphincter and pain sensation, the patient develops the fear to defaecate and this leads to constipation, Stool will be hard and so stretching of the fissure occurs tearing the epithelium more and so more pain. (1,2,3)

Once there is a crack in the epithelium and then a break in continuity of the mucus membrane, the anal canal being a place with micro organisms, infection occurs, and this leads to the chronicity of the fissures. There is repeated injury by the hard stools also. With time the fissures heal with fibrosis and so anal stenosis, may occur. (10)

The “sentinel pile” a hypertrophied anal papilla seen in the majority of chronic anal fissures, results from oedema with contributions made by a combination of infection, lymphostasis, surface irritation by discharge from fissures and squeezing up effect produced by a tight sphincter. (11)

The undermining of the sentinel tag occurs with abscess formation which then drains in the anal canal after rupture due to the tight sphincter and then complicates into a subcutaneous fistula (12).

Clinical features.

From history the patients will complain of the following: -

- 1). Anal pain, usually described as burning or tearing
The pain usually occurs with and immediately after defecation. Usually pain ceases in a few minutes but occasionally it may persist for hours. The pain is also related to bowel movements. (13)
- 2). Bloody stools: - the bleeding is usually minimal and frequently occurs only as streaks on stool or on toilet paper, for those who use leaves as toilet tissues in the villages the blood will be seen on the leaves but sometimes blood will be seen in the toilet bowl. Occasionally the patient may report no bleeding. (13)
- 3). Mucoïd discharge- this may be from the open wound, pruritus or from a burst subcutaneous abscess. (13)
- 4). A swelling in the anal opening (Sentinel tag)
The patient may complain of a swelling in the anal opening that may be painful or not painful. (13)

5). Others: - They may complain of problems with micturation (retention, urgency, frequency) and dyspareunia due to perinial muscular spasms. Dyspareunia or problems with micturation may occur in both acute and chronic fissures.

Loss of appetite due to failure to defecate, constipation and pain may be reported. (13)

Constipation is the antecedent event, but once pain develops the fear of the act of defaecation and refusal to the call to stool can exacerbate tears in the anal mucosa.

This anxiety leads to faecal impactions particularly in children and the elderly. (13)

Physical exam

Patient is placed in left lateral decubitus position with knees drawn up towards the chest.

Inspection is done, of the anal opening, by gentle retraction of the glutea. Due to pain the anal sphincter muscles may be in spasms, therefore digital rectal examination is discouraged unless a local anaesthetic is used where the depth of fissure can be accessed and its orientation to the midline often described using clock orientation of the hour hand. (13)

Palpation may demonstrate a spastic anal sphincter or tight anal canal and will exacerbate patient's discomfort. There may be no fibrosis in the acute phase as the tear is superficial. (13)

Anoscopic examination will confirm the location of the fissures.

Acute fissures are seen to be erythematous and bleed easily.

With chronic fissures, classic fissures triad may be seen (13).

- a) Deep ulcer
- b) Sentinal pile, which forms when base of fissures becomes oedematous and hypertrophic
- c) Enlarged anal papillae. This can be mistaken for tumours. (13)

In chronic cases anoscopy can be accomplished, as pain in these cases is not very severe.

Proctosigmoidoscopy may be performed to rule out the possibility of a tumour or distal inflammatory bowel disease. (13)

Occasionally the base of the ulcer may form an abscess that leads to a fistula. The fistulas are superficial and usually 1-2cm distal to the skin tag. A probe always can go through the fistula but the internal anal sphincter is not traversed. (13)

In patients with severe pain, rectal examination may be carried out under general anaesthesia to rule out other anal pathologies such as haemohroids, fistula and tumours. (13)

Diagnosis

- 1). Patient history and physical examination will often strongly indicate the correct diagnosis
- 2). Endoscopic examination is helpful to exclude higher pathology in the rectum but may be impossible to perform adequately at the initial stages due to discomfort and pain. (3)

Differential diagnosis

The fissures in ano can be differentiated from diseases such as:

Perianal abscess: may mimic a fistula. (14)

Pruritus ani: may have mucosal splits of skin and mucous membrane secondary to irritation from the discharge. (14).

Thrombosed haemorrhoids: with this condition the patient feels a lump in the anal opening. (14)

Proctalgia fugax: the discomfort in this case is not related to bowel action, also the patient feels the discomfort high up and more deeply-seated, while that of the fissures in ano is subcutaneous. (14)

Anal haematoma: may follow surgery of haemorrhoids or trauma in the anal region. (14)

Bartholin's gland abscess in females: could resemble an anterior fissures. (14)

Tuberculous ulcer: it is small in the beginning, but grow with time with undermined margins. (14)

Tumours: these are tumours such as squamous cell carcinoma of the anus or adenocarcinoma of the rectum that invades the anal canal and anal opening. With this other symptoms may be found such as inguinal lymph nodes and a huge mass in the rectum. (14)

Syphilitic chancre: the most characteristic feature here is a symmetrical lesion on the opposite wall of the anal canal. Diagnosis is made by presence of spirochaetes found in the discharge from the wound by dark ground illumination or by the Wassermann reaction. (14)

Crohn's disease: usually has multiple lesions and the diagnosis is always made on sigmoidoscopy, which reveals multiple lesions in the rectum. (15)

Ulcerative colitis is also a differential. Colonoscopy could be done to confirm its diagnosis. (15)

Leukaemia: usually the fissures are off the expected location and are multiple. A diagnosis is by white cell count and a blood film examination. (15)

HIV infection and AIDS: there are multiple wounds in the anal region. (15)

Herpes Simplex: a viral disease affecting the mucous membrane and skin, and can cause fissures. (15)

Hidradenitis suppurativa: infection of the hair follicles around the anal opening. These produce a lot of fluid/ purse. (16)

MANAGEMENT

An anal fissure is a very painful condition, which needs prompt and adequate treatment. This will rid the patient off the agony of pain and restore comfort. (3)

Vicious cycle of the disease has to be broken to inhibit the contraction of the internal sphincters by stopping the spasms and then allowing the free drainage of the fissure or the subcutaneous abscess that may form and so allowing the healing process to take place. One should also take care of constipation to reduce the rate of repeated trauma by hard stools.

Epithelization of the fissure occurs leading to complete closure.

Complications such as fistula formation and sinus formation are therefore prevented.

Conservative

The Conservative or medical management is mostly for acute fissures. It breaks the vicious cycle and allows for the healing to occur. This includes:

The use of the **W.A.S.H.** regime in the treatment of anal fissures.

W --- Warm water shower or Sitz baths after bowel movements.

This is done two to three times in a day using warm salt water. It cleans the fissure and is used also as an antiseptic. It also has a soothing effect in the wound and reduces oedema. (3)

A --- Analgesics (3)

S --- Stool softeners. Such as diotyl sodium sulfosuccinate have been used. Bulking agents such as psyllium (Fiberall, Meta Mucil, Idonsyl) have been used. In Kenyatta National hospital we commonly use dulcolax and sennakot (3)

H --- High Fibre diet – should be encouraged to these patients especially in the initial stages when the symptoms begin, this is to avoid constipation. (3)

Following the above procedure most of the uncomplicated fissures resolve in 3-5 weeks. A chronic fissure may need frequent treatment.

Anaesthetic agents have been used. They only relieve the pain and relax the sphincters, making it possible also for one to examine the patients with very minimal pain. Lignocaine has been used; Anusol and proctosedyl have also been used. One can use them as suppositories or gels. Long acting anaesthetic agents have been discarded. It is aimed at blocking the inferior rectal nerve. (16)

Injections of botulinum toxin are being used. Botulinum toxin is an exotoxin produced by bacteria clostridium botulinum and is a potent neuro toxin that causes botulinism (5). The injections given produce a reduction in the maximum resting anal pressure, which can be sustained for two to three months and this should translate to improved healing. The research done by Giuseppe et al, randomly sampled 50 patients. Injection of 20units of botulinum toxin into the internal anal sphincter of each sides of anterior midline was done twice daily. Botulinum toxin causes denervation of the internal sphincters. It acts faster and prevents the release of acetylcholine by presynaptic nerve terminals. (17) Paralysis occurs within a few hours and the transmission of neuromuscular impulses resumes after the growth of new axon terminals. (17)

Topical application of glyceryl trinitrate ointment twice daily for 8 weeks, can be effected. Glyceryl trinitrate 0.2% ointment provides rapid sustained relief of pain in patients with chronic fissures in ano. It reduces the maximum resting anal pressure and improves pain by increasing the anodermal blood flow as well as reduction in maximum anal pressure. It has side effects such as headaches and burning sensation around the anal opening. All this side effects are short lived. The glyceryl trinitrate ointment breaks down to form Nitrous Oxide a vasodilator, which relieves spasms. This sustains relieve of spasms and the fissure heals. However this needs follow up of patient for a long time. (18)

Isosorbide dinitrate has been used as a gel or a spray applied around the anal opening. It increases the blood flow to the anal sphincter and so improves spasms. Isosorbide dinitrate pharmacological activity is attributed to its active metabolite isosorbide mononitrite. It has side effects such as headaches (7).

Calcium channel blockers such as diltiazem have been tried as a form of chemical sphincterotomy. The drugs used in this class are such as oral nifedipine 20mg twice daily. It reduces maximum resting pressure and so sustaining the healing process. It causes headaches and flushes. This are short lived (18).

Topical diltiazem 2% gel has proved to be effective with less side effects. It demonstrated more profound reduction in maximum resting pressure and therefore better healing process than glyceryl trinitrate.

Antibiotic should be prescribed to patients with fever and discharge indicating infection. (18)

Operative Management

Surgical therapy of anal fissure is reserved for patients who have failed medical therapy, or have developed a fissure-fistula. Most of the methods here involve the disruption of the internal anal sphincter. The operative methods or techniques commonly used for anal fissure include

- (1) Anal stretch well known as manual anal dilation (MAD). (19)
- (2) Open Lateral internal sphincterotomy. (19)
- (3) Closed lateral internal sphincterotomy. (19)
- (4) Tailored sphincterotomy
- (5) Posterior midline sphincterotomy + Fissurectomy. (19)
- (6) Dermal flap coverage of the fissure. (19)

Anal Stretch – M.A.D.

Anal stretch was originally described by Recamier in 1838 for the treatment of proctalgia fugax and anal fissure. (5) Such a procedure can be carried out with a local anaesthetic infiltration, but a brief general anaesthetic is preferable. Patient is placed in the lithotomy position and draped. An index finger of one hand is inserted into the rectum followed by the index finger of the opposite hand. The long finger of the same hand is inserted followed by the long finger of the opposite hand. Gentle lateral retraction with each finger commences for approximately 30 seconds. With four fingers in place, the anal canal stretched cautiously for four minutes. In men, it is easier to stretch the sphincter in the anteroposterior plain because of the narrowness of the pelvic outlet. Sphincter stretch in women should be done transversely as the disruption of the anterior sphincteric support is a real possibility. (20)

The disadvantages of the M.A.D. includes the following:

- (1) With this procedure fissure may be widened and can end up bleeding more. Widening the fissure, more micro organisms may invade the wound and septicaemia can occur. (20)

- (2) M.A.D may cause incontinence of flatus and occasionally faeces, due to interference of the complete closure of the anal opening that leaves a small hole defect due to rupture of the external sphincter fibres. (20)
- (3) Recurrence of the fissure is very common after M.A.D. (20)
- (4) A haematoma may form after M.A.D. (20)
- (5) Subcutaneous anal abscess may form latter especially when the microorganism invade the raw widened fissure after MAD. (20)

Open Lateral Internal Sphincterotomy

The first person to perform this operation was Brodie in 1839. With the pretext that when the sphincter goes into spasm the anal pressure goes up and this makes the fissure worse: (21) Hilton in 1863 gave credit to the operation due to its success. Miles believed that he was dividing what he called the band in his operation and so mistook this for the internal anal Sphincter. (21)

Eisenhammer in 1951 had to work on cadavers and then went ahead to do proper internal anal sphincterotomy by dividing the right muscles. Internal anal sphincter is the continuation of the distal portion of the circular muscles of the rectum. There is a groove in between the internal anal sphincter and the external sphincter that lies lateral. (21)

Internal anal sphincter maintains the anal canal in the closed position, action is involuntary. External sphincter is a striated muscle voluntary. The procedure is done under general anaesthesia with the patient in lithotomy position, a groove between the internal and external sphincters is palpated. An incision is made above the internal sphincter either on the left or right lateral position then with the use of a curved mosquito forceps (Halsted). It is brought out from the incision and cut by use of a knife or diathermy. The wound may be left open and patient goes on saline sitz baths twice a day. (21)

Patients may also be put in a prone Jack-knife position depending on the surgeons preference and availability of appropriate table and the buttocks strapped laterally to expose the anal region. An incision is made on the left or right and latter can be sutured with absorbable sutures. (21)

Post operatively the patient goes on analgesics, saline sitz baths. Time taken for these patients to heal is about six weeks. Complications of open lateral internal sphincterotomy are such as:

1. Ecchymosis around the entrance wound if the closed technique is used. (22)

2. A massive haematoma is usually the result of failure to apply enough pressure to the site. (22)
3. Haemorrhage may occur especially if a blood vessel is severed. (22)
4. Perianal abscess occurs in 1% of the internal anal sphincterotomies these may lead to fistula and anal discharge. (23)
5. Very rarely does incontinence of flatus and faeces occur.

Fissurectomy and midline sphincterotomy

Fissurectomy is used in chronic anal fissure. It involves the excision of the fissure in a triangular manner and removes the fibrotic tissues this will enable the wound to re-epithelialize and so heal. This method was tried long time back by Gabriel in 1948. This operation had many disadvantages, which included the following: - (24)

1. The wound left after the fissurectomy was very large and took too long to heal.
2. The anal opening is a dirty area and so during defecation the wound being raw is contaminated by organisms, which may pass to the bloodstream.
3. The condition, with this method may become worse with reduced chances of healing and may even form a subcutaneous abscess at the site of excision.
4. The patient is fed on fluid diet for not less than seven days and started on stool softeners. This is to allow the healing process to take place. (24)
5. Fistula may form after the operation. (24)
6. Keyhole deformity is another troublesome consequence of the excision. The result may produce symptoms of mucus discharge, pruritus, and soiling of the undergarments.

This method, because of the disadvantages is now obsolete.

Closed lateral internal sphincterotomy:

Closed lateral internal sphincterotomy almost the same as open lateral internal sphincterotomy, and is more recent. (25) It was first tried by Park in 1967 and supported by his colleague Sohn and others. (25)

The method does not need bowel preparation although is necessary. It can be done in a clinic under local anaesthesia, with the patient in a jack knife position or lithotomy position.

General anaesthesia is recommended because all muscles are relaxed with patient in lithotomy position. A finger is put in the anal canal. Internal sphincter palpation is done and the junction between it and the external sphincter delineated. (25)

A blade is passed between the mucosa and internal sphincter up to the junction of the upper third and lower two thirds of the internal sphincter (Dentate line), and then rotated laterally cutting the internal muscle with manual assistance. (25)

Pressure is then applied for two minutes over the mucosa to stop haemorrhage. Sofratule on gauze may be inserted in the opening to stop haemorrhage and removed later when the patient is awake. Patients later go home with stool softeners, analgesics and instructed to have daily sitz baths.

Sentinel tags or piles, oedematous tissue of skin above the fissure should be excised to allow drainage of the fissure.

The advantages in this case outweigh the disadvantages in that:

1. The healing is faster (26)
2. The discomfort is not there post operatively and the anal pressure falls immediately. (26)
3. Incontinence is a rare entity in this case as documented by Callopy and Ryan in 1978. They found out that a very small percentage of patients were incontinent. (26)

The disadvantages of the method include:

1. Haematoma formation at the site (26)
2. Injury to nerves and major vessels as it is done in a closed manner. (26)
3. One may not be sure to have done complete division of the muscle (26)
4. There may be severe bleeding especially when a vessel is severed. (26)

Tailored sphincterotomy

This procedure differs from standard lateral internal sphincterotomy in that the division of the sphincter is more conservative and is carried out cephalad only for the length of the fissure rather than to the dentate line. Flatus and solid incontinent rate are very low and recurrence has not been reported and (7).

Dermal flap coverage of the fissure:

Dermal flap coverage is a new method in proctology. (27). A flap is rotated from the skin side taking care not to sever any of the sphincters and rotate it to the fissure after freshening the edges and suture. The hygiene is of paramount importance here. Rotated flaps carries its own blood supply. (28)

Disadvantage of the method is that the failure rate may be high due to hindered drainage from the flap and also abscess formation below the flap leading to failure of the flap to take. Flap failure is attributed to the increased pressure that is not relieved. (28.29)

AIM OR MAIN OBJECTIVE

The aim of the study was to evaluate the outcome of lateral internal sphincterotomy as compared to manual anal dilatation as a basis for future practice.

SPECIFIC OBJECTIVES

1. To compare the outcomes of the procedures done (Manual Anal Dilatation and Lateral Internal Sphincterotomy).
2. To determine the length of hospital stay after lateral internal sphincterotomy and manual anal dilation.
3. To determine complication rates between the two methods.

JUSTIFICATION/ RATIONALE OF THE STUDY

Anal fissure is a common anorectal condition. It is associated with spasms of the internal anal sphincter. Several methods both surgical and pharmacological exist in the management of fissures. Comparison of the two surgical methods (lateral internal sphincterotomy and manual anal dilatation) will be of importance so as to show the better modality of treating this condition. The study in this region is an attempt to shift from manual anal dilatation to lateral internal sphincterotomy as a mode of fissure management. A similar study was done at Kenyatta National Hospital on retrospective basis 13 years ago and viewed fissures in ano treatment in general. Today there is need to update ourselves in the current status of fissure management.

METHODS AND PATIENTS

Study design and setting

This was a nine months (June 2004 – February 2005) hospital based prospective study comparing lateral internal sphincterotomy to manual anal dilatation. The research was conducted at KNH surgery department, the main outcomes of interest were duration of symptoms, complications, other previous interventions, duration of hospital stay, sex distributions, location of the fissure, distribution of the disease as per age group, recurrence of the disease post surgery. And lastly, complications that arose due to the surgery, not forgetting other local and systemic diseases that are associated with the disease. Patients were admitted through SOPC to either ward 5A, 5B or 5D. Those who did not meet eligibility as per the criteria of inclusion were not recruited into the study, all patients were clerked, examined by the investigator and a diagnosis of fissure in ano was confirmed.

Consent was taken from them as per the questionnaire. They were all investigated and prepared for surgery by taking blood for urea and electrolytes and creatinine. Haemoglobin levels were also done. The procedure was explained to the patients before surgery. Choice of the procedure was selected by the consultants surgeon assisted by the investigator. The choice of the anaesthesia depended on the availability of the drugs. The scrub nurse set the instrument and brought them for the surgeon and the investigator to use depending on the procedure. Soap enemas were given on the morning of surgery including stool softeners (dulcolax) prescribed 48 – 72 hours prior to surgery.

After anaesthesia patients were put in lithotomy position, cleaned and draped. For MAD the goal being to reduce sphincter tone by controlled manual stretching of the internal sphincter. After insertion of the right index finger, the left index finger followed. Both the two were held in position for thirty seconds. These were followed by the right long finger and then the left long finger. Eventually four fingers were in position and held for thirty seconds to one minute. This led to lateral destruction of the anal sphincter. In women this was done transversely. The anal canal was compressed with sufratule and a small piece of gauze. For LIS after draping the patient, the fissure was identified and a lateral incision was made. A halsted artery forcep was used to bring out the sphincter in the wound, which was subsequently divided either by use of diathermy or knife. One stitch was placed on the wound and one stitch applied. Compression was done with sufratule and gauze. Patient was reversed for further observation in the ward by the nurses and the investigator. After the procedure the anal opening was compressed with sufratule and a small piece of gauze. Post operatively all patients were started on pethedin 50mg and declofenac 50mg as start doses to

continue with oral analgesics. Prophylactic and biotics were prescribed for 48 hours. In lateral internal sphincterotomy the wound was closed with one stitch before compression. Saline sitz baths were commenced from day two, twice a day and after bowels. Surgical complications, visual analogue pain score (1-10) during hospital stay were recorded. At day one, two weeks and four weeks post treatment all patients were reviewed for fissure healing and complications. The pain was assessed by visual analogue without asking the patients then later asked how they were feeling. They were asked how they felt how much analgesic they were taking.

These was recorded against the procedures that were carried out on them as cured, better, same, pain and worse. Once stable with no complaints they were discharged off the clinic after four weeks. The decision to discharge them was done by the consultant surgeon of the unit and the investigator. The files were all marked and kept aside for further scrutiny.

Acute fissure was defined as a superficial ulcer in the anoderm with sharply demarcated edges with a duration of less than six weeks while chronic fissure was defined as benign indurated ulcer with undermined edges with visible internal sphincter fibres. The duration of symptoms was more than six weeks. Chronic fissures were associated with skin tag. Healing was defined as complete disappearance of clinical symptoms and re-epithelization of the anal canal mucosa.

Study area

Patients were sampled at random according to ward admissions. The study groups were patients who meet eligibility in wards 5A, 5B and 5D – Kenyatta National hospital.

To undergo lateral internal sphincterotomy or manual anal dilatation the eligibility criteria was:

- i) A patient diagnosed to have fissure in ano and granting an informed consent to participate in the study.
- ii) Free from complications such as haemorrhage or sepsis

CRITERIA OF EXCLUSION FROM THE STUDY

- Paediatric patients were excluded from the study
- Those seen earlier in our surgical outpatient clinic and surgical wards with a diagnosis of fissures in ano before the study began were left out.
- Patients without proper and convincing information about the diagnosis and had other anal diseases at the initial examination stages were left out of the study.
- Those with pre existing faecal incontinence were excluded.
- Those with previous anal sphincter surgery were left out.

CRITERIA OF INCLUSION INTO THE STUDY

- 1) All patients who were seen during the period of the study with fissures in ano
- 2) Had no other bowel diseases at the initial examination.

LIMITATIONS OF THE STUDY

The study was conducted at Kenyatta National Hospital over a period of nine months (June 2004 – February 2005). Procedures were carried out under spinal or general anaesthesia. Limiting factors were such as admission criteria where the patients had to pay a mandatory deposit which some felt was high. Long waiting queues before surgery was also a limiting factor. The study was carried out at the time when Mbagathi Hospital was opened as a District Hospital, so some patients were directed there making it difficult for us to reach them. During counselling, some patients felt that we could be investigating them for AIDS and so refused to enter into the study. This was a single centre study and so the catchments area was limited. Due to the economic hardships some patients did not come to the clinic for review as they were coming from outside Nairobi.

$$n = \frac{\left\{ Z_{1-\alpha/2} \sqrt{2P(1-P)} + Z_{1-\beta} \sqrt{P_1(1-P_1) + P_2(1-P_2)} \right\}^2}{P_1 - P_2}$$

Where; n = sample size to be determined

Z = standard errors from mean (Z_{1-α/2} = 1.96)

α = level of significance (Z_{1-α} = 1.645)

β = level of significance <80 = 0.842, >90 = 1.282

P₁ = proportion developing complications with method 1 (MAD)

P₂ = proportion developing complications with method 2 (Lateral internal

Sphincterectomy)

d = absolute precision (5% or 0.05 as a proportion)

Assumption value MAD = 50% } Complications arising as per previous studies.
 Calculated value LIS = 20% }

Therefore complications of MAD are more than LIS.

ETHICAL CONSIDERATIONS:

1. Informed consent was granted by the patient to participate in the study. Nobody was otherwise included in the study
2. Strict confidentiality was ensured to safeguard the privacy of the patient.
3. The information gathered was used for the disclosed purpose of the study only and for no other reason.
4. Data entry was by code numbers and not by names
5. Approval to conduct the study was sought from the research and ethics committee of Kenyatta National Hospital and the study only begun once this was granted. This was on 31st May 2004.



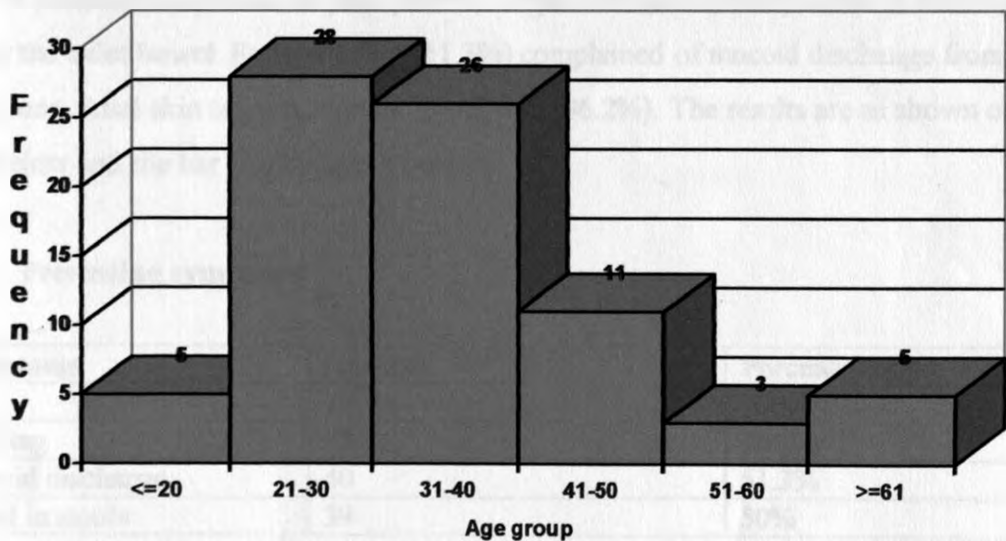
RESULTS

In this study a total of 78 patients were seen with a diagnosis of fissure in ano. Of these 78 patients 40 underwent lateral internal sphincterotomy while 38 underwent manual anal dilatation (Lords procedure). Patients were operated on by a consultant surgeon of the said unit assisted by the investigator

Age distribution

The age distribution of the patients who underwent the procedures are shown in figure 1 below. The youngest patient was 17 years old and the oldest was 82 years. The highest number of patients were in 21 – 40 age group (56 (68.2%)) The mode was 28 years found in ages 20 – 30 years.

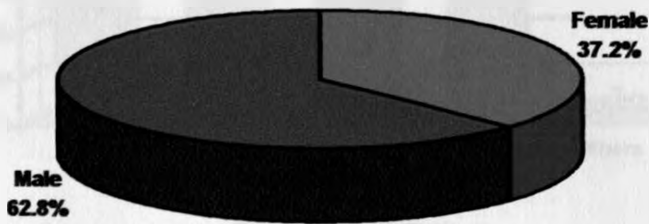
Figure 1: Age distribution



Sex distribution

Forty-nine patients were males (62.8%) while 29 were females (37.2%) as shown in figure 2 below. The ratio of male to female was 1.66:1

Figure 2: Sex distribution



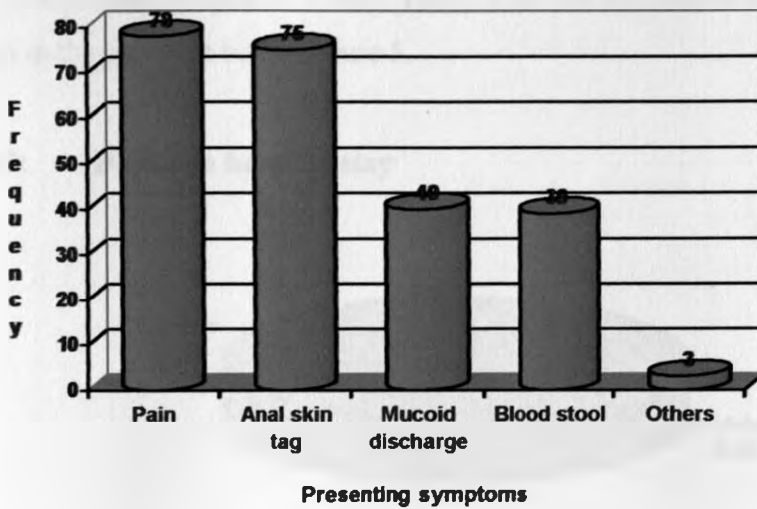
Presenting symptoms

All the 78 patients complained of pain (100%). 39 patients (50%) complained of blood on stools or the toilet bowl. Forty patients (51.3%) complained of mucoïd discharge from the anal opening. Anal skin tag was seen in 75 patients (96.2%). The results are as shown on table 1 below and the bar chart figure 3 below.

Table 1. Presenting symptoms

| Symptoms | Number | Percentage % |
|------------------|--------|--------------|
| Pain | 78 | 100% |
| Anal tag | 75 | 96.2% |
| Mucoïd discharge | 40 | 51.3% |
| Blood in stools | 39 | 50% |

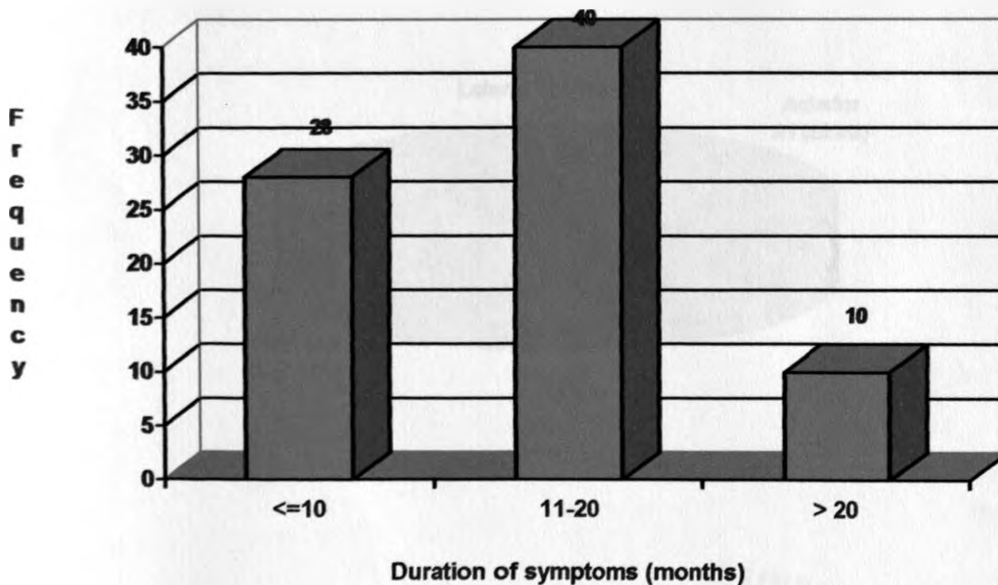
Figure 3: Presenting symptoms



Duration of symptoms prior to presentation to hospital

Patients took long before presenting themselves to hospital. The shortest duration was seen in one patient (2 months). The longest duration was seen in two patients (36 months) The mode was 40 patients seen between 11 and 20 months. The mean was 12.6 months. The results are as shown in figure 4 below

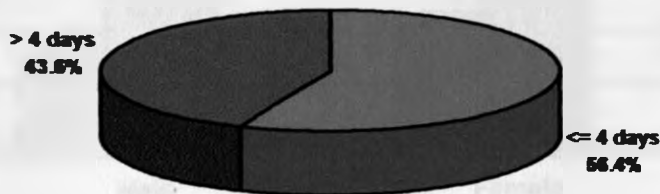
Figure 4: Duration of symptoms prior to presentation in hospital



Duration of hospital stay (grouped)

In the study group of 78 patients who underwent the procedures (MAD, LIS), 44 (56.4%) stayed for less than or equal to 4 days. Thirty-four (43.6%) stayed for more than 4 days. This is shown in the pie chart below figure 5.

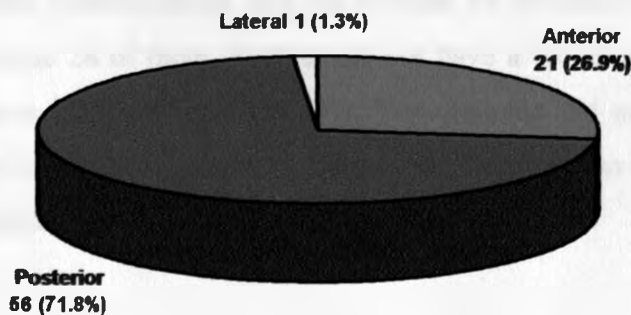
Figure 5: Duration hospital stay



Location of fissures

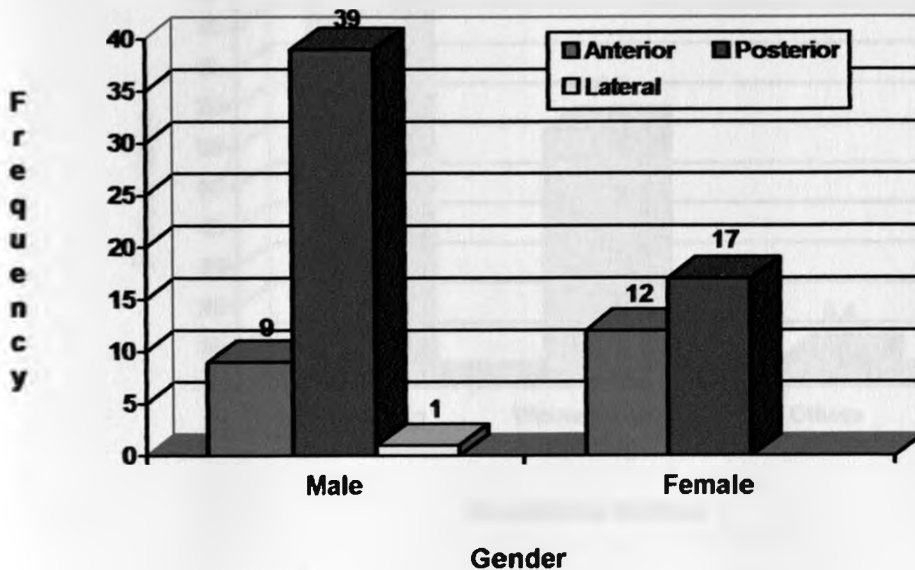
Fifty six patients were found to have fissures posteriorly placed (71.8%) while 21 patients (26.9%) had them anteriorly placed. One patient had fissure laterally placed (1.3%) the results are as shown below on the pie chart figure 6.

Figure 6: Location of the fissure



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Figure 7: Location of fissures by gender

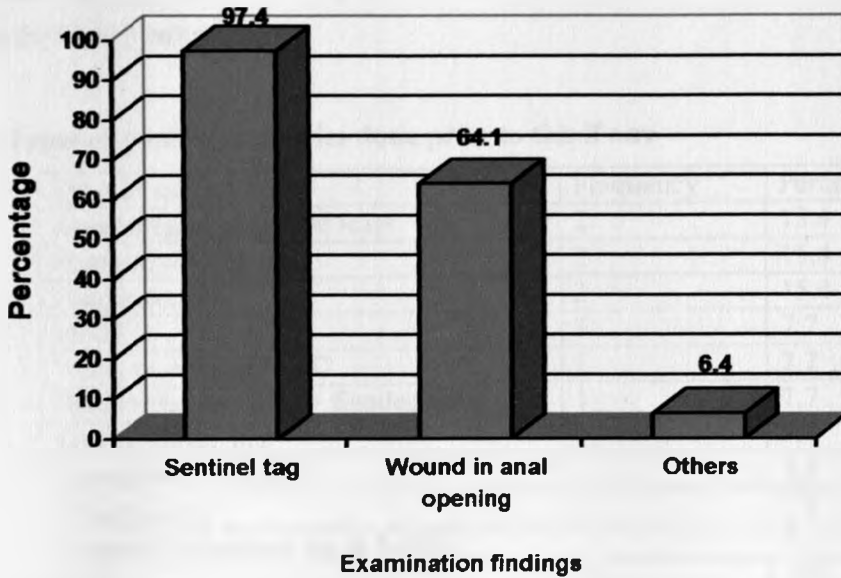


in the study group of the 78 patients, nine males had fissures anteriorly placed (11.5%) while 12 females had them anteriorly placed (15.4%). Thirty-nine males (50.0%) had them posteriorly placed while 17 females (21.8%) had them posteriorly placed. One male (1.3%) had the fissure laterally placed, the results are shown on the bar chart figure 7.

Examination findings

Seventy eight patients were reviewed, and out of these 50 of them (64.1%) had a wound in the anal opening, while 28 of them (35.9%) did not have a wound on the anal opening. A sentinel tag was seen in 76 patients (97.4%). Two patients did not have a sentinel tag. Induration was seen in one patient, haemorrhoids were found in two patient. Other swellings were found in one patient. This is shown on the bar chart figure 8.

Figure 8: Examination findings



Associated diseases to anal fissures

Seventy eight patients who were seen, of whom, 9 of them had haemorrhoids associated (11.6%). Fistula in ano was seen in one patient (1.3%), anal warts in one patient (1.3%), Vaginal tears at delivery in one patient (1.3%). These results are tabulated in table 2.

Table 2: Associated diseases to anal fissures

| Primary disease | Frequency | Percent |
|--------------------------|-----------|--------------|
| Haemorrhoids | 9 | 11.6% |
| Anal warts | 2 | 2.6% |
| Fistula in ano | 2 | 2.6% |
| Vaginal tear at delivery | 1 | 1.3% |
| Total | 14 | 18.1% |

Types of perineal surgeries done prior to this study

Fissure in ano may also follow some perineal surgeries as a complication. This has been shown on the table 3 below.

Table 3: Types of perineal surgeries done prior to this if any

| Type of surgery | Frequency | Percent |
|----------------------------------|-----------|---------|
| Accidental or perennial tears | 2 | 15.4 |
| Haemorrhoidectomy | 2 | 15.4 |
| Fistulectomy | 2 | 15.4 |
| MAD | 1 | 7.7 |
| Warts excision + MAD | 1 | 7.7 |
| Haemorrhoidectomy + fistulectomy | 1 | 7.7 |
| Tear of suture line | 1 | 7.7 |
| Resection of warts | 1 | 7.7 |
| Fissurectomy | 1 | 7.7 |
| Removal of sentinal tag & MAD | 1 | 7.7 |
| Total | 13 | 100 |

TYPES OF TREATMENTS

Conservative methods used – Post Surgery

Of the patients seen seventy-one (91%) were treated on the W.A.S.H. regime. Forty-nine of them were treated with anaesthetic suppositories. The results are tabulated on table 4.

Table 4: Conservative methods used

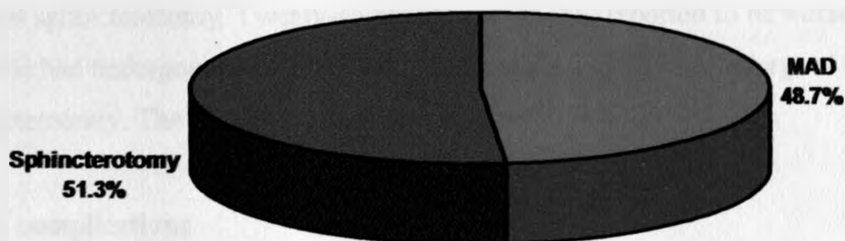
| Conservative methods used | Frequency | Percent |
|------------------------------|-----------|---------|
| WASH | 71 | 91.0 |
| Anaesthetics (Suppositories) | 49 | 62.8 |

Some of the patients underwent both conservative methods of WASH and Anaesthetics Suppositories simultaneously.

Operative methods used

The author reviewed 78 patients and out of this, 40 (51.3%) underwent lateral internal sphincterotomy while 38 patients (48.7) underwent manual anal dilatation. The results are as shown on the pie chart figure 9.

Figure 9: Operative modalities



Operative methods used versus duration of hospital stay

Seven patients (18.9%) who underwent manual anal dilatation stayed for less than four days while thirty-one (81.1%) stayed for more than four days. Thirty-seven (90%) underwent lateral internal sphincterotomy stayed for less than four days while three stayed for more than four days. The results are as shown below in table 5.

Table 5: Operative methods used versus duration of hospital stay

| | Duration of hospital stay | | Total |
|-----------------------|---------------------------|------------|-----------|
| | <= 4 days | > 4 days | |
| MAD | 7 (18.9%) | 30 (81.1%) | 37 (100%) |
| Sphincterotomy | 37 (90.0%) | 4 (10.0%) | 41 (100%) |
| Total | 44 (56.4%) | 34 (43.6%) | 78 (100%) |

Operative methods used versus outcome

Seventy eight patients were reviewed and of these, 41 (52.6%) reported cured or better. Five (13.2%) had undergone manual anal dilatation while 36 had undergone lateral internal sphincterotomy. Nine patients (11.5%) reported no change. Seven (18.4%) from the nine who reported no change had undergone manual anal dilatation while 2 (5%) had undergone lateral internal sphincterotomy. Twenty-eight patients (35.9%) reported to be worse. Of these, 26 (68.4%) had undergone manual anal dilatation while 2 (5%) had undergone lateral internal sphincterotomy. The results are as shown in table 10.

Early complications

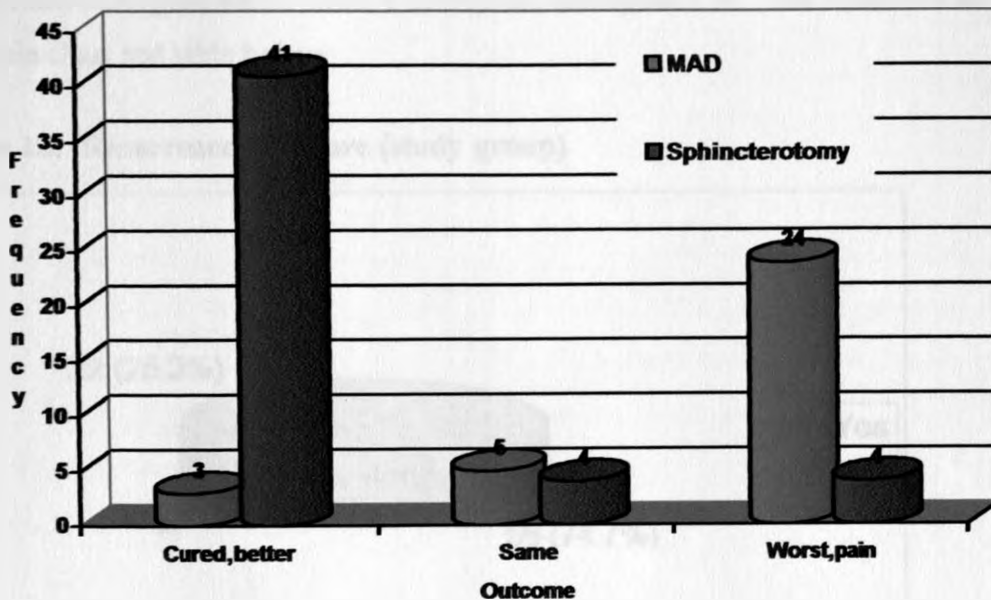
Eighteen patients from the 38 who underwent MAD had minimal bleeding to warrant change of the pack three times in 24 hours. At any one time the author used one rytec gauze. 20 did not bleed.

Forty patients underwent LIS, of this 3 bleed. Therefore the total number of patients who bleed were 21. Three patients who underwent MAD developed haematoma and five who underwent LIS developed haematoma totalling to eight. The results are as tabulated in table 6.

Table 6: Early complications

| Complication | MAD | LIS | Total |
|--------------|------------|----------|------------|
| Bleeding | 18 (23.1%) | 3 (3.8%) | 21 (26.9%) |
| Haematoma | 3 (3.9%) | 5 (6.4%) | 8 (10.3%) |

Figure 10: Operative method used versus outcome



Complications at four weeks follow up, patients with complications (n=46)

Forty-six patients developed complications such as incontinence of flatus, abscess formation, incontinence of stool and discharge (pruritus ani). In the study group that underwent manual anal dilatation had 28 patients (75.7%) with incontinence of flatus, 23 patients (62.2%) with abscess formation 21 patients (56.8%) with incontinence of stool and sixteen patients (32.2%) with discharge (pruritus ani). Complications which were seen in those patients who underwent lateral internal sphincterotomy were as mentioned above. Five patients (55.6%) had incontinence of flatus, two patients (22.2%) had abscess formation non had incontinence of stool but four patients (44.4%) had discharge. Thirty two patients out of the study group of 78 did not have complications.

The results are illustrated in table 7.

Table 7: Comparison of complications at four weeks follow up (n=46)

| Complication | MAD | LIS | Total |
|--------------------------|------------|-----------|------------|
| Incontinence of flatus | 28 (75.7%) | 5 (55.6%) | 33 (71.7%) |
| Abscess formation | 23 (62.2%) | 2 (22.2%) | 25 (54.3%) |
| Incontinence of stool | 21 (56.8%) | 0 (0.0%) | 21 (45.7%) |
| Discharge (Pruritus ani) | 16 (32.2%) | 4 (44.4%) | 20 (43.3%) |

Seventy-eight patients were seen, and out of these 22 (25.3%) had recurrences, 56 patients (74.7%) did not have recurrences. Twenty-two patients had recurrence and out of these, 14 (36.8%) had undergone MAD while 8 (20%) had undergone LIS. The results are as shown on the pie chart and table below.

Figure 12: Recurrence of fissure (study group)

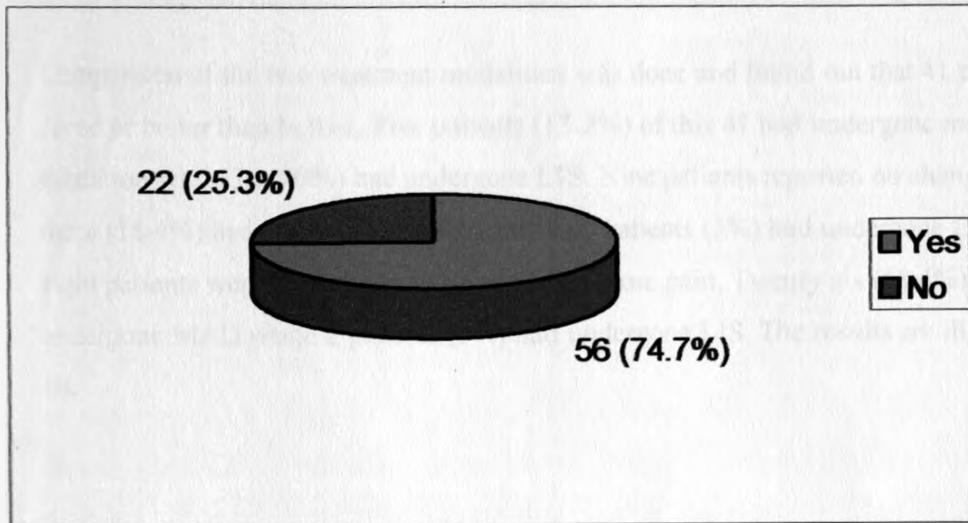


Table 8: Recurrence: MAD & LIS compared

| | MAD | LIS | Total |
|------------|------------|------------|--------------|
| YES | 14 (36.8%) | 8 (20%) | 22 (25.3%) |
| NO | 24 (63.2%) | 32 (80%) | 56 (74.7%) |
| | 38 (100%) | 40 (100%) | 78 (100%) |

OUTCOME

In the study group of 78 patients 25(32.1%) reported that they were cured. Sixteen (20.9%) reported that they were better than before, Nine patients did not notice any change while four (5.1%) felt a lot of pain. Twenty four patients (30.7%) indicated that they were worse. The results are illustrated in table 9.

Table 9: Outcome at four weeks follow up (n=78)

| Outcome | Frequency | Percent |
|---------|-----------|---------|
| Cured | 25 | 32.1 |
| Better | 16 | 20.9 |
| Same | 9 | 11.5 |
| Pain | 4 | 5.1 |
| Worse | 24 | 30.7 |
| Total | 78 | 100 |

Comparison of the two treatment modalities was done and found out that 41 patients were cured or better than before. Five patients (13.2%) of this 41 had undergone manual anal dilatation while 36 (90%) had undergone LIS. Nine patients reported no change, seven of these (18.4%) had undergone MAD while two patients (5%) had undergone LIS. Twenty eight patients were worse than before and felt more pain. Twenty six (68.4%) of them had undergone MAD while 2 patients (5%) had undergone LIS. The results are illustrated in table 10.

Table 10: Outcome at four weeks follow up versus operative methods

| | Operative methods used | | Total |
|--------------|------------------------|----------------|------------|
| | MAD | Sphincterotomy | |
| Cure, better | 5 (13.2%) | 36 (90%) | 41 (52.6%) |
| Same | 7 (18.4%) | 2 (5%) | 9 (11.5%) |
| Worst, pain | 26 (68.4%) | 2 (5%) | 28 (35.9%) |
| Total | 38 (100%) | 40 (100%) | 78 (100%) |

DISCUSSION

Anal fissure is one of the main proctological disorders encountered in general surgical practice. It may be wrongly diagnosed as haemorrhoids and perianal fistula. Despite the lesion's small size can cause great discomfort and pain which sometimes becomes incapacitating. Spasms of the anal sphincters have been noted in association with anal fissures, and for many years the aim of the treatment has been to reduce hypertonia of the sphincters. Operation techniques commonly used for fissure in ano include, anal stretch, open lateral internal sphincterotomy, closed lateral internal sphincterotomy, posterior midline sphincterotomy and to a lesser extent dermal flap coverage of fissure (27). In our study we compared two methods, which were gentle dilatation of the anal opening and open lateral internal sphincterotomy. Both methods work by reducing resting anal pressure to normal. As occurred in our series anal fissure is reported to affect more males than females. From studies done by Parklea in 1992 showed that the incidence of this disease in our SOP Clinic was 2.6: 1000 in Kenyatta National Hospital (14).

In our study we had 62.8% men and 37.2% women, this when calculated tells us that anal fissure affects more men than women in our country i.e. M: F 1.6887: 1, this contradicts the studies done in European clinics and Asian clinic where the ratio is 1: 1 (14)

Marvin L. Corman also noted that men and women are equally affected to the ratios of 1:1 (15). Studies done in Asia have also yielded the same results. The most recent studies done in Madrid (2004) tallies with our results well. They found that 65% were men while 35% were women (16). The condition is very common in the second and third decade, dropping slightly in the fourth decade. The mean age is 34.92 yrs of the patients who were seen, while the median is 32.00 yrs. The mode was found to be 24.00 years of age. According to studies done in our hospital in 1992 it was noticed that the average age was 29 years. During the study more males were seen than females. In our study, the predominating group are males of 34 years and females between 28-30 years. (15)

From our results, it is clear that the majority of patients are between 21yrs of age and 40yrs of age. It is less common below 24yrs of age and above 50yrs of age. From studies done in America, the age bracket was 20yrs, but with our people, the age bracket was from 30yrs onwards. (2)

The Asian community has an age bracket of 35 years. (15) With the condition being seen more in the 4th decade. It is rare in the elderly and children.

The possible explanations for the disease being rare in the young people and the aged is not very clear, but a few theories may be formulated.

The author may say that, in the old it is rare because of the muscular atony that develops with age.

Working in hot climatic conditions makes our people more dehydrated and so leaves the stools very hard to injure the anal mucosa.

Anal pain and anal skin tag are the commonest symptoms, others are such as mucus and blood in stools. This tallies well with other studies done by the Europeans. Anal pain in our study was seen in 100% of patients while anal tag was seen in 96% of patients. This tallies well with the European studies of 94% and 84% respectively. (15) Blood on stools was seen in 50% of patients while mucoid stools were seen in 51.3% of patients. In the Caucasians population, these are only reported to occur. Pruritus ani, appetite loss and other discharges are less frequently seen. In our setup, patients come in chronic stages and so these are seen. (15)

The studies done in the west have yielded the same results like ours, pain being the main symptom seen in all the patients, anal tag seen in all of them as well. This is because of the healing process of epithelization. (16)

Our patients take too long before they are seen in our clinics. On average they take upto 12 months. The shortest duration seen in our clinic was 2 months while the longest was 36 months. The majority presented to the clinic after 12 months. This was found to be 33.3% of the patients. This long duration of symptoms makes the whole process chronic. Pain and bleeding are the main symptoms but pain is not as severe as that with acute fissure. Frequently the patients symptoms are attributed to secondary changes such as the presence of a lump in the anal opening (23). The mean duration of symptoms from other researches done in Spain was 17.6 months (range 1-70) The possible explanations why the patients presented late could be:

Many of them presented to other health institutions, treated for different anal diseases before they got to us.

The perineum is an area where people may not want to mention to be sick and so patients take long before coming to hospital. Before the onset of HIV/ Aids the condition was there but people had the reluctance of talking about ones own anus and this led to delay in diagnosis. (14)

The long appointments also extend the duration.

Many patients have known that perinial ulcers are a presentation of HIV/ AIDS and so many will stay away in the fear that they may be screened for the same since it is still a stigma in our community.

Majority of the patients who were seen had fissures posteriorly. Fifty Six of the patients (71.8%) (39 males and 17 females) had fissure posteriorly. These outcomes have also been seen in Europe and North America where studies have been done (1,3,11). In studies done in Madrid, they found that 65% men and 35% women had fissures posteriorly while 60% women and 39% men had them anteriorly while 1-2% of the population had them laterally, both men and women alike (20).

For fissures to heal, the vicious cycle must be broken, this happens very rarely – only in acute cases.

The vicious cycle as above can only be broken if one is constantly on treatment, but this is not the case here. Some people do not follow the instructions. For the patients using suppositories, occasionally may have no money to buy the drugs. So the problem may persist. Comparison was made between two methods: manual anal dilatation and lateral internal sphincterotomy. The author looked at the outcome of the procedures, the advantages of lateral internal sphincterotomy and manual anal dilatation as well as the complication rates between the two methods. The length of stay of the patients in hospital after the procedure was also analysed by grouping them into two as per their duration of stay in hospital post surgery. We grouped our patients into two, those who stayed longer than four days after surgery and those who stayed for less than four days. Patients who underwent manual anal dilatation seven (18.3%) stayed for less than four days while 30(81.1%) stayed for more than four days. This is costly to the patients and a lot of time is wasted in a hospital bed. In lateral internal sphincterotomy, thirty-seven (90.0%) stayed for less than four days while four (10%) stayed for more than four days. Complication rates in manual anal dilatation were more than in lateral internal sphincterotomy. Incontinence of flatus and stool, abscess formation, discharge (pruritus ani) were some of the complications. Others were such as bleeding from the tears, fibrous formations and anal stenosis and haematoma formation post procedure.

This tallies well with the western literature. Those who underwent lateral internal sphincterotomy improved well, termed as epithalization of the fissure and absence of symptoms as before (20). Recurrence rate in manual anal dilatation was also found to be higher than in lateral internal sphincterotomy (20).

In manual anal dilatation the patients complicated as above and so they were to be observed longer and the complications treated. While in lateral internal sphincterotomy the complications were few. Others did not complicate.

Majority of the patients who were done manual anal dilatation complained of a haematoma formation and anal abscesses. This were sorted out and led to the long stay in hospital

Some patients who underwent manual anal dilatation developed severe bleeding. This was because of the atony of the sphincter due to the dilatation. In lateral internal Sphincterotomy there were none who bleed. This has been shown in the Western literature (29).

Incontinence of stool was seen in 71% of the patients who underwent manual anal dilatation, as well as incontinence of flatus. This depressed the patients so they needed counselling and further treatment of the complications.

Review of the patient after four weeks follow up reflected that more complications were seen in patients who had undergone manual anal dilatation than lateral internal sphincterotomy. Results tally well with the Western Literature that more complications are seen in manual anal dilatation

Those Patients who developed anal stenosis had undergone manual anal dilatation and so the wounds created during the procedure healed with fibrosis leading to Stenosis. This is clear in the Western Literature (30,31).

CONCLUSION

- 1) The incidence of fissures in ano should be higher than what we have found in our study. This is because of the short duration of the study.
- 2) Our study shows that the number of men with fissures is higher than that of women. This is because more men were seen with the condition during our study than the females. The ratio of male to female was 1.66:1.
- 3) Our patients present themselves to a surgeon very late and in the chronic stage, which does not respond to conservative management.
- 4) Most of the patients are labelled to have haemorrhoids; this is because of the old belief that any pain and bleeding around the anal opening are due to haemorrhoids. Misdiagnosis has played a bigger role here. Some clinicians do not examine their patients properly especially when it is an acute fissure and with a lot of oedema and pain.
- 5) Some perineal surgeries lead to anal fissures especially haemorrhoidectomy when not well done and the wound heals leaving some areas bare then end up as fissures.
- 6) Complications are seen to be more rampant with manual anal dilatation than lateral internal sphincterotomy. These are such as incontinence of flatus and stool, Abscess formation and pruritis ani.
- 7) Recurrence was found to be more with manual anal dilatation than open lateral internal sphincterotomy.

- 8) Our operative management from the study is more superior now to the conservative management because many of them come in chronic stages. Lateral internal sphincterotomy then is the superior method.
- 9) Surgery can be done under spinal anaesthesia or local anaesthesia as per the western literature.
- 10) Conservative management cannot work in chronic anal fissures. It can only work in acute fissures because the vicious cycle can easily be broken in acute cases than chronic cases.

RECOMMENDATIONS

After a long struggle to find out the best operative method, I highly recommend that:-

- 1) Lateral internal sphincterotomy should be the method to be adopted in our set up and around the country as it gives the best results ie: shortest hospital stay, less complications, can be carried out in a clinic under local anaesthesia and as a day case.
- 2) To avoid recurrence, we recommend that before surgery, our patients are given a health education talk about their disease, the surgery to be done, the diet, post surgery care and when they should start taking their daily duties.
- 3) The author recommends that the simplest anaesthetic methods be used such as local anaesthesia and spinal anaesthesia. This will reduce the morbidity of the patients. This also reduces the expenses and time that the patient will stay in hospital occupying a hospital bed.
- 4) The anatomy of the anal region is of importance to understand fissures in ano, this should be taught at both district and provincial levels before one can start the procedure of lateral internal sphincterotomy. Doctors at internship should also be taught this method.
- 5) We advice that research on this painful condition be done at other levels countrywide. This will give us a proper picture of fissure in ano in the country. Our study only looked at fissures at Kenyatta National Hospital
- 6) Publications of literature about fissures in ano to the districts and provincial levels about the new modes of management.

QUESTIONNAIRE

1. Patients Personal details

- Name
- Age:
- Weight:
- Sex:
- DOA:
- DOD:

2. Presenting Symptoms

- Pain in the anal area
- Blood stool
- Mucoid discharge
- Anal skin tag
- Others

3. Duration of symptoms prior to presentation in hospital

4. Examination findings

- Wound in the anal opening
- Sentinel tag
- Others

5. Location of the fissure

- Anterior
- Posterior
- Others

6. Any primary diseases associated with anal fissure:

.....
.....
.....

7. Types of perineal surgeries done prior to this if any:

.....
.....
.....

8. Complications at least four weeks follow up outcome (cured, better,same,worst,pain, ulceration)

- Incontinence of flatus

- Incontinence of stools
- Discharge (Pruritus ani)
- Abscess formation
- Others

9. Recurrence formation Yes No

10. Operative methods used

- MAD
- Sphincterotomy

11. Conservative methods used

- W.A.S.H
- Anaesthetics (Suppositories)

I do hereby acknowledge that I have been given adequate information about the purpose of the study. I have understood its importance and benefit to the medical personnel and the general public. I am also informed that the participation in or withdrawal from the study will not result in discrimination against me in terms of treatment and care. I am further informed that the information obtained shall be treated with confidentiality. Refusal to sign this consent will not interfere with your management.

Signed _____

Witness _____

Patient/ Guardian

Investigator/ Assistant

Date _____

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Date: 31 May 2004

Dr. Daniel O Alushula
Dept. of Surgery
Faculty of Medicine
University of Nairobi

Dear Dr. Alushula

**RESEARCH PROPOSAL "FISSURES IN ANO PROPOSAL FOR A PROSPECTIVE STUDY
AT KENYATTA NATIONAL HOSPITAL" (P122/10/2003)**

This is to inform you that the Kenyatta National Hospital Ethics and Research Committee has reviewed and **approved** the revised version of your above cited research proposal for the period 31 May 2004 – 30 May 2005. You will be required to request for a renewal of the approval if you intend to continue with the study beyond the deadline given.

On behalf of the Committee, I wish you fruitful research and look forward to receiving a summary of the research findings upon completion of the study.

This information will form part of database that will be consulted in future when processing related research study so as to minimize chances of study duplication.

Yours sincerely,

PROF. A N GUANTAI
SECRETARY, KNH-ERC

Cc Prof. K M Bhatt, Chairperson, KNH-ERC
The Deputy Director (C/S), KNH
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