CONTENTS

A study of chronic subdural haematomas at Kenyatta National Hospital Nairobi.

Dissertation submitted in part fulfillment for the degree of Masters of Medicine (Medicine)

University of Nairobi 1979

BY

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This dissertation is my original work and has not been produced in any other study before.

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CHRONIC SUBDURAL HAEMATOMAS

Presentation, diagnosis, management and results of chronic subdural haematomas seen at the Kenyatta National Hospital from January 1976 to December 1978.

SUMMARY

All patients presenting with chronic subdural haematomas over the three year period to Kenyatta National Hospital, Nairobi have been reviewed. Of the fifty-one patients five were diagnosed at postmortem. Of the remaining forty-six patients who were operated seven died.

Diagnostic failure was caused mainly by failure to consider the possibility of a chronic subdural haematoma.

An attempt has been made to characterise the clinical pattern that may suggest the presence of chronic subdural haematoma.

THE PRINCIPAL AIMS AND OBJECTIVES OF MY STUDY WERE:-

1. To determine the incidence of chronic SDH at Kenyatta National Hospital over a 3 year period.
2. To study the clinical presentation, diagnosis and management of chronic SDH over this period.
3. To evaluate cases which were missed so that in future our diagnostic acumen could be improved.
4. To localise factors which caused delay in treatment so that our future management of these patients could be improved.

INTRODUCTION

Chronic Subdural Haematomas (SDH) is a relatively common condition. Its particular importance is that because of the bizzare presentation the diagnosis is often not obvious and yet it is vital. Correctly diagnosed and treated progress is excellent. As these patients have relatively little associated brain damage timely surgical intervention is most rewarding. Missed diagnosis often results in death.

Subdural Haematomas are usually classified into acute, subacute and chronic but the limts of each group vary (Mckissock, Richardson and Bloom 1960, Gilmartin 1964). At this institute SDH was regarded as acute if the patient presented within 3 days of injury, subacute if after 3 days but within 3 weeks and chronic if after 3 weeks or without any history of head injury (Rosenbluth et. al 1962, Alexander 1964).

Patients with acute and subacute Subdural Haematomas are not included in this study.

EPIDEMIOLOGY IN AFRICA

UGANDA

Subdural Haematomas are said to be common in East Africa. One of the first references to this condition was made by Hutton P. W. 1956 in neurological disorders in Uganda. He reported 3 cases in 6 years (1949 - 1955). Outcome of these cases is not stated.

In 1957 Shaper and Shaper analysed medical admissions to Mulago Hospital, Kampala and reported 5 cases of subdural haematomas.

From the same hospital de Souza and Rankin in 1962 reported 23 cases seen over a 3 year period. 12 out of 23 of their patients died without a correct diagnosis having been made.

Billinghurst J. W. studied the pattern of adult neurological admissions to Mulago Hospital, Kampala from June 1966 to May 1968 and reported subdural haematomas in 16 males and one female. The mortality rate was 47% and carotid angiogram was positive in 7 out of 8 survivors and in 2 of those who died.

KENYA

Ojiambo H. P, in 1966 reported 5 cases from April 1965 to October 1965 in his analysis of neurological diseases at Kenyatta National Hospital.

Mngola E. N. and Alouch J. A. in a retrospective study of subdural haematomas admitted to the medical wards at Kenyatta National Hospital reported 38 cases in a 2 year study 71/72 found a mortality of 42% and drew attention to the high number of initial misdiagnosis.

Harris J. R. in neurological disorders in Kenya in 1972 reported 4 cases in 5 years in Kenys.

J. D. Stewart, D. Bedi and F. Mwongera in a 13 month period ending March 1974 reported 2 subacute (1 died) and 18 chronic cases of SDH of whom 4 died giving a mortality of 22%.

Sande in the neurological conference at Kenyatta National Hospital in 1977 reported 74 patients from 1974 - 1976 - 68

TANZANIA

Haddock D. R. W. analysed neurological disorders in Tanzania in 1965 but did not report any cases of subdural haematoma.

ZAMBIA

B. Umerah and J. Singarayer reported 39 chronic cases of SDH from October 1973 to 1976.

GHANA

Haddock D. R. W. reported 5 cases between 1968 and 1969 in Accra, Ghana in his analysis of neurological disorders in Ghana.

NIGERIA

Adeloye A and Odeku E. L. reported 7 cases of chronic subdural h@ematomas in a 27 month period, ending December 1973 in University College Hospital, Ibadan.

MATERIALS AND METHODS

The Kenyatta National Hospital has 800 beds and serves a population of 540,966 (Kenya population census 1969). In addition patients from all over the country may attend for medical attention.

In this retrospective study 51 patients with chronic SDH who presented to KNH over a three year period from January 1976 to December 1978 to both medical and surgical wards were studied.

The clinical details were obtained from the medical wards, admission records, neurosurgery operation records, postmortem records and carotid angiography records in the radiology department.

A retrospective study such as this has some obvious limitations in that some information is missing from the files but by and large most of the essential information was recorded.

BREAKDOWN OF ADMISSIONS

A large percentage of patients with chronic subdural haematoma present with bizzare clinical signs so that they are admitted to the medical units.

Only those who have a definite history of head injury or previous records of hospital admissions for a head injury get admitted to the surgical wards.

The chart below gives the breakdown of the admission of the patients with chronic subdural heomatoma to the medical and surgical wards in relation to the total admission.

	TOTAL ADMISSIONS	CHRONIC SUBDURAL HAEMATOMA	%
Medical Wards	22,200	37	0.17
Surgical Wards	10,856	14	0.13

TRIBAL DISTRIBUTION

Patients admitted to Kenyatta National Hospital were roughly in the proportion of the major tribes in Nairobi i.e. Kikuyu, Luhya and Kambas (Kenya population census 1969).

The breakdown of tribal distribution of patients admitted with SDH in the period January 1976 to December 1978 was as follows:-

Kikuyu	33	64.7%
Kamba	9	17.6%
Luhya	3	
Masai	2	
Ugandan	1	
Luo	1	
Teso	1	
Kalenjin	1	
Total	51	

Number of patients transferred to KNH from various centres were as follows:-

Machakos	4	
Mathari Psychiatric Hospital	3	
Kikuyu Nursing Home	3	
Gatundu	1	
Kangundo	1	
Kitui	1	
Aga Khan Hospital	1	
Thika Hospital	1	
Nakuru	1	
Nyeri General	1	
Total	17	= 33.3%

AGE AND SEX

There was no patient below the age of 23 years and half of the patients were in the 50 - 70 year group.

AGE IN YEARS	MALES	FEMALES	TOTAL
20 - 29	5	1	6
30 - 39	5		5
40 - 49	9	1	10
50 - 59	11	2	13
60 - 69	11	2	13
70+	4		4
Total	45	6	51

The average age of 45 male patients was 49.4 years with a range of 23 years to 75 years while the average age of the 6 female patients was 51.3 years with a range of 26 years to 67 years.

The male to female ratio was about 7.5:1.

ETIOLOGICAL FACTORS

HEAD INJURY:- A total of 20 patients gave a definite history of head injury while 15 others gave history of some form of trauma but not specifically head injury. In 16 patients there was no history of any form of trauma forthcoming.

CATEGORY	NO.	ALCOHOLISM	%
1. Definite history of head injury	20		
a. Loss of consciousness	10	6	60%
b. No. loss of consciousness	10	5	50%
2. History of other trauma	15	3	20%
3. No. history of trauma	16	5	31%
	45♂, 6♀	19	42%

Nineteen out of 45 males i.e. 42% had a history of heavy intake of alcohol.

Two of these patients had a spontaneous subdural haematomas. One female patient aged 26 years had it after child birth while a male patient aged 75 after a severe attack of asthma.

Details of the type of accident resulting in any form of trauma was elicited in the 35 patients who gave a history of trauma, not necessarily head trauma.

The average interval between trauma and admission in 35 patients was 102 days.

ETIOLOGICAL FACTORS IDENTIFIED	MALES	FEMALES	TOTAL
Brawl	11	0	11
Road traffic accident	7	3	10
Domestic accident	2	2	4
Attacked by thieves	4	0	4
Insignificent trauma	6	0	6

ALCOHOLISM:- A definite query was made about intake of alcohol. In this series none of the 6 females took alcohol while 19 out of the 45 men took alcohol regularly.

No history of epilepsy or liver disease was documented in any patient in this series and there was no patient on anticoagulant

On reviewing the old records of these patients it was discovered that a number of them had either attended the casualty or one of the clinicsor were admitted for history or complaints referable to the condition

Four of the patients had been seen at the casualty at the Kenyatta National Hospital for head injury and discharged after first aid.

Breakdown of patients who attended casualty:-

PATIENT	CASUALTY KNH	LOSS OF CONSCIOUSNESS AFTER INJURY	INTERVAL BETWEEN CASUALTY ATTENDANCE AND ADMISSION TO KNH
1	Yes	Yes	6 months
2	Yes	Yes	5 months
3	Yes	No	33 days
4	Yes	No	5 weeks

There were another 5 patients who were attending the clinics at the Kenyatta National Hospital, three of these had been attending the filter clinic and 2 of them the psychiatric clinic.

Six patients had history of previous admission to hospitals for the initial head injury.

Breakdown of inpatient admission:-

PATIENT	HOSPITAL WHERE ADMITTED	DURATION OF ADMISSION	LOSS OF CONSCIOUSNESS AFTER INJURY	DURATION	INTERVAL BETWEEN DISCHARGE AND ADMISSION TO KNH
1	A.O.W.	1 week	Yes	1 hour	3 months
2	R.W.	2 days	No	-	1 month
3	Kitui Hospital	2 months	No	-	2 months
4	Embu Hospital	1 month	Yes	10 days	6 months
5	Thompson Falls	3 days	Yes	12 hours	25 days
6	Nakuru Hospital	2 days	Yes	24 hours	7 weeks

Severity of trauma and its relationship to the age of the patient.

All the patients were grouped age wise in relation to the severity of the trauma to establish any relationship between the two.

AGE GROUP	TOTAL NUMBER	MAJOR TRAUMA	MINOR TRAUMA	NO. HISTORY OF TRAUMA
20 - 29	6	0	5	1
30 - 39	5	2	2	1
40 - 49	10	5	3	2
50 - 59	13	6	1	6
60 - 69	13	5	3	5
70 - 79	4	2	1	1
TOTAL	51	20	15	16
		39.4%	29.4%	31.4%

11 out of 26 patients in the age groups between 50 - 69 did not give any history of trauma.

CLINICAL PRESENTATION

The clinical features were diverse and for most of the cases non specific, thus the basis for diagnosis besides an adequate history is a high index of suspicion.

Symptoms of raised intracranial pressure	45
Hemiparesis	9
Incontinence of urine	8
Dysphasia	7
Paraparesis	7
Focal Seizures	2
Quadriparesis	2

Symptoms of raised intracranial pressure which consisted of headache and vomiting were found in 45 out of 51 patients.

Localizing signs in the form of hemiparesis dysphasia or focal seizures were seen in a small number of cases.

Incontinence of urine was the presenting complaint in 8 of the cases. A confused patient may present with incontinence.

Paraparesis in 7 patients is very significent because in subdural haematomas weakness often starts and is most marked in the lower limbs. It is also very interesting that objective weakness of the lower limbs was only detected in one patient with bilateral subdural haematoma.

CLINICAL FINDINGS:-

1. Level of consciousness.

LEVEL OF CONCIOUSNESS	NO.
Fully conscious	7
Confused or drowsy	26
Responding to pain	14
Comatose	4
TOTAL	51

A large majority of the cases presented with altered consciousness of the 26 cases who came in confused or drowsy, 19 had fluctuating levels of consciousness while 5 had progressive deterioration of level of conciousness during hospitalisation.

2. Other clinical findings:-

NEUROLOGICAL DEFICIT:-

1. No localizing sign	25
2. Focal Neurological deficit	25
3. Paraparesis	1
Pupillary inequality	16
Papilloedema	15
Bradycardia	10
Rise in blood pressure	4

Approximately half the patients had no localising signs while the other half had some localising sign in the form of either hemiparesis, dysphasia, upper motor neurone seventh nerve paresis, unilateral extensor plantar or local seizures.

One patient had paraparesis.

Evidence of raised intracranial pressure by demonstrable papilloedema was seen in 15 patients.

Pupillary inequality which also localised the lesion was seen in 16 patients.

Persistent bradycardia in 10 patients was an important diagnostic sign in some of the patients where there was no localisation in evidence of raised intracranial pressure.

A correlation between the neurological deficit and the presence of a clot unilaterally or bilaterally showed that 22 out of 42 unilateral and 3 out of 9 bilateral subdural haematomas showed no focal neurological deficit.

NEUROLOGICAL	TOTAL	UNILATERAL	BILATERAL
No Neurological deficit		22	3
Contralateral hemiparesis		18	-
Ipsilateral hemiparesis		2	-
Hemiparesis		-	5
Paraparesis		-	1
TOTAL	51	42	9

Two of the unilateral haematomas had ipsilateral hemiparesis while 5 out of the 9 bilateral haematomas had signs referable to only one side.

A patient with bilateral subdural haematoma had paraparesis.

DIAGNOSIS

In clinical examination the initial diagnosis of chronic subdural haematoma was made only in 13 out of 51 cases.

Chief diagnosis SDH	13
Differential diagnosis SDH	25
Chief diagnosis SOL	3
Differential diagnosis SOL (as this also includes 5 who also had a diffierential diagnosis of SDH ∴ 15 - 5	15
Total cases	51

The bizarre presenting symptoms and the clinical profile of some of these cases led to a number of other conditions being considered in the differerential diagnosis. The various conditions grouped together were:-

1. Neurological disorders	16
2. Metabolic causes	15
3. Vascular causes	10
4. Psychiatric causes	9
5. Infection	6
6. Musculo skeletal	4

The common neurological disorders being considered were pre-senile dementia or low pressure hydrocephalous besides some of these patients were considered to have had a stroke.

In a number of patients blood sugar or urea reports led to a delay in the diagnosis as the initial diagnosis of a metabolic inbalance was entertained.

Nine patients were diagnosed as psychiatricdisorders and some of them had psychiatric treatment before the correct diagnosis

INVESTIGATIONS

Plain x-rays of the skull were done in 46 patients, the remaing five were diagnosed at autopsy (one bilateral SDH, 4 were unilatera SDH) therefore though 38 of these cases were unilateral subdurals no plain x-ray localisation could be made as none of the x-rays showed a calicified pineal. Calicified pineal is believed to be rare in Africans (Murphy N. B. 1968, Daramola G. F. & Olown A. O. 1972, Mugondi S. G. &. Poltera A. A. 1976).

Carotid angiogram was performed in 43 out of 46 patients and was diagnostic in all of them.

There was no morbidity or mortality because of the carotid angiograms in this series.

Although echoencephalogram is available for us here it was not used because in our hands it has not proved to be very reliable.

Electro encephalogram was not done as a routine in any one of our patients.

The facilities for radioisotope scanning have become available to us and have been used in some patients subsequent to this series.

Glycosuria was recorded in 7 cases, while blood urea was estimated in 31 cases and haemoglobin level in 32 cases.

TREATMENT

Forty-six out of 51 patients underwent surgery for the evacuation of the chronic subdural haematoma. These hamatomas were routinely evacuated by placing the burr holes on the side of the clot in the frontal and parietal region. If during this procedure the clot was found to be encysted then a craniotomy flap was raised to excise the capsule. In these series 16 of these cases were encysted and 15 of these required a craniotomy, while one was diagnosed at postmortem.

This chart gives the breakup of the type of surgery performed:-

TYPE OF SURGERY	NO.
Burrhole evacuation	31
Craniotomy	15

Thirty cases either had a bilateral carotid angiogram or bilateral burr holes ensuring no bilateral lesion was missed.

Thirteen cases had unilateral carotid angiogram and a unilateral exploration.

RESULTS

As one of the aims of this study was to establish the delays in the hospital due to various reasons in either establishing the diagnosis, investigating the patients or taking them for surgery, each of these feature was individually studied.

DELAY IN DIAGNOSIS:- Twenty-six of these patients were diagnosed clinically on admission while the rest stayed for a varying period of time in the wards before the clinical diagnosis was entertained.

TIME INTERVAL	NO.
Within the first day	26
Within the first week	15
Within 2 weeks	3
Over 3 weeks	2
Total patients	46

Once the clinical diagnosis was made and a carotid angiogram asked for. only 10 cases got the carotid angiogram done the same day. Carotid angiogram was performed in 43 cases.

PERIOD AFTER HOSPITAL ADMISSION	NO.
Within 24 hrs.	10
24 - 48 hrs.	12
2-4 days	9
5 - 8 days	5
7 - 15 days	1
> 15 days	1
Total	43

A large majority of these patients i.e. 39 of these operated within 24 hours of angiographic confirmation and other three were operated without angiography because of deterioration of clinical status.

PERIOD BETWEEN FIRM DIAGNOSIS AND SURGERY	NO.
Same day	34 (3 *)
1 day	8
3 days	1
6 days	2
7 days	1
TOTAL	46

* Operated without angiogram.

The operative and autopsy findings regarding the unilateral or bilateral occurrance of the subdural haemotoma showed that 42 of these were unilateral and 9 were bilateral.

Unilateral SDH	42	82.3
Bilateral SDH	9	17.7

Total 51 (* 5)

In five of these patients one bilateral and four unilateral the diagnosis was established at autopy.

It was clearly established that the mortality or morbidity was not related to the delay at various stages but to the level of consciousness at the time of surgery. Of course some of the patients due to the delay did slip into a category below and thus made the prognosis worse.

Of the patients who were fully conscious none died.

LEVEL OF CONSCIOUSNESS	NO. OF PATIENTS	NO. OF DEATHS	%
Fully conscious	7	0	-
Confused or drowsy	26	6	23.1
Responding to pain	14	4	28.6
Comatose	4	2	50

THE RESULTS OF SURGERY ASSESSED AT THE TIME OF DISCHARGE of the patients from the hospital showed that 26 out of 39 were fully conscious on discharge with no neurological deficit. Of the 7 patients who died following surgery, 2 were deeply unconscious on admission and 4 more died of complications.

CONDITION ON DISCHARGE	NO.	% OVERALL MORLATITY
Conscious	26	
Confused	4	
Focal neurological deficit	9	
Died	12 (5 *)	23.5%

* Autopsy

SURGICALLY TREATED	SURGICALMORTALITY	% SURGICALMORTALITY
46	7	15.2%

The post-operative complications seen were:-

	NO.	DIED
Cerebral abscess	2	2
Grand mal epilepsy	2	1
Aspiration pneumonia	4	2

In terms of mortality due to chronic subdural haematoma in relation to mortality in general surgical and medical wards it was found that chronic subdural haematomas accounted for 0.46% of the mortality in the medical wards and 0.003% in the surgical wards.

JAN. '76 - DEC. '78	TOTAL WARD DEATHS	DEATHS DUE TO CHRONIC SDH	%
Medical cases	1,971	9	0.46
Surgical cases	1,168	3	0.003

The average stay of the patients was estimated in relation to the outcome and it was found that the patients who ultimately died had a longer stay in the hospital.

PT. DISCHARGED	PT. DIED
39	12
30.7 days	43.4 days

DISCUSSION

The first postmortem report of this condition is credited to Wepfer (1657). Virchow called it "Pachymeningitis haemorrhagica" (Virchow 1857). Trotter (Trotter 1914) first ascribed it to rupture of the superior cerebral vein as it traverses the subdural space.

Chronic subdural haematoma may be the result of trauma or it may arise spontaneously. Several predisposing factors are mentioned. The liability of alcoholics to develop chronic SDH is well known (Brain 1962) and its occurence has also been reported in epileptics (Feldman Pincus and McEntree1963). Other known causes are ruptured intracranial aneurysms or angioma, carcinomatosis or sarcomatosis of the dura (Russell and Cairns 1934 Brain 57, 32), blood dyscrasias and infections (Logue 1951) and anticoagulant therapy (Wiener and Nathanson 1962, Strang and Tovi 1962).

It may be difficult to distinguish even pathologically the spontaneous from the traumatic variety.

The traumatic variety of chronic subdural haematoma is due to injury to one of the cortical veins which cross the subdural space from the cortex to reach one or other of the venons sinuses.

Commonly the bleed occurs from the superior cerebral vein which enters the saggital sinus, the inferior cerebral veins which enters the sphenoparietal sinus, the tributaries which reach the lateral sinus from the posterior or inferior part of the temporal lobe or anomalous veins entering the dura from the convexity of the cerebral hemispheres (Voris 1941).

Three theories have been proposed to explain the mechanism by which the haematoma expands to produce cerebral compression.

The first is that repeated bleeding from the cerebral vein occurs into the subdural space whenever the cerebral venous pressure which is usually low or negative exceeds the intra cranial pressure as in early hours after haemorrhage due to coughing, sneezing or stooping (Putnam and Cushing 1925). Normally the veins are kept open only by their dural attachments and when a vein is injured there is little immediate tendency for blood to escape.

The second theory is that initially this haematoma has an osmotic pressure equal to that of the cerebrospinal fluid but when haemolysis occurs due to the protein content the osmotic pressure of the clot rises and cerebrospinal fluid is attracted into the clot swelling it (Gardner 1932 and Zollinger and Gross 1934)

The third theory is that the presence of blood induces a fibro blastic reaction in the inner layer of the dura. The stimulation is attributed to the fibrin in the blood.

This fibroblastic reaction envelopes the haematoma as neovascular sinusoidal channels develop in this membrane. There is recurrent haemorrhage into the old haematoma causing an increase in the size (Appelbaum et al 1974)

There is no reason to believe that these mechanisms are mutually exclusive.. If the mechanism of expansion of the haematoma is a result of repeated bleeding it might be expected that the older haematomas would be more solid. However as suggested earlier an osmotic mechansim alone may account for the expansion and consistency of the haematoma.

The concept of 'membrane' formation with chronic lesions is less clear. Membranes are sometimes well developed within a few days of injury and at other times this may be completely absent for at least 2 weeks. This was noted in the 16 encysted subdural haematomas found in this series.

The haematoma may be completely encapsulated by a pseudomembrane of connective tissue but in more recent cases the capsule may be defective on the surface in contact with the arachnoid and it is always thinner on this surface. It is adherent to the dura mater but can be stripped from it without difficulty. It does not become adherent to the arachnoid except in rare cases where the arachnoid has been torn by the injury. This may be explained by the peculiar layer of endothelial and exothelial cells (del Horlege 1933) which covers the arachnoid and which does not react to the presence of blood in the same way as the inner layer of dura mater on which there is no true endothelium (Leary 1939). The arachnoid is however usually stained yellow by blood pigment. Thin walled sinus like vessels are present in one or both layers of the capsule most usually that in contact with the dura mater and in time the capsule acquires great toughness from the deposition of collagen fibres. The contents of the Sac consists of a dark turbid fluid and a varying amount of apparently fresh red corpuscles.

From the figures quoted in epidemiology it appears that subdural haematoma is a commonproblem in this country and is being recognise more frequently than in the past.

Mongola E. N. and Alouch J. A. in a retrospective study of subdural haematomas admitted to the medical wards at Kenyatta National Hospital reported 38 cases in a 2 year study 71/72 and found a mortality of 42% and drew attention to the high number of initial misdiagnosis.

Sande in the neurological conference at Kenyatta National Hospital in 1977 reported 74 patients from 1974 - 1976. Sixty eight were males and six were females.

Seventeen patients died giving a mortality of 22.2%.

B. Umerah and J. Singarayer reported 39 chronic cases of SDH from October 1973 to 1976 from Zambia.

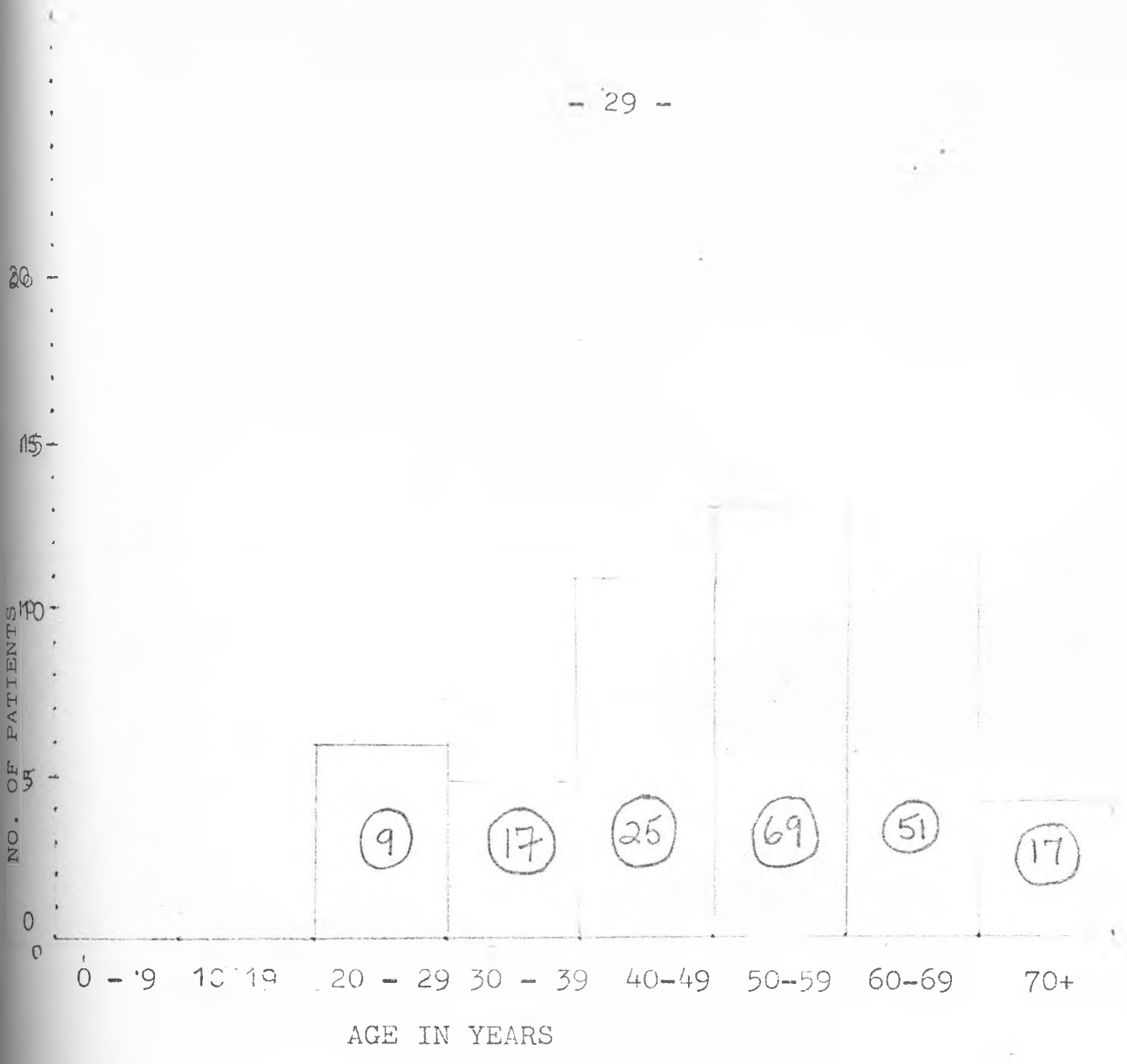
The incidence of chronic subdural haematoma increases with increasing age and is maximum in the fifth and sixth decade. In this series the finding of rising incidence with increasing age conforms to that of McKissock et al 1960.

McKissock et al in 1960 noted that in chronic SDH there was a peak in the 50 - 59 age group. In this study the age distribution was similar.

McKissock et al suggested that the decline in incidence after the age of 60 might be due to failure to diagnose the condition particularly in cases without a history of trauma in whom the alternative diagnosis of cardio vascular accident would be adequate.

In Kenya fewer chronic SDH in over 75 years age group may be related to fewer people in that group.

The likely explanation for rise in incidence with age is that the relatively atrophic brain of the elderly is subject to greater shifts within the skull from acceleration - deceleration forces (Porter J. M. 1970). This diminition in brain bulk also results in stretching of the bridging vein across the subdural space. An additional factor may be the relative inelasticity and fragility of these ageing cerebral veins.



McKissock's figures superimposed on figures obtained in this series.

The preponderence of males (88.2%) in this series is in accord with experience elsewhere and besides their numbers being less population wise, females are less prone to physical trauma as compared to males. The incidence of alcoholism is also less in the females and in this series none of the six females took alcohol while 42% of the males did.

History of trauma to the head was forthcoming in only 20 cases (just less than 40%) and out of these only 10 lost consciousness during the initial trauma. Out of the rest 29.4% gave history of some trauma and the other 30% gave no history of trauma whatsoever.

As a large majority of them present with no history of trauma most of the patients get admitted to the medical wards. In this series 37 patients (72.5%) were admitted to the medical wards and only 14(27.5%) were admitted in the surgical wards.

Besides the lack of history of head injury the presentation can be bizzare as is evident from one fact that a large majority of these patients were treated in the clinics or in the wards for other disorders. The common conditions mistaken for were various forms of neurological, psychiatric or metabolic disorders.

Any patient in the elderly age group with ahistory of trauma however trivial, alcoholism, headache, fluctuating level of consciousness with or without neurological deficit should be suspected of harbouring a chronic subdural haematoma.

Urinary incontinence, behaviour changes and subjective feeling of weakness of the lower limbs were other helpful clinical features in suspecting the diagnosis.

The commonest presenting complaint was headache and was present in 45 (88.2%) of the cases. The other symptoms suggesting raised intra cranial pressure were vomiting blurring of vision and haematemesis (in 3 cases). Incontinence of urine was a presenting complaint in 8 cases.

A very interesting presenting complaint in 7 patients was weakness in the lower limbs or inability to walk. On objective testing only one of these patients had paraparesis with a bilateral subdural haematoma. Of the other patients showing focal neurological deficit, eighteen with unilateral SDH had hemiparesis on the contralateral side, while five had contralateral hemiparesis with bilateral SDH and two had ipisilateal hemiparesis.

Ipisilateral hemiparesis is explained by the free edge of the tentorium on the opposite side producing a notch (Woltman - Kernohan notch) on the cerebral peduncle following an uncal herniation on the side of the clot and the resultant shift of the brain stem.

Convulsions are uncommon with chronic subdural haematomas and in this series only two patients had convulsions as a presenting feature. One patient who developed post operative infection and a subsequent brain abscess had convulsions.

In spite of the long history and delay in diagnosis by the time the patients were taken for surgery only four were deeply unconscions while another fourteen were responding to pain only. A large majority of them (26) were in the varying stages from confusion to semi-consciousness. Fluctuating level of con sciousness have been attributed to the changes in the size of the haematoma (Putnam & Cushing 1925, Gardner 1932, Zollinger & Gross 1934, Appelbaum et al 1974)

Seven patients were fully conscions. Though 38 cases of unilateral subdural haematomas had skull x-rays, this did not prove to have any localising value as no calcified pineal gland was seen.

N.B. Murphy in 1968 reviewed 100 consecutive cases in Uganda and noted that calcified pineal was an uncommon finding.

Adeloye and Felson in a study of Black Americans in the United States showed a 9.8% calcification rate of pineal glands.

S. G. Mugondi & A. A. Poltera in 1976 made a radiological study of 200 cases of Ugandan Africans and found that 43 per cent of all pineal glands after the age of 10 years were likely to be detected on an ordinary skull x-ray. Females showed more calcification than males.

Daramola G. F. & Olown A. in 1972 - found only a 5.04% pineal calcification rate in Nigeria.

Investigations on urine and blood estimations may sometimes mislead the clinician. Glycosuria may be an incidental finding in raised intracranial pressure and unless substantiated by raised blood sugar levels should be assessed carefully. Similarly raised urea in the elderly patients whose intake may be inadequate due to the neurological status may be misleading and this accounts for one of the cases diagnosed at autopsy.

Lumbar puncture was performed in 10 cases and was unyielding but the pressure has been reported to be low in cases with chronic SDH (Walker et al 1968). Lumbar puncture can positively be dangerous and lead to tentorial herniation and death and is to be avoided even if the diagnosis of chronic subdural haematoma is suspected.

Echoencephalography and electroencephalography although available to us were not used as routine investigative procedures. Radioisotope brain scan using technitium has been found useful in cases of SDH (Gilday et al 1973, Hurwitz S. R. et al 1974) was not available for us during the period of study but has been available since then.

The most helpful diagnostic tool the computerised axial tomography has been found to be extremely accurate (Ambrose J. 1973, Paxton R. et al 1974) in the diagnosis of subdural haematomas but facilities for its use are limited to very few centres in the developing countries.

In recent years conservative management of the chronic SDH has been advocated (Bender M. B. 1960, Suzuki 1970, Gjeris F. et al 1974, Bender M.B. et al. 1974) with use of bed rest corticosteroids, daily mannitol combination. In most of the series the results are gratifying although a number of patients had to undergo surgery if the medical treatment did not succeed while in one series (Gjeris 1974) the trial with mannitol had to be given up halfway because of the failures. It would be fair to sum up and say that the treatment of choice for management of chronic SDH is still surgical.

In spite of the delays at various stages of hospital stay it was clearly established that the results of surgery depend really on the level of consciousness at the time of surgery. Thus no deaths occurred in patients who were fully conscious while two of the four who were deeply unconscious died. The overall operative mortality of this series was 15.2%. This shows marked improvement since the previous reports of mortality of 42% in the series by Mongola in 1971/72, Stewart J. D. 1974 22%.

Sande 1974/76 - 22.2%

The improvement in mortality may be partly due to better neuro-surgical cover and also because of the awareness about the encysted variety of chronic SDH. A large number of patients who used to die following burr hole exploration for encysted variety could have been saved if a frontal craniotomy with excision of the cyst walls was done as is done routinely now. The mortality figures here compare favourably with figures from elsewhere (Echlin et al 1956) - 20.4% and McKissock 1960 - 6%.

The results could be better if the referrals are made earlier and there is greater awareness in the peripheral hospitals as 33.3% of patients came from the peripheral hospitals. Inspite of all the delays a fair percentage of patients (56.5%) was discharged fully conscious with no neurological deficit.

CONCLUSION

Chronic subdural haematoma is a common problem in this country and the relative frequancy could be attributed to alcohol, assault and acceleration. (Ravel S. K. 1974)

Patients in the elderly age groups presenting with headache, behaviour changes, incontinence and even indefinite localising signs should be suspected to be harbouring a chronic subdural haematoma.

Adequate investigative facility and correct operative measures can reduce the mortality considerably as is shown by this series. The mortality was greatly related to the level of consciousness of the patient at the time of surgery.

Greater awareness at the peripheral hospitals and earlier referral would not only reduce the mortality figures but also improve the quality of survival.

ILLUSTRATIVE EXAMPLES

Patients in whom diagnosis of SDH was made at postmortem:-

CASE 1

A 75 year old Kikuyu male was admitted to a private hospital with a history of sudden left sided weakness a day earlier. After a week he was transferred to KNH for financial reasons and was admitted to a medical ward.

On admission the patient was confused and disorientated in time and place. He had left hemiparesis, his blood pressure was 150/100 mm Hg. and his pulse was 88 per min regularly irregular. His other systems were within normal limits. The patient's blood pressure the next day was 120/90 mm Hg. with a pulseof 88 per minute.

In view of his age a diagnosis of cerebrovascular accident and arteriosclerosis was entertained.

A week later the patient had become confused and was treated for E. Coli urinary tract infection. His neurological status had deteriorated further. Patient was not talking any more and had developed bilateral hyper reflexia.

Patient died 18 days later and the post mortem revealed bronchopneumonia and a right chronic subdural haematoma.

Patient's age and hemiparesis of sudden onset contributed to a diagnosis of cerebro vascular accident being made.

The patient's deteriorating neurological status was attributed to senile changes (Bedford 1957, Stutteville and Welch 1958, Perlmutter 1961, Spencer 1964)

CASE 2

A 60 year old Luo patient was admitted to a medical ward with a history of headache of three days duration and loss of appetite for 1 day. Patient was not orientated in space time and person and had marked hyper reflexia of all limbs. The other systems were normal but the patient had a temperature of 37.8°. A diagnosis of

Eight days later patient went into a coma. He had exhibited fluctuating level of conscionsness in the intervening 8 days but no importance was attached to this. A history of heavy alcohol intake was also obtained from relatives.

On the 8th day he was deeply unconscious and the pupils were dilated and fixed. There was no neck stiffness and fundi were normal. Diagnosis of a chronic subdural haematoma was considered but it was felt that a diagnosis of hepatic encephalopathy in view of patients heavy alcoholic intake was more appropriate. A dextrose drip was set up and repeat lumbar puncture advised.

The patient died 5 hours later. Postmortem diagnosis revealed a left subdural haematoma.

Here because of absence of localising signs and lack of history of obvious trauma, fluctuating levels of consciousness with hyper reflexia was ignored. A history of alcoholism should be considered synonymous with trauma.

CASE 3

A 60 year old male Kikuyu was transferred from a peripheral nursing home after 3 days where he had presented with an eleven month history of headache. His blood pressure in this nursing home was 160/100 mm Hg. The patients condition deteriorated and the patient became unconscious. A day later he was transferred to KNH.

Patient was seen in the casualty and was found to be unconscious with stetorous breathing. Both the pupils were dilated left more than right, the blood pressure was 90/60 mm Hg., pulse was 72 per min. The right planter was extensor and the left was equivocal. The chest was clear and there was no oedema

A diagnosis of chronic subdural haematoma was suggested but due to glycosuriaa diagnosis of diabetes mellitus was made in spite of a normal blood sugar of 90 mg%. No further steps were taken that evening and patient died the next day.

Post mortem revealed a chronic encysted left subdural haematoma.

A dilated fixed pupil with depressed neurological status and extensor planter response should raise the possibility of an expanding intra cranial lesion. Glycosuriamay be found in raised intra cranial pressure.

CASE 4

A 65 year old male Kikuyu was transferred from a peripheral nursing home with a 10 year history of asthma. He had been in the nursing home for 9 days prior to admission to Kenyatta National Hospital. He was treated there for left lobar pneumonia and was improving 7 days later when he suddenly started vomiting.

He was seen in casualty as a sick looking patient in a semi comatose state. Chest x-ray showed apicalscarring and patchy infiltrate with pleural thickening at the apices and patient had patchy bronchial breathing. He had atrial fibrillations and pitting oedema of both limbs. There were no chest signs to suggest asthma. The consultant commented that the patient coughed and retched and that vomiting was an inconsistent aberrant feature in this case.

He was given digoxin for his atrial fibrillation and started ongentamycin and penicillin.

He appeared to be improving and was transferred to a medical ward where he was found to be drowsy with no localizing neurological signs. Patient had improved sufficiently 6 days later and was walking around the ward.

Later in the ward patient developed cheynes stokes respiration and became dyspnoeic. He was treated with aminophylline and put on antifailure regime. Sputum was cultured for T. B. Patient died 16 days later.

Postmortem diagnosis revealed congestive cardiac failure due to hypertension, bronchopneumonia and subdural haematoma.

Asthmatics because of repeated expiratory problems are liable to develop SDH. Vomiting as a sign of raised intracranial pressure was not considered. Cheynes stokes respiration can be an indication of diffuse encephalopathy. In the absence of localising signs, fluctuating level of consciousness was not evaluated in the correct way. This case was very difficult to diagnose and inspite of prompt care all the way diagnosis was missed.

CASE 5

A 33 year old Kalenjin patient was admitted to Mathari with a history of abnormal behaviour of some months duration. He was treated as a case of catatonic schizophrenia with stelazine and later electro convulsive therapy. Following E.C.T. the patient had difficulty in holding objects and his level of consciousness deteriorated. He was transferred to KNH after 3 months at Mathari.

Patient was found to be semiconscious with pupils dilated equally and reacting sluggishly to light. There was a mild 7th nerve palsy on the left side. Patient had generalised hyper reflexia of all limbs with upgoing planters. There was reduced air entry in both the lungs bases but cardio-vascular system was within normal limits.

Carotid angiogram was booked 2 days after admission and done in 9 days time and was reported provisionally as normal.

Meanwhile the patient was started on anti T.B. therapy on the basis of lymphocytes and increased protein in the CSF. There was considerable improvement.

Nine weeks later the patient became confused and refused to feed. He died 5 days later.

Postmortem revealed bilateral organised SDH, bronchopneumonia and haemorrhage of 'R' adrenal.

ACKNOWLEDGEMENTS

I wish to express my utmost thanks to Dr. J. Dar M.B.B.S., M.S. (Surgery) M.Ch., (Neurosurgery) for his sincere advice and help.

My thanks to Professor Kungu of the Pathology Department at Kenyatta National Hospital and Mr. Mbogo, Superintendent in charge of the mortuary at Kenyatta National Hospital for the help in enabling me to trace the postmortem records.

Mrs. Ndegewa and Mr. Kirika of the records section at Kenyatta National Hospital were extremely helpful to me throughout my study in allowing me access to the files at all times. My utmost thanks to them.

Mr. Muchemi and Mr. Joshua in the statistics section of the records office compiled the figures on admissions and deaths on medical and surgical wards for the period under study and I would like to express my thanks to them.

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