A REVIEW OF THE PATTERN OF PRESENTATION AND DIAGNOSIS OF TUBERCULOSIS OF THE ABDOMEN AT KENYATTA NATIONAL HOSPITAL.

A DISSERTATION SUBMITTED IN PART FULFILMENT FOR THE DEGREE OF MASTER OF MEDICINE IN SURGERY, UNIVERSITY OF NAIROBI.

BY
DR. MUGALO EDWARD LUMADEDE
MB ChB (NRB).

2000

DECLARATION.

This thesis is my original work and has not been presented for a degree in any other university.

Signed..... Date 29.11.2000

DR. MUGALO EDWARD LUMADEDE
M.B.ChB (NAIROBI)

This thesis has been submitted for examination with my approval as university supervisor.

Signed..... Date 29.11.2000

PROF. J.A. ADWOK, MBBS, M.MED (SURG), FICS, FRCS
ASSOCIATE PROFESSOR OF SURGERY
DEPARTMENT OF SURGERY
UNIVERSITY OF NAIROBI.

ACKNOWLEDGMENTS

My special thanks go to my supervisor Prof. J.A. Adwok of Department of Surgery, University of Nairobi for his guidance and assistance during the entire period of the study and preparation of this manuscript. His patience in reading and providing criticism has been greatly appreciated.

I also wish to thank all the lecturers whose wise advice was sought for during the time of the study.

My thanks go to Mrs. Karen Muli of the hospital's Records Department for assisting in retrieval of patient records, Wangeci of Department of Pathology for assisting in tracing histology reports that were a very crucial part of the study, and Janet Musia for expert typing and computer work of compiling the data and putting this work in its current form.

Last but not least to my parents Samuel Mugalo and Doricah Mugalo and my sister Mary Jimase who were of so much assistance without which my education would not have been successful.

DEDICATION.

This work is dedicated to my beloved wife Hellen Lumadede and my two children, Caren Buyanzi and Dennis Mulindi for their sacrifice for many days on end by allowing me to be away from them and for their inspiration, encouragement and consistently committing me to prayer during the trying period of my post graduate course.

CONTENTS.

ABBREVIATIONS.

AFB	-	Acid Fast BAcili
ZN staining	-	Ziel Nelson Staining
HIV	-	Human Immunodeficiency Virus
CT-scan	-	Computerized Tomography Scan
AIDS	-	Acquired Immunodeficiency Syndrome
TB	-	Tuberculosis
ESR	-	Erythrocyte sedimentation rate
FNAC	-	Fine Needle Aspriate Cytology
BCG	-	Bacille Calmette - Guerin

LIST TABLES AND FIGURES.

PAGE

SUMMARY

The purpose of this study was to carry out a retrospective study of the pattern of presentation and methods of investigations in patients with abdominal tuberculosis at Kenyatta National Hospital. The period of study was between 1st January 1990 upto 31st December 1999.

A total of 69 patients with abdominal tuberculosis presented at a mean age of 26 years with majority (71.4%) aged between 21-41 years. Males formed 44.8% and females 55.2% of the patients. Approximately 80.9% of the patients were from low social economic status either unemployed or low income jobs or small scale business.

The most common complaint was abdominal swelling (78.3%) followed by abdominal pain (60.9%), weight loss (27.5%) and fever 24.6%. Clinical ascites (59.3%) was the most common physical finding followed by abdominal distension (42.4%), abdominal mass (32.2%) and abdominal tenderness in 16.9%. Majority of patients presented with chronic symptoms (84.1%) and only 15.9% presented with acute abdominal symptoms.

The most important investigation to confirm the diagnosis was histology showing granulomatous lesion with epithelial and Langhan's giant cells and caseation in 89.5% o patients, while 9.0% had AFBs seen on ZN staining and 1.5% had a positive culture of AFBs. The tissue biopsies were obtained by blind percutaneous needle biopsy in 33.3%

and in 66.7% by laparotomy.

The mean haemoglobin was low (10.6gm/dl) while white blood cell count and lymphocyte count were normal. The mean red blood cell count (3.9×10^{12}/l) was low. The mean erythroycte sedimentation rate was high (46.2mm/hr). The mean total serum protein was normal (70.4gm/l) but the mean serum albumin levels were low (25.3gm/l). Among those patients tested for HIV, 78.6% were negative and only 21.4% were positive. Tuberculin test was positive in 77.8% and 22.2% had either negative result or not reported.

Plain abdominal x-ray revealed dilated loops of intestines in 34% of patients, air fluid levels in 17.4%, ascites in 17.4% and calcifications in 4.3% while 26.9% revealed no significant findings. The most common finding on abdominal ultrasound was ascites in 27 (55.1%) patients, enlarged lymph nodes in 5(10.2%), hypertrophic intestinal lesions in 8 (16%) and cystic mass in 7 (14.3%). Majority of the patients who had CT-scan done (64.3%) showed no significant findings while the most common finding was adherent bowel loops (14.4%).

The most important laparotomy finding was the presence of widespread tubercular nodules in 25 (62.5%) patients and widespread adhesion in 22 (55%) patients, thickened peritoneum and omentum in 17 (42.5%) and ascites in14 (35%) patients in that order.

INTRODUCTION.

Abdominal tuberculosis denotes involvement of the gastrointestinal tract, peritoneum, lymph nodes and solid viscera e.g. liver, spleen and pancreas by the disease. Worldwide, the incidence of tuberculosis is increasing especially with the advent of HIV. While previously rare in western countries, the incidence is now rising because of immigrants and AIDS patients (1,2).

Tuberculosis of the abdomen is the next common site of tuberculosis after pulmonary tuberculosis and tuberculous adenitis (3). Tuberculosis of the abdomen is difficult to diagnose even in endemic areas. Knowledge of the pattern of presentation is therefore important to the clinician giving a high index of suspicion and hence minimal time taken to carry out the relevant investigations. The patient can therefore be started on appropriate treatment in good time.

The three known pathological entities of abdominal tuberculosis are tuberculous peritonitis, gastrointestinal tuberculosis and the glandular type which involved abdominal lymphnodes. The first two are common while the third one is rare although it is now more common in tropical countries (4,5,6).

Each of these pathological entities can either present in acute or chronic form (7). Primary gastrointestinal tuberculosis is rare because of pasteurization of milk and

eradication of bovine tuberculosis. However, primary tuberculosis still occurs in countries where there is pasteurization of milk. The mode of infection is either by ingestion of infected sputum or as a sequel to miliary tuberculosis. The primary lesion is usually in the mesenteric lymp nodes and spreads from there. In some cases the disease is blood borne and spread, through the lymph nodes into the peritoneum. The lymph nodes enlarge and get matted together. If they rupture, infection spreads into the peritoneal cavity and effusion (ascites) occurs. The adhesion of nodes to bowel may cause obstruction. Fistulae may occur between the bowel and bladder or bowel and abdominal wall or between two adjacent bowel loops (8) .

The secondary form occurs in patients with pulmonary tuberculosis who swallow infected sputum. The mycobacteria in the sputum infects the wall of the intestines (usually the ileum) and cause ulceration. Fistulae may occur or infection may spread into the abdominal cavity and cause ascites (9).

LITERATURE REVIEW.

Historical.

Hippocrates in the 5th century recorded that "diarrhoea in a person with phthisis was a mortal symptom". This is probably the first recorded connection between gastrointestinal symptoms and tuberculosis (10)

The earliest probable references to intestinal tuberculosis was made in 1643 when the autopsy of Louis XIII showed ulcerative intestinal lesions associated with a large pulmonary cavity (11). Microscopic tubercles were described by John Hunter "..... in the liver, the spleen, the uterus, the coats of the intestines, the peritoneum....." (12). He postulated that these tubercles probably arose from the lungs. This was followed by the description of a tubercle causing an ulcer in the mucous membranes of the intestines resulting in disruption of the wall and leading to intestinal phthisis.

Incidence.

Abdominal tuberculosis is an uncommon condition in western societies. However, Europe and America have noted an increase in this condition in immigrants and AIDS patients (1,2). In the Netherlands, immigrants (above all Morocans, Somalians and Turks) are a risk group for tuberculosis. In 1994 the incidence in immigrants was 124 per 100,000 and in Dutch people 5.8 per 100,000. In about one-third (1/3) of these patients tuberculosis has an extrapulmonary location e.g. abdominally (3).

In the western world there are two distinct patient population with abdominal tuberculosis. Immigrants and those infected with the HIV virus. In one study, more than 40% of patients with extrapulmonary tuberculosis were HIV positive (13) and in another, more than 65% of patients with abdominal TB were HIV positive (14). In tropical and developing countries the disease is still a scourge (15). In Nigeria, 10% of post mortem examinations in patients over the age of 10 year and 24% of adults presenting with ascites showed evidence of abdominal tuberculosis (16,17). A high incidence has been reported in India, Iran and the black population of South Africa (18). The association of abdominal tuberculosis with AIDS in African countries has not yet been clarified. Other causes of suppression of the host defenses such as malnutrition, weight loss, alcoholism, diabetes, chronic renal failure and some stressfull conditions cause abdominal tuberculosis by reactivation of a dormant focus (18,19).

Abdominal tuberculosis can occur at any age, but it is predominantly a disease of young adults; two thirds (2/3) of the patients are 21-40 years old (20) and the mean age of the patients is 30 years (21,22). Although some reports mention a higher incidence in females (23) it seems that the disease affects both sexes equally (20).

Pathology.

The characteristic granulomatous lesion containing collections of epitheloid and Langhan's giant cells occur in pathological mycobacteria which cause chronic disease, but caseation occurs only in tuberculosis.

Two common pathological entities are tuberculous peritonitis and gastrointestinal tuberculosis, each of which can be acute or chronic. The patient may have one or both entities. The glandular type of abdominal tuberculosis (tuberculous lymphadenitis) constitute a third pathological type which is now more common in tropical countries (4). However solid viscera e.g. liver, spleen and pancreas may also be involved.

The common sites of involvement in the gastrointestinal tract are the ileum (11) and the ileocaecal region (5) followed by the colon and the jejunum. Three types of intestinal lesions are commonly seen - ulcerative, stricturous lesions and hypertrophic circatricial healing of the ulcerative lesions resulting in strictures. Occlussive arterial changes may produce ischaemia and contribute to development of strictures (6). These morphological types can co-exist e.g ulcero-constrictive and ulcerohypertrophic lesions. Small intestinal lesions are ulcero-hypertrophic. Colonic lesions are also ulcero-hypertrophic. Colonic lesions are usually associated with ileocecal or ileal involvement but isolated segmental colonic tuberculosis does also occur (24,25).

Some patients have involvement of peritoneum and lymph nodes alone without involvement of the gastrointestinal tract. Peritoneal involvement may be of either an ascitic or adhesive (plastic) type. The lymphnodes in the small bowel mesentry and the retroperitoneum are commonly involved and these may caseate and calcify (18).

Disseminated abdominal tuberculosis involving the gastrointestinal tract, peritoneum, lymphnodes and solid viscera has also been described. Chan et al (26) reported disseminated involvement of the abdomen in 21 out of 60 patients with large bowel tuberculosis, while most of the 96 patients with tuberculous hepatitis reported by Essap et al (27) had disseminated disease. Multiple lesions are also common. Bhansali (20) reported that small intestinal strictures were multiple in 71 out of 119 patients and as many as 12 (28), 6 (29) and 19 (30) strictures have been found in a patient.

Tuberculous appendicitis, not a common problem, can present acutely, indistinguishable from acute appendicitis or it might be a histological surprise in an otherwise normal looking appendix or as a chronic right iliac fossa disease (31). The liver is frequently affected in association with milliary tuberculosis. In 41 patients with hepatic tuberculosis in South Africa, the liver was found to vary in size and consistency (32). Hepatic involvement tends to be diffuse, macronodular forms are rare (33).

Pancreatic tuberculosis can mimic pancreatic tumours. Patients present with abdominal pain, loss of weight, anorexia and/or occassionally with obstructive jaundice due to a mass at the head of the pancreas (34). Tuberculous pancreatic abscess has been reported in patients with AIDS (35).

In acute tuberculous peritonitis, there is ascites, scattered tubercles with inflammatory reaction around them, and a thickened omentum forming a transverse band across the

abdomen. There is profuse, clear, straw coloured effusion, sometimes bloody or even chylous (36). The tuberclous nodules may simulate fat necrosis or peritoneal carcinomatosis.

Clinical presentation.

The history usually goes back some 3 or 4 years. The mean duration of stay of immigrants in the UK before diagnosis of gastrointestinal tuberculosis is 4 years (range between 3 months to 15 years) (37). Presentation may range from frank malabsorption (20) to biochemical evidence of malabsorption (38).

Various factors may ultimately modify the clinical picture. These include the location of the lesion, the mode of presentation (acute or chronic) and the extent of the disease, i.e. whether it is entirely abdominal, or extraabdominal lesions are present and whether there is peritoneal involvement. It is usually a complex clinical situation of abdominal pain, weight loss, fever and sweats, diarrhoea and vomiting (7). Abdominal tuberculosis should be considered in any patient with unexplained and chronic abdominal symptoms (39) and should be thought of whenever a diagnosis of Crohn's disease or gastrointestinal malignancy is being intertained (40).

In a review of 24 cases of abdominal tuberculosis Underwood et al reported that the most common primary complaint was abdominal pain (41), which is usually non-specific and long-standing, often mimicking other conditions such as Crohn's disease or gynacological

malignancy. Anorexia, weight loss, abdominal distension, fever and night sweats are common. On examination, 30% of patients have a mass in the right iliac fossa. Ascites is present in 50% of the patients, while others have a dry peritonitis indicating a plastic form of the disease. The lump in patients with abdominal tuberculosis is firm, mobile and only slightly tender.

Ano-rectal tuberculosis presents as stricture (42), fistula-in-ono (43), or fissure-in-ano (44). Tuberculous fistulae are usually multiple, and as many as 12 out of 15 multiple fistulae. It can also present as peptic ulcer with gastric out let obstruction (9) or perforation and may mimic carcinoma.

The clinical manifestations are classified into acute and chronic forms. In gastrointestinal tuberculosis, nearly 30% of patients present with an acute abdomen (7)

Acute form:

It is generally rare, it includes tuberculous acute appendicitis which clinically is indistinguishable from acute appendicitis. Acute or subacute intestinal obstruction results from tuberculous strictures of the small bowel (45). Acute tuberculous peritonitis is rare, but when it occurs, it is due to perforation (46).

Chronic form.

Fifty percent (50%) of these patients present with an abdominal mass (47), usually due to hyperplastic caecum and/or tabes mesenterica. Patients present with abdominal pain, distension and ascites (48). Clinical examination reveals ascites in more than 95% of patients (49).

The glued bowel and omentum may give rise to a 'doughy abdomen' a sign which is rarely detected (50). In the majority of patients, general examination reveals an ill, undernourished and often anaemic patients. The differential diagnosis includes carcinoma of the caecum, Crohn's disease and an appendicular mass (29).

Abdominal tuberculosis may present with pyrexia of unknown origin and the diagnosis may be established only after an exploratory laparotomy (51). Tuberculous peritonitis is more common in females in the 3rd and 4th decades of life. The most common presenting symptoms, in order of frequency, are abdominal swelling, fever, night sweats, anorexia, loss of weight and abdominal pain and 95% and above on examination reveals ascites (48).

Tuberculosis is predominantly a disease of lower socio-economic groups and this is reflected by its demographic distribution. Studies in western suburbs of Melbourne showed highest incidence of tuberculosis in victoria with over 120 new cases a year per 100,000 of the population (52).

Investigations.

Investigations short of tissue diagnosis are often non-specific (53). Laparoscopy is the diagnostic procedure of choice and can be performed under local anaesthesia if necessary (54). The classic appearance of abdominal tuberculosis is of ascites with tuberculous milliary seeds adhering to the peritoneum. Laparoscopy has a diagnositc accuracy of 75%. Alternatively, there may be dense white adhesions or a deep abdominal mass from involved mesenteric lymph nodes (55). The Hasson technique for establishing pneumoperitoenum is recommended as it is associated with fewer complications, especially where adhesions may be present. Satisfactory peritoneal biopsies can be obtained using sigmoidoscopy biopsy forceps through a 5-mm port site. Histological features are similar to those of T.B at other sites with caseating granulomata containing Langhan's giant cells, but demonstration of acid fast bacilli or culture of mycobacterium is necessary to confirm diagnosis.

The haemoglobin is low as well as serum albumin. There is leucocytosis mainly lymphocytosis (56,57). Seventy percent (70%) of the patients have a high erythrocyte sedimentation rate (22).

An exudative ascites with a protein level of > 30g/dl and a white cell count of > 1000/mm^3 (predominantly lymphocytes) support the diagnosis (58). In a study of 3 cases,

S. MacLaughlin et al found serum protein levels within the normal range but a predominantly gammaglobulinaemia (59).

Serological tests such as soluble antigen flourescent antibody and enzyme-linked immunosorbent assay are prone to give both false-negative (due to immune response) results and can only suggest a probable diagnosis of tuberculosis. This gives a diagnostic accuracy of 84% (60). Polymerase chain reaction (PCR) of aspirated material is helpful for early confirmation of tuberculosis and in discriminating M. tuberculosis complex from Mycobacteria avium in patients with AIDS (61).

ZN-staining of ascitic fluid is only positive in 3% of cases while, culture of the same is positive in 20% (62,63,64). The yield can be increased by examining the sediment of a large quality of the fluid (up to 1 litre) (48,65).

Another test for early diagnosis of tuberculous peritonitis is the determination of adenosine deaminase activity in the peritoneal fluid. In a study comparing the enzyme activity in various forms of ascitic fluid, it was found to be significantly higher in tuberculous ascites (66).

For radiologic diagnosis of abdominal tuberculosis CT-scan and ultrasound are advocated although they are non-specific. They may show ascites and lymphadenopathy with bowel wall thickening (66). Recently, echography and an echo-guided biopsy of mesenterium,

omentum or peritoneum were shown to be very effective to diagnose tuberculous peritonitis. Localised ascites with thin septa and thickening of mesenterium, omentum and peritoneum are characteritistic signs of tuberculous peritonitis. When these signs are present, a less invasive echo-guided puncture can replace a diagnostic laparascopy or laparotomy (13).

In Prakash's series of 300 patients (23), no patient had active pulmonary tuberculosis but 39% had evidence of healed tuberculosis on chest x-ray. Chest x-ray is more likely to be positive for tuberculosis in patients with ulcerative intestinal and ascitic peritoneal types and those with acute complications. A chest radiography may show concomitant pulmonary tuberculosis in 7-72% of patients (28,30).

Abdominal x-ray films may show dilated intestinal loops and air fluid levels even in the absence of clinical intestinal obstruction (13), calcified lymphnodes , enteroliths and ascites. The radiological findings in small bowel barium enema are mucosal irregularity and rapid emptying (ulcerative), floculation and fragmentation (malabsorption), dilated loops and stricture, displaced loops due to, (enlarged lymph nodes) and adherent fixed loops (adhesive peritoneal disease).

Barium contrast studies of the bowel show non-specific abnormalities in more than 50% of patients (57). Double contrast barium enema in ileo caecal tuberculosis shows a shortened ascending colon, deformed (irregular and shortened) caecum, deformed and

incompetent ileo caecal valve, dilated ileum and distorted ileocaecal junction with increased (obtuse) ileocaecal angle. Barium studies are sensitive for ileocaecal and colonic lesion (54) but small bowel strictures may be missed and extra intestinal lesion (peritoneal and lymph nodes) may be misinterpreted as intestinal strictures.

Tuberculin test (mantoux or heaf) is positive in upto 15%-100% of patients (28,58). Endoscopic appearance in tuberculosis include hyperaemic nodular friable, irregular ulcers with sharply defined margins and undermined edges, pseudopolyps and cobble stoning and may mimic Crohn's disease and malignancy (69). FNAC may increase the yield where endoscopic biopsy fails to reveal granulomas (70,71).

Treatment

Medical treatment.

Therapeutic trial although advocated by some authors, is not generally recommended because it may delay the diagnosis and treatment of more serious diseases such as malignancy, lymphoma and Crohn's disease which can mimic tuberculosis clinically and even radiologically. It can also alter the histologic picture in tuberculosis so that the diagnosis cannot be confirmed or refuted at a latter date (72).

All patients with abdominal tuberculosis should receive full course of antitubercular therapy. Conventional regimens include antitubercular therapy for 12 to 18 months (73). Short course regimens including ethambutol, rifampicin and isoniazid for 6 months or

pyrazinamide, ethambutol, rifampicin and isoniazid for 4 months are effective for abdominal tuberculosis (74). It is important to administer a correct and complete course, as inadequate drugs, dose or duration is the most important cause of emergence of anti-drug resistant tuberculosis. Management depends on the site and type of presentation.

Table 1: Management of abdominal tuberculosis

Site	Type	Suggested treatment
Any site	• Acute abdomen	• Emergency surgery
Intestinal	• Ulcerative • Stricturous • Hypertrophic	• ATT • Stricture plasty resection • Resection
Peritoneal	• Ascitic, adhesive	• ATT + ?steroids
Lymph nodes	• Ascitic, adhesive	• ATT

ATT - antitubercular therapy.

Surgical treatment.

Before adequate anti-tubercular drugs became available, various reports recommended the use of radical procedures such as right hemicolectomy, in an attempt to eradicate the disease. The recommended surgical procedures for tuberculosis today are conservative. Tuberculosis is a systemic disease and cannot be eradicated by surgery alone (18).

The indications for surgical treatment are: an undiagnosed abdominal mass, acute abdomen (e.g. acute appendicitis), an acute or more commonly, sub-acute intestinal

obstruction. When the diagnosis of appendicitis is made, appendicectomy should be performed and the appendix examined histologically (31).

The patient with an intestinal stricture, resection and anastomosis used to be the standard therapy. There was a belief that medical treatment increased fibrosis, thus causing obstruction and perforation (75). However, recent reports suggests that most patients with tuberculous strictures respond well to medical treatment and surgery should be resorted to only if therapy fails (8). Stricture - plasty for cases with multiple strictures was introduced as a better technique than multiple stricture resection and multiple enteroanastomoses because it does not sacrifice any part of the small bowel and it avoids the occurrence of a `blind loop' syndrome (76).

In case of bowel perforation, the defect is closed, resection and anastomoses are performed for the distal stenosed part, in addition to the peritoneal toilet and anti-tuberculour therapy (77). However, other workers suggest that emergency surgery should be limited to the perforated area and any additional procedure postponed (78).

The mortality rates for abdominal tuberculosis in the severely ill patients presenting later ranges between 6% (69) and 37% (30) in developed countries and between 14% and 50% in developing countries (79). Morbidity includes delayed wound healing with occurrence of incisional hernia, recurrent obstruction and faced fistula.

OBJECTIVES OF THE STUDY.

Broad objectives.

To determine the pattern of clinical presentation and mode of diagnosis of tuberculosis of the abdomen at the Kenyatta National Hospital.

Specific objectives

1. To determine the pattern of clinical presentation of tuberculosis of the abdomen
2. To determine type of investigations carried out
3. To study outcome of investigations carried out.
4. To advice on most appropriate investigation to be done within our set-up.

RATIONALE.

1. There is a need to know the pattern of presentation of tuberculosis of the abdomen so as to make an early diagnosis and institute appropriate treatment because tuberculosis is a disease that is amenable to treatment by the easily available antimicrobials.

2. Worldwide, there is an upsurge in the incidence of tuberculosis, and with the advent of HIV, tuberculosis of the abdomen is reported to be on the rise in developing countries.

3. Locally, there has been no study to gather information on the pattern of clinical presentation of tuberculosis of the abdomen. Such information would be important to clinicians to enable them have a high index of suspicion and order appropriate investigations to make an early diagnosis.

4. Information about relevant investigations helps the clinician avoid those investigations that do not help in arriving at a diagnosis.

MATERIALS AND METHODS.

Study design:

This was a retrospective descriptive study over a period of 10 years from 1st January 1990 to December 31st 1999.

Study population.

All patients who had a confirmed diagnosis of tuberculosis of the abdomen were included into the study.

Sample size.

A total of 69 patients were included into the study.

STUDY METHODOLOGY.

The study was conducted by the principal investigator under the guidance of a supervisor from the Department of Surgery, University of Nairobi. The instruments of the study comprised medical records of patients who had been diagnosed to have tuberculosis of the abdomen.

The principal investigator studied these medical records in order to determine the history of presentation, clinical findings and the results of investigations carried out; and also record the demographic data. The information gathered was then tabulated and analysed.

ELIGIBILITY CRITERIA.

a. **Inclusions.**

All those patients who were diagnosed to have tuberculosis of the abdomen and treated at the Kenyatta National Hospital. These included:

1. All those patients who were diagnosed to have tuberculosis of the abdomen after a histological confirmation of biopsy specimen.
2. All those patients who were diagnosed to have tuberculosis of the abdomen after confirmation by microbiological ZN staining of specimen or by culture.
3. All those patients with a positive tuberculin test.

b. **Exclusions.**

1. Those very sick patients with a diagnosis of abdominal tuberculosis as part of generalised milliary tuberculosis with primaries in the lungs and with multiple system involvement.
2. Patients diagnosed and confirmed to have a specific abdominal condition but found to have tuberculosis as an incidental finding.
3. Patient on empirical treatment for unconfirmed tuberculosis of the abdomen.

LIMITATIONS OF THE STUDY.

The principal investigator relied on history, clinical examination and investigations undertaken by someone else.

There were many cases of inadequate history and clinical findings recorded. Many investigations were either not done or information was missing. In this way, the study was denied vital information for compiling a good data size for analysis.

To overcome this, data was traced in a retrograde manner by first tracing records on tuberculosis of the abdomen from the Department of Pathology and then trying to trace the files with that information. There were still many hindrances as sometimes the recorded information in the two departments were not tallying.

Ethical considerations.

Permission was sought from the Ethical and Research Committee for the study. The study was commenced only after permission had been granted.

Data management.

Data was entered and analysed using the SPSS (Statistical Package for Social Sciences) programme. The result of this analysis is presented below.

RESULTS.

Total number of patients treated for TB abdomen 172

Total number of patients confirmed to have TB abdomen 69 (40.1%)

Table 2: Frequency table for age distribution.

Age group	Frequency	Percentage
0 - 10	8	11.6
11 - 20	18	26.1
21 - 30	18	26.1
31 - 40	16	23.2
41 - 50	6	8.7
> 50	3	4.3
Total	69	100

Age range 1.4 years - 57 years

Mean age = 26 years

mode=16 years

Table 2 shows age distribution with the age ranging between 1.4 years to 57 years with a mean age of 26 years and mode of 16 years. 49.3% of the patients were between 21-40 years old.

Figure 1: Age distribution.

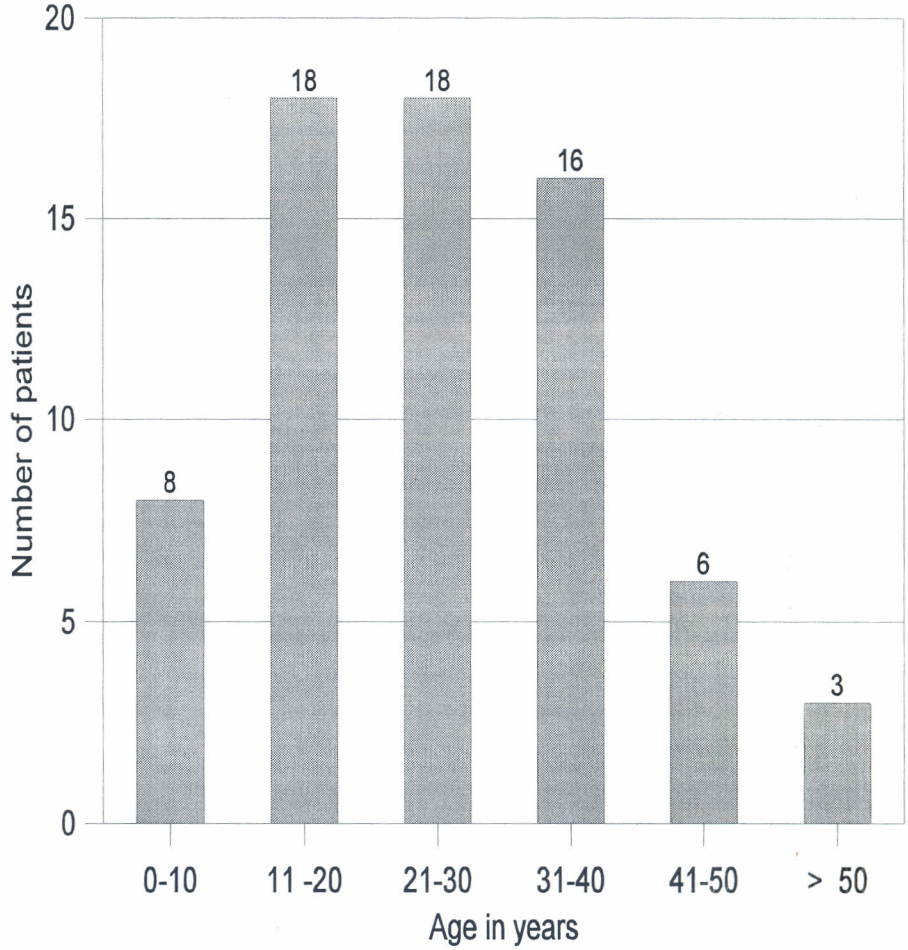


Table 3: Sex distribution

Sex	Frequency	Percentage (%)
Male	30	44.8
Female	37	55.2
Total	67	100

Table 3 shows the sex distribution in 67 patients whose data was available. 44.8% were males and 55.2% were females.

Figure 2: Sex distribution.

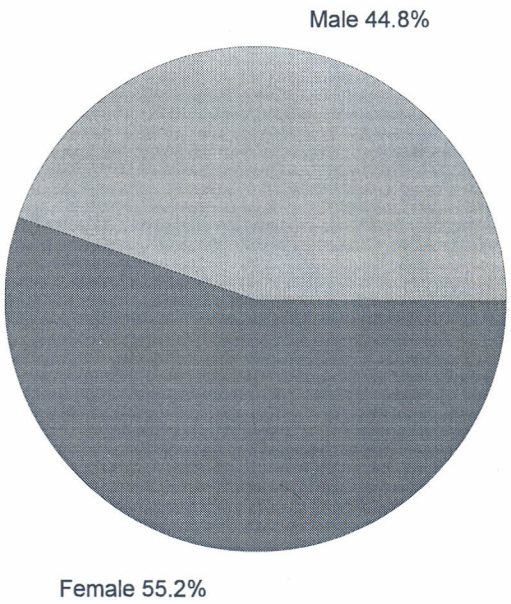


Table 4: Ethnic distribution

Tribe	Frequency	Percentage (%)
Kikuyu	23	36.5
Luo	12	19.1
Kamba	11	17.5
Luhya	4	6.3
Others	13	20.6
Total	63	100

Table 4 shows ethinic distribution in 63 patients. The Kikuyu showed the highest frequency (36.5%) among all the other tribes.

Figure 3: Ethnic distribution

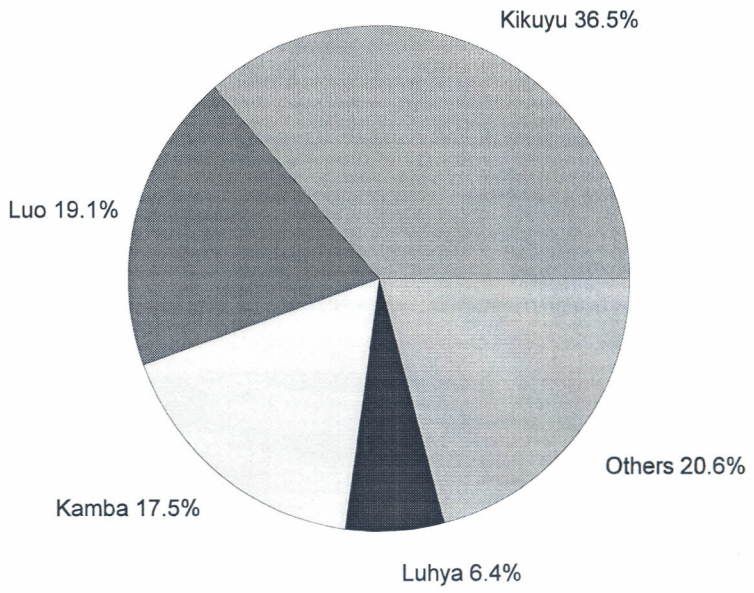


Table 5: Distribution of occupation.

Occupation	Frequency	Percentage
Unemployed	35	53.9
Salaried employment	11	16.9
Student/pupil	10	15.4
Self employed	6	9.2
Peasant farmer	3	4.6
Total	65	100

Table 5 shows the distribution of occupation in 65 patients whose data was available.

Majority of the patients (53.9%) were unemployed.

Table 6: Symptoms and duration

Complaint	Frequency	mean duration in days	Percentage
Anorexia	12	70.9	17.4
Weight loss	19	145	27.5
Abdominal swelling	54	89	78.3
Abdominal pain	42	105.4	60.9
Fever	17	130.2	24.6
Night sweats	13	239.4	18.8
Vomiting	15	22	21.7
Diarrheoa	11	33.1	15.9
• Others			
• Lethargy	1		1.4
• P.V. discharge	1		1.4
• Constipation	6		8.7
• Gaseousness	1		1.4
• Umbilical discharge	1	125.1	1.4
• Cough	4		5.8
• Chest pain	1		1.4
• Backache	1		1.4
• Jaundice	1		1.4

The duration of symptoms ranged between 22 days and 239.4 days.

Table 6 shows the distribution of symptoms and their duration. The most common complaint was abdominal swelling (78.3%) followed by abdominal pain (60.9%). The complaint with the longest mean duration was night sweats (239.4 days) while the complaint with the shortest duration was vomiting (22 days).

Table 7: Nature of presentation of TB abdomen

Presentation	Frequency	Percentage
Acute ≤2weeks	11	15.9
Chronic > 2 weeks	58	84.1
Total	69	100

Table 7 shows the form of presentation of abdominal tuberculosis. 15.9% presented in the acute form while 84.1% presented in the chronic form.

Figure 4 : Nature of presentation of TB abdomen.

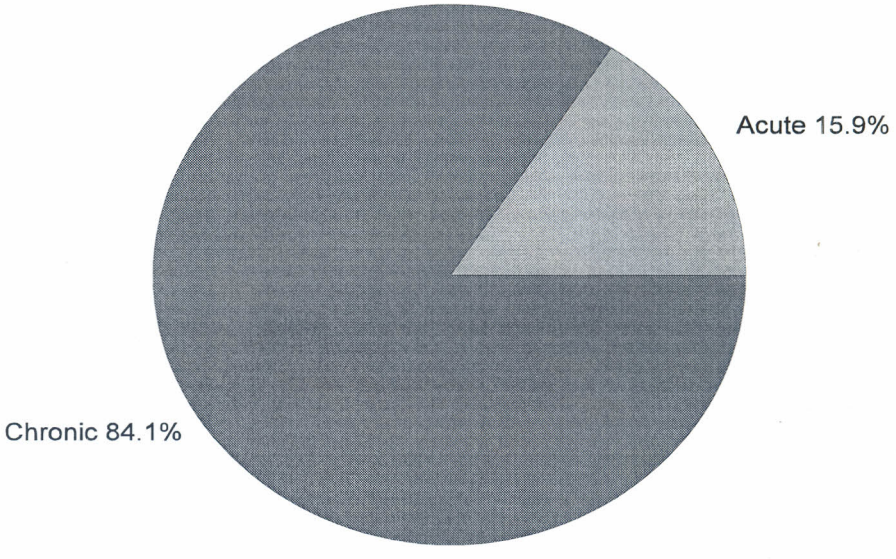


Table 8: Physical findings in acute presentation.

Physical finding	Frequency	Percentage of occurrence
.Intestinal obstruction	6	54.5
Intestinal obstruction + peritonitis	5	45.5
Acute tubercular appendicitis	2	18.2
Acute lymphatic adenitis	1	9.1

Table 8 shows the frequency of physical findings in acute presentation. The most common physical finding was intestinal obstruction 6 (54.5%) followed by intestinal obstruction and peritonitis 5 (45.5%).

Table 9: Physical findings in patients with chronic presentation

Physical finding	Frequency	Percentage of occurrence
Ascites	35	59.3
Abdominal distension	34	42.2
Abdominal mass	19	32.2
Tenderness	10	16.9
Hepatomegally	7	11.9
Splenomegally	7	11.9
Rectal bleeding	4	6.8
Adnexal mass	1	1.7
Recurrent appendicitis	1	1.7

Table 9 shows the frequency of physical findings in patients with chronic presentation. Ascites was the most common finding 59.3%, followed by abdominal distension 42.2% and third was abdominal mass 32.2%.

Table 10: Haemoglobin levels

Haemoglobin gm/dl	Frequency	Percentage
1-5	1	1.6
5.1-10	25	40.3
10.1-15	34	54.9
15.1-20	2	3.2
Total	62	100

Range: 4.6gm/dl-16.9gm/dl

Mean haemoglobin 10.6gm/dl

Mode 8.2 gm/dl

Table 10 shows haemoglobin levels in 62 patients whose data was available. The levels ranged between 4.6gm/dl-16.9gm/dl. Both the mean (10.6g./dl) and the mode (8.2gm/dl) were below the normal levels.

Figure 5: Haemoglobin levels.

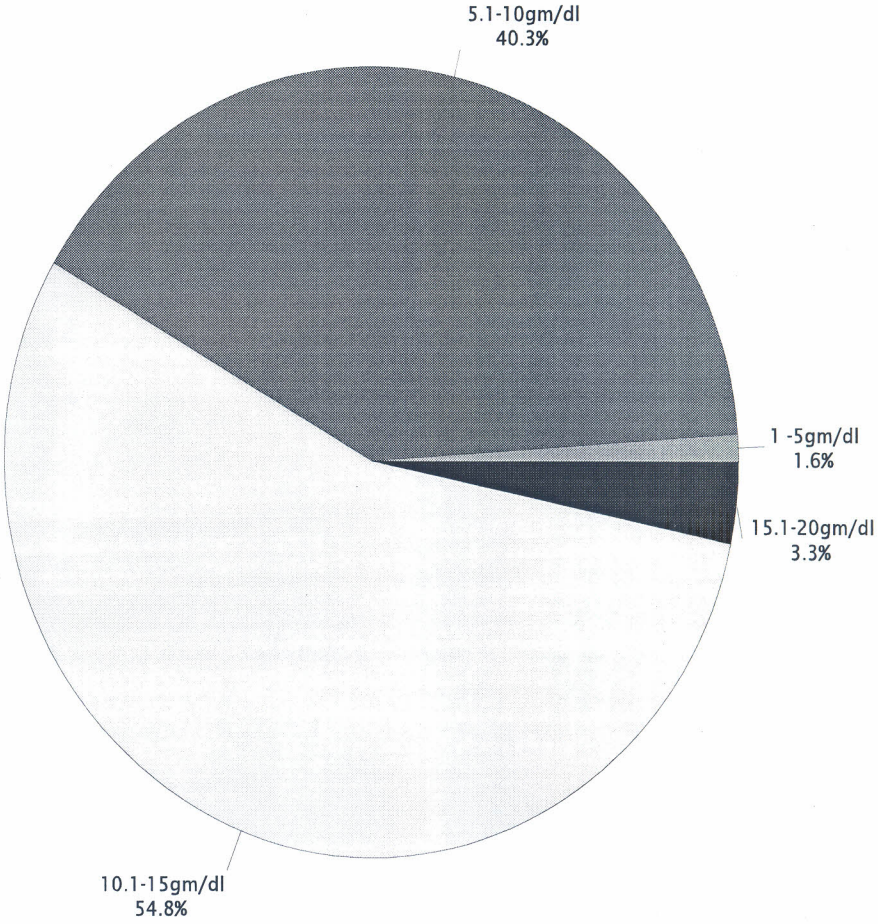


Table 11: Red blood cell count.

Count ($x10^{12}/l$)	Frequency	Percentage
< 3	5	15.2
3-6	28	84.8
Total	33	100

Red blood cell count range: $1.8x10^{12}/l$-$5.9x10^{12}/l$

Mean: $3.9x10^{12}/l$

Mode :$3.8x10^{12}/l$

Table 11 shows the frequency of red blood cell count in 33 patients whose data was available. The range of red blood cell count was between $1.8x10^{12}/l$-$5.9x10^{12}/l$. Both the mean $3.9x10^{12}/l$ and the mode $3.8x10^{12}/l$ were below normal.

Figure 6: Red blood cell count ($x10^{12}$/l).

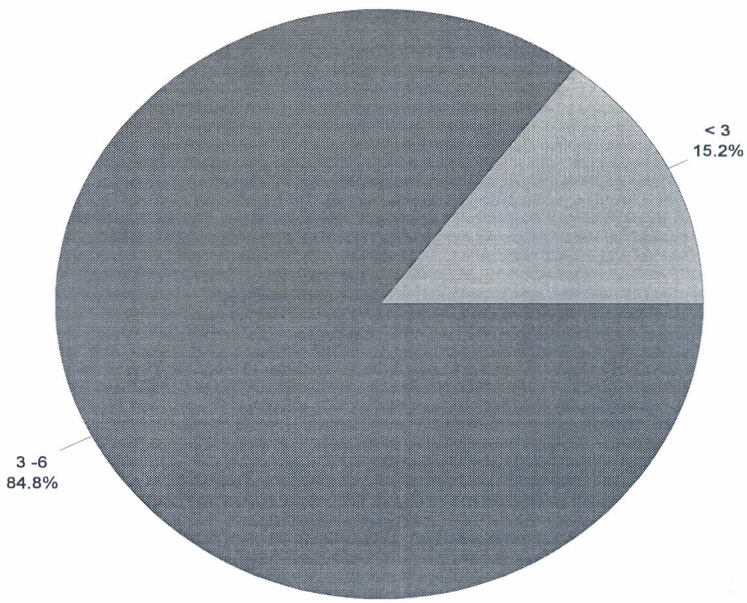
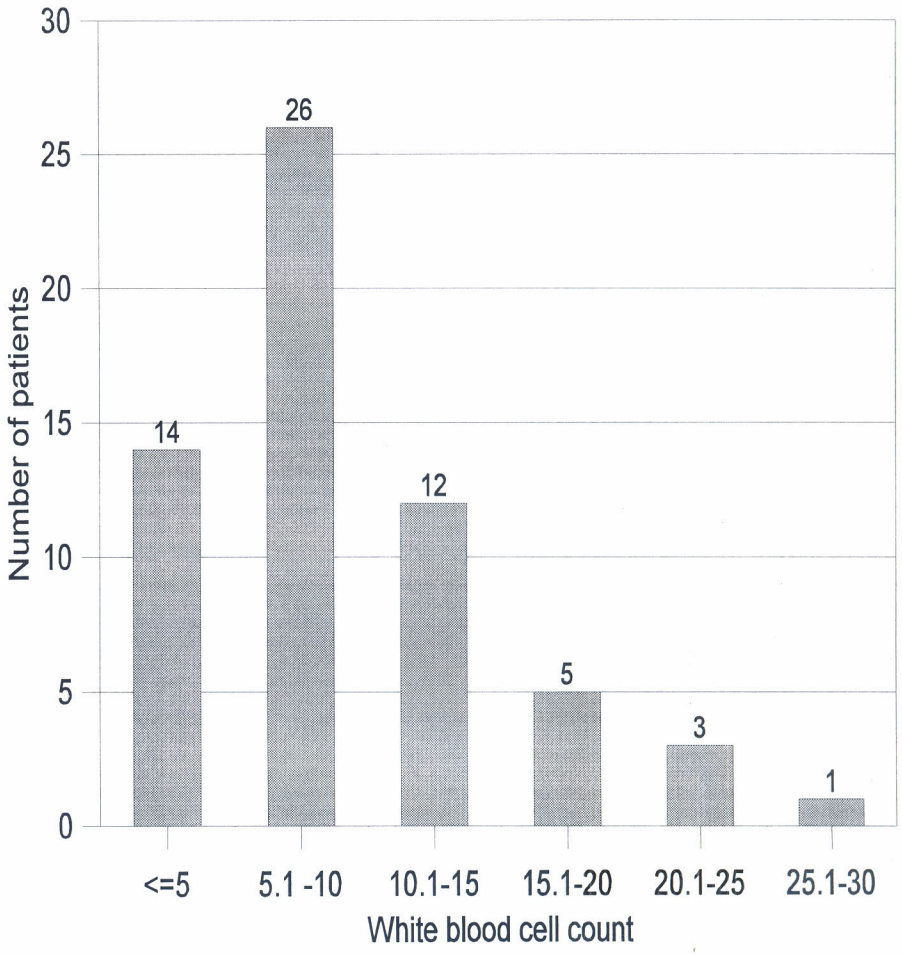


Table 12: White blood cell count

Count (10^{12}/l)	Frequency	Percentage
≤ 5	14	23
5.1-10	26	42.6
10.1-15	12	19.7
15.1-20	5	8.2
20.1-25	3	4.9
25.1-30	1	1.6
Total	61	100

Table 12 shows white blood cell count in 61 patients whose data was available. The range was between $3.1x10^9$/l-$29.9x10^9$/l. Both the mean ($9.4x10^9$/l) and the mode ($4.9x10^9$/l) lie within the normal range.

Figure 7: White blood cell count.



Range:3.1x10^9/l-29.9x10^9/l.

Mean: 9.4x10^9/l.

Mode : 4.9x10^9/l.

Table 13: Percentage lymphocyte count.

Count (%)	Frequency
< 10	3
10.1-20	9
20.1-30	6
30.1-40	7
40.1-50	7
50.1-60	3
60.1-70	1
70.1-80	2
80.1-90	2
> 90	1
Total	41

Table 13 shows percentage of lymphocyte count with a range between 4.3%-94.0%, the mean was 37.0% and mode 30% while both were within the normal range. Data for 41 patients was available.

Figure 8: Percentage lymphocyte count.

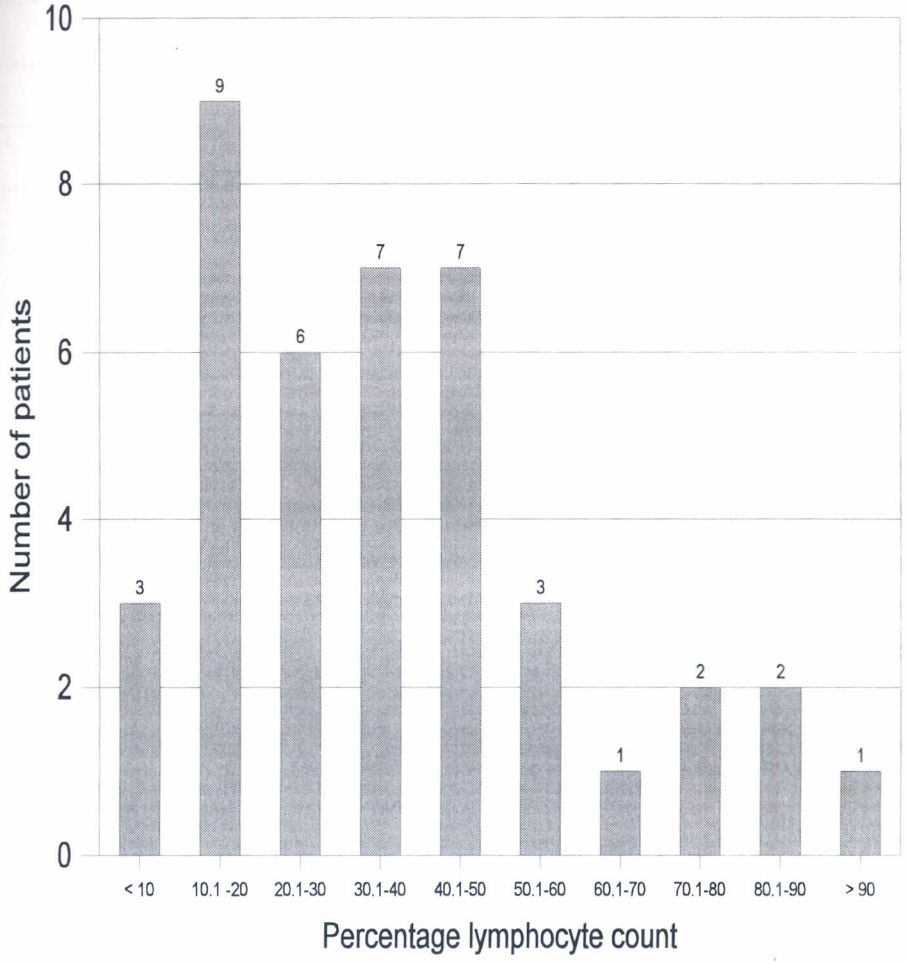


Table 14: Erythrocyte sedimentation rate.

MM/hr	Frequency	Percentage
< 10	2	5.3
11-20	4	10.5
21-30	4	10.5
31-40	3	7.9
41-50	6	15.8
51-60	9	23.7
61-70	6	15.8
> 70	4	10.5
Total	38	100

Range 6mm/hr - 73mm/hr

Mean ESR 46.2 mm/hr

Mode: multiple modes exist.

Table 14 shows the distribution of erythrocyte sedimentation rate with a range between 6mm/hr to 73mm/hr. The mean ESR was 46.2mm/hr which was raised. 73.7% of the patients had ESR above 30mm/hr.

Table 15: Tuberculin test according to age.

Tuberculin test +ve	Age (years)
12mm	4
20mm	4
not reported	9
20mm	15
reported as +ve	30
reported as +ve	36
20mm	48
20mm	55
Done but -ve	35

Total = 9 patients

+ve = 77.8%

-ve = 22.2%

Range 10mm-22mm

Table 15 shows the tuberculin test according to age in 9 patients whose data was available of which 77.8% of patients had a positive tuberculin test and 22.2% had a negative tuberculin test.

Figure 10: Tuberculin test.

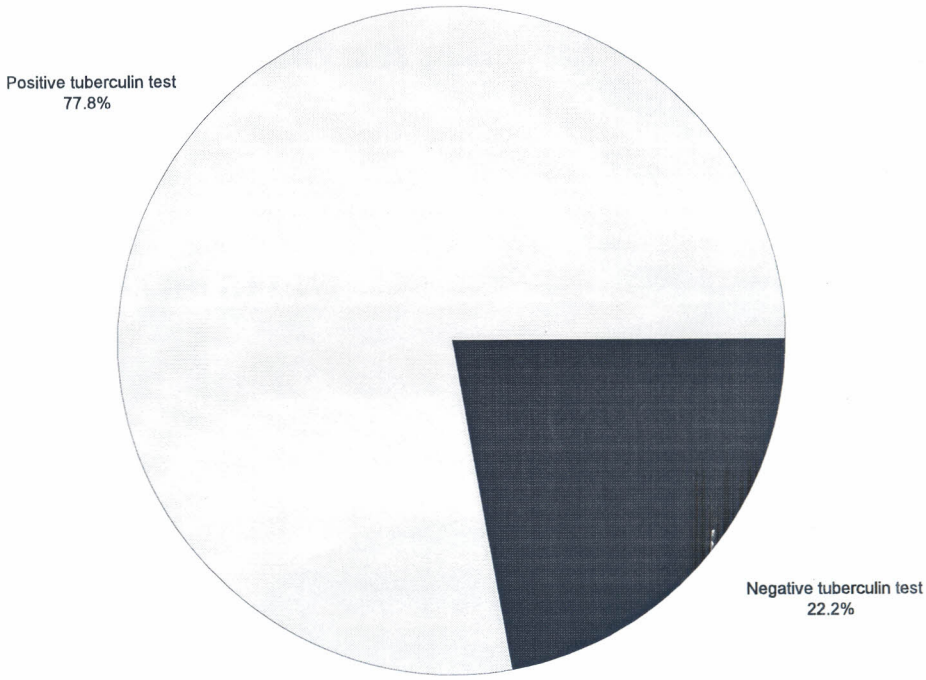
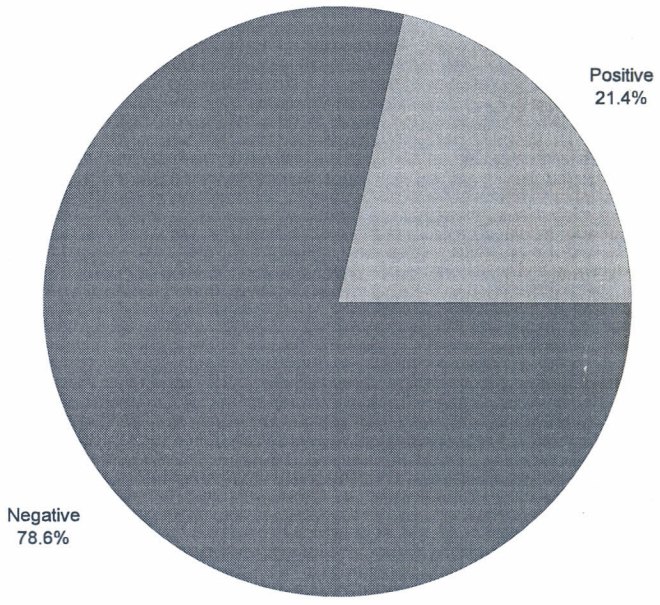


Table 16: ELISA test for HIV

Test	Frequency	Percentage
Negative	22	78.6
Positive	6	21.4
Total	28	100

Table 16 shows Elisa test for HIV in 28 patients. 78.6% tested negative while 21.4% tested positive.

Figure 11 : ELISA test for HIV.



ZN staining for FNAC

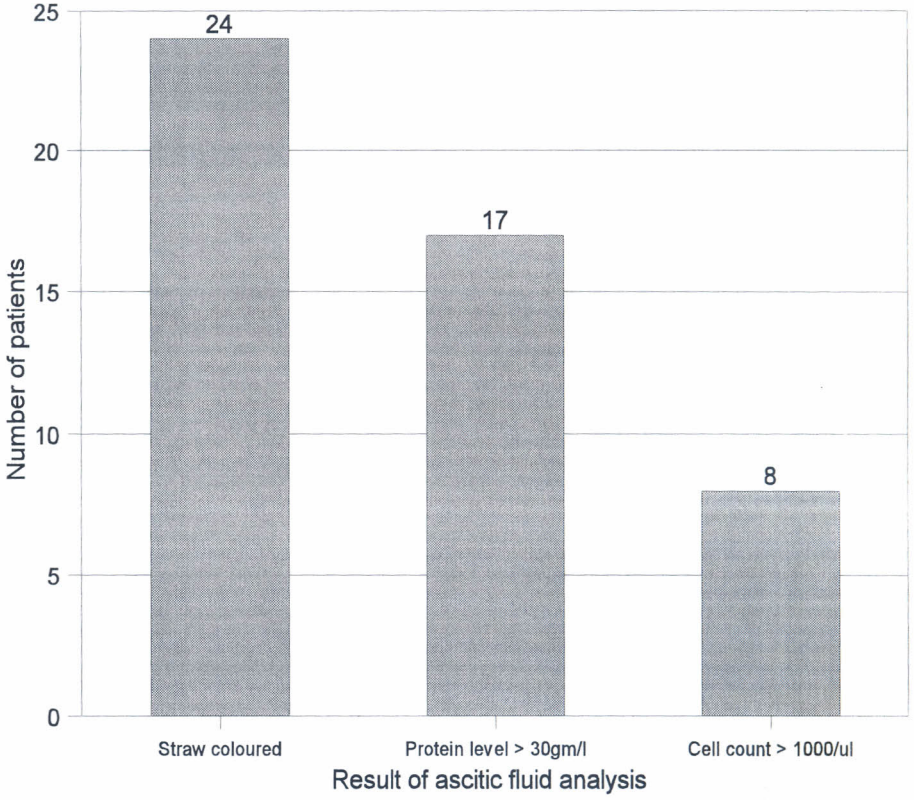
- only one patient (1.45%) of all the 69 patients had a positive ZN for AFB.

Table 17: Result of ascitic fluid analysis.

Result	Frequency	Percentage
Straw coloured	24	60
Protein level > 30gm/l	17	42.5
Cell count > 1000/ul	8	20.5
Total	40	100

Table 17 shows the result of analysis of ascitic fluid in 40 patients. Sixty percent (60.0%) was straw coloured, 42.5% had protein levels > 30gm/dl and 20.5% had a cell count > 1000/ul.

Figure 12: Results of ascitic fluid analysis.



Fourteen patients had ascitic fluid glucose levels determined.

Range of glucose levels was 0.4mmol/l-6.3mmol/l

Mean levels =5.0mm/l

Table 18: Total serum protein.

GM/L	Frequency
37.0	1
42.0	1
50.3	1
53.0	1
54.0	1
59.0	1
76.0	1
77.0	1
81.0	1
81.9	1
84.1	1
86.0	1
88.0	1
91.0	1
95.0	1
Total	15

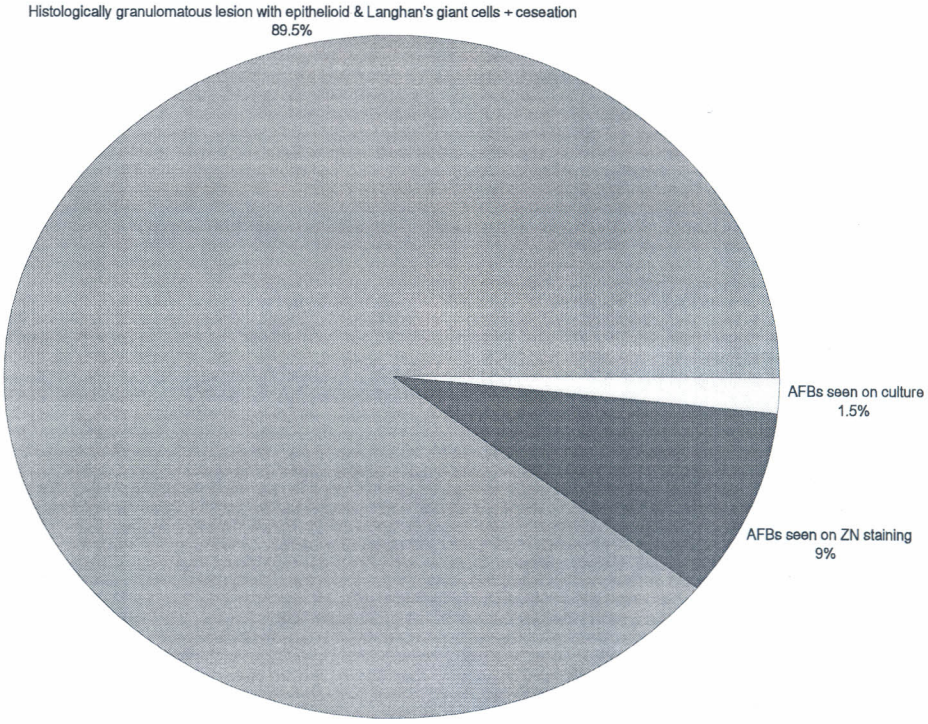
Table 18 shows the levels of total serum protein in 15 patients with a range between 37.0gm/dl to 95gm/l. The mean of 70.4 gm/l was within normal range.

Table 20: Biopsy results.

Biopsy result	Frequency	Percentage (%)
Histologically granulomatous lesion with epithelioid and Langhan's giant cells + ceseation	60	89.5
AFBs seen or ZN staining	6	9.0
AFBs on culture	1	1.5
Total	67	100

Table 20 shows the result of biopsy in 67 patients whose specimens were taken, 89.5% of specimen histologically showed granulomatous lesion with epitheliod and Langerhan's giant cells and caseation, 9.0% showed AFBs on staining and 1.5% cultured AFBs on special culture media.

Figure 13 : Biopsy results.



Twenty (33.3%) specimen biopsies were obtained by blind percutaneous needle biopsy while 40 (66.7%) were obtained by laparotomy.

Six (10%) specimen biopsies were positive for AFBs after ZN staining.

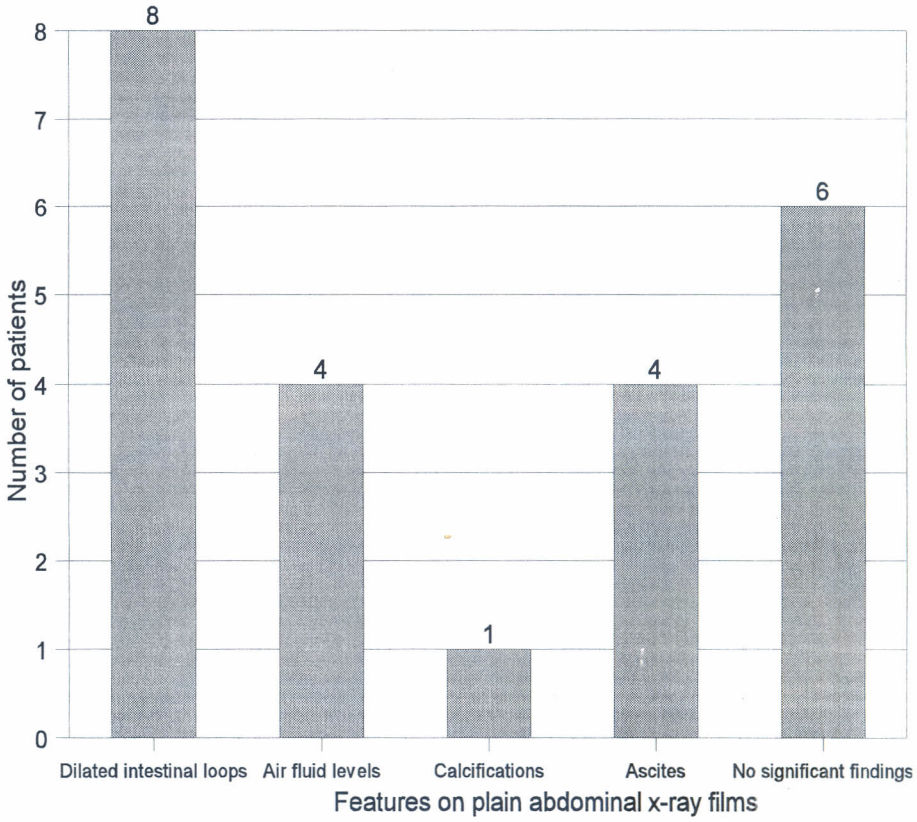
Culture of ascitic fluid grew AFBs in one (1.5%) specimen.

Table 21: Features on plain abdominal x-ray films in 23 patients.

Feature	Frequency	Percentage (%)
Dilated intestinal loops	8	34.0
Ascites	4	17.4
Air fluid levels	4	17.4
Calcifications	1	4.3
No significant finding	6	26.9
Total	23	100

Table 21 shows plain abdominal x-ray film findings in 23 patients whose data was available. Majority of patients 34.0% had dilated intestinal loops, 17.4% had ascites, 17.4% had air fluid levels and 4.3% showed calcifications. No significant findings were seen in 26.9% of the patients.

Figure 14 : Features on plain abdominal x-ray.



No barium enema or colonoscopy was performed in any patient.

Table 22: Abdominal ultrasound results.

Abdominal ultrasound results	Frequency	Percentage
Ascites	27	55.1
Enlarged lymph nodes	5	10.2
Hypertrophic intestines	8	16.3
Others		
• Cystic mass	7	14.3
• Matted intestines	1	2.0
• Hydronephrotic kidney, cystic mass	1	2.0
• Appendicular mass	2	4.0
• Ovarian mass	1	2.0
• Splenomegally	2	4.0
• Pelvic mass	2	4.0
• Matted bowel	1	2.0
• Adnexal mass	2	4.0
• Loops matted	1	2.0
• Abdomen mass	3	6.0
• Hepatosplenomegally	1	2.0
• Hepatomegally	2	4.0
• Retroperitoneal mass	1	2.0
• Loops floating in fluid	1	2.0
• Thick walled ovarian cyst	1	2.0
• Gas filled intestines	1	2.0

Table 22 shows the results of abdominal ultrasonography performed on 49 patients. Ascites was demonstrated in 27 (55.1%)of the patients, enlarged lymphnodes in 5 (10.2%) of patients and hypertrophic intestinal lesions were seen in 8 (16.0%) of patients.

Table 23: CT Scan findings in 14 patients.

Features	Frequency	Percentage
Adherent bowel loops	2	14.4
Thickened omentum and irregular soft tissue density	1	7.1
Thickened bowel wall	1	7.1
Massive ascites	1	7.1
No significant findings	9	64.3
Total	14	100

Table 23 shows CT-scan findings in 14 patients where by 14.4% showed adherent bowel loops, 7.1% had thickened omentum and irregular soft tissue density, 7.1% had thickened bowel wall, 7.1% had massive ascites and 64.3% showed no significant findings.

Table 24: Important laparotomy findings in 40 patients

Finding	Frequency	Percentage of occurrence %
widespread tubercular nodules	25	62.5
Wide spread adhesions	22	55.0
Thickened peritoneum + omentum	17	42.5
Ascites	14	35.0
Abdominal mass	10	25.0
Pus	4	10.0
Dilated loops of intestine	2	5.0

Table 24 shows important laparotomy findings in 40 patients in whom laparotomy was performed. The commonest finding was widespread tubercular nodules (62.5%) followed by wide spread adhesions (55.0%), then thickened peritoneum and mentum (42.5%) and ascites constituted 35.0% while abdominal mass occurred in 25.0%..

DISCUSSION.

Demography.

No local study has been done to determine the pattern of presentation and investigations for abdominal tuberculosis in our set up. A total of 172 patients were treated for abdominal tuberculosis during the period of study, January 1991-December 1999. Only 69 patients (40.1%) were recruited into the study because they had a confirmed diagnosis.

The age range was between 1.4 years to 57 years with a mean age of 26 years and a mode of 16 years. This confirms that this disease occurs at any age but it is predominantly a disease of young adults (21,22). There were 30 (44.8%) males and 37 (55.2%) females. These figures are not very different. Although some reports mention a higher incidence in females (23), it seems that the disease affects both sexes equally (20).

The Kikuyu ethnic community showed the highest frequency (36.5%) followed by Luo (19.1%) and third was the Kamba (17.5%). The Kikuyu is the most populous ethnic group in Kenya, and its close proximity to Nairobi and the fact that Nairobi has a higher population of this ethnic group explains this finding.

The highest number of patients were unemployed (53.9%), people in salaried employment were 16.9%, students/pupils were 15%, self employed were 9.23% and peasant farmers were 4.62%. Most of the patients were unemployed and hence of low

socio-economic background. These are people deprived of nutritious food which helps build strong body defenses against infections. They are people who also live in poor unhygienic environment with crowded houses which subjects them to higher chances of being infected. A similar trend was found by Bistani et al (15) in a study of 30 cases.

History and physical findings.

The most common complaints were abdominal swelling in 54 (78.3%) patients followed by abdominal pain in 42 (60.9%) patients. Weight loss occurred in 19 (27.5%), fever 17 (24.6%), vomiting 15 (21.7%), night sweats 13 (18.8%) and diarrhoea 11 (15.9%). The presence of abdominal swelling corresponds well with ascites and dilated intestinal loops which are commonly found in patients with abdominal tuberculosis. This is same for abdominal pain that may be associated with abdominal distension and the presence of abdominal masses.

The mean duration of symptoms ranged from 22 days to 239.4 days. This is because abdominal tuberculosis is mainly a chronic illness. Eleven (15.9%) of the patients presented as acute abdomen and majority (84.1%) presented with chronic abdominal symptoms. In other studies, history goes back some 3 or 4 years (37), and the mean duration of stay of immigrants in the U.K. before diagnosis of gastrointestinal tuberculosis was 4 years (37).

For those patients who presented with acute abdominal symptoms, majority of them 6 (54.6%) presented with intestinal obstruction and 5 (45.5%) with intestinal obstruction together with peritonitis. Acute tubercular appendicitis occurred in 2 (18.2%) and acute lymphatic adenitis in only one patient (9.1%). Acute form of abdominal tuberculosis is rare; it includes acute tuberculous appendicitis, acute intestinal obstruction and acute tuberculous peritonitis due to perforation (45,46). Majority of the patients 35 (59.3%) presenting with chronic symptoms had ascites, followed by abdominal distention 34 (42.4%) and abdominal mass 19 (32.2%). In other studies, more than 95% of patients have ascites (49) while an abdominal mass occurred in 50% of these patients (47). The abdominal distension corresponds very well to the high frequency of ascites which is the likely cause of the distension. In this study, abdominal mass occurred in fewer patients than what has been shown in other studies. It would have been expected that an abdominal mass masked by ascitic fluid would be demonstrated by ultrasonography or by CT-scan or at laparotomy. But this is not seen in this study. The most common pathological entity of tuberculosis of the abdomen in our set up is most therefore tuberculous peritonitis. This is the pathological entity most likely to be associated with ascites than the gastrointestinal or the glandular types both of which can present with a mass.

Investigations.

The range of haemoglobin was between 4.6gmdl and 16.9 gm/dl with a mean of 10.6gm/dl and a mode of 8.2gm/dl. The normal range of haemoglobin between 12gm/dl

and 16 gm/dl for females and between 14gm/dl and 18 gm/dl for males. The mean haemoglobin and the mode lies below the two normal ranges. Patients with abdominal tuberculosis tends to have a low haemoglobin as seen in other studies (56,57). These are patients who have a chronic illness. Coupled with anorexia and poor social economic status, they are people who are nutritionally deprived ending up with anaemia.

The patients had an erythrocyte sedimentation rate between 6mm/hr and 77mm/hr with a mean of 46.2mm/l which is higher than the normal range of between 0 and 30mm/hr. Twenty eight (73.7%) patients had ESR above 30mm/hr. Al Hadeedi et al (22) in a different study found out that 70.0% of the patients had a raised ESR. A raised ESR is a common finding in patients with chronic inflammatory or infectious illness such as abdominal tuberculosis.

The range of red blood cell count was between $1.8x10^{12}$ and $5.9x10^{2}$/l with a mean of $3.9x10^{12}$/l. This mean was below the normal range of between $4.7x10^{12}$/l and $6.1x10^{12}$/l. Although other studies do not put any emphasis on the red blood cell count, this figure seems to tally well with anaemia which is common in these patients. It could be a good parameter in confirming anaemia.

The range of white blood cell count in this study was between $3.1x10^{9}$/l and $29.0x10^{9}$/l with a mean of $9.4x10^{9}$/l and lymphocyte count range between 4.3% and 94.0% with a mean of 37.0% and mode of 30%. In this study, both the white blood cell count and the

lymphocyte count were within the normal range. This is contrary to other studies which indicate that most patients have a leucocytosis mainly lymphocytosis (56,57). Most of our patients being of poor nutritional status, as indicated by the low serum albumin level (table 19) are unable to mount an adequate cell mediated immunity to combat the disease. Its only in immunocompromised patients that you find a similar picture, but in HIV positive patients you get a pancytopaenia.

Seven patients (78.6%) had a positive tuberculin test while 2 (22.2%) had either a negative result or not reported at all (table 15). Darrney et al (28) and Jakubawski A. et al (58) in their different studies emerged with the conclusion that up to 15%-100% of patients with abdominal tuberculosis have a positive tuberculin test. However, this test is not helpful in patients who have been immunised with B.C.G. before and especially so in children. ELISA test for HIV was performed in 28 patient and only 6 (21.4%) patients had a positive result. Different studies have given different incidences in HIV positive patients ranging from 40.0% (13) in one study to more than 65% (14) in another. The figure in our study is lower than what other studies show. Perhaps other factors such as tropical diseases e.g. malaria and malnutritional state could be predisposing factors rather than HIV *per se*.

Only 1 (1.5%) patients of all the 69 patients had a positive Z.N. staining for A.F.B.s. This is low just as in other studies which gave positive results in 3.0% of patients (62,63,64).

In the analysis of ascitic fluids, 60.0% of the cases had straw coloured ascitic fluid, 42.5% had a protein level of > 30gm/l and only 20.5% a cell count > 1000/ul. Fourteen patients had their ascitic fluid glucose levels determined with a range of between 0.4mmol/l and 6.3mmol/l and a mean of 5.0mmol/l. Blood glucose was not tested. Ascitic fluid in tuberculosis of the abdomen is usually exudative with a protein level > 30g/dl and a white cell count of > 1000/mm^2 predominantly lymphocytes. These findings usually support the diagosis (58). The glucose level in ascitic fluid in this study (5.0mmol/l) is similar to blood sugar levels (3.5-5.6mmo/l). This confirms that the type of ascitic fluid in abdominal tuberculosis is mainly an exudate following some form of peritonitis.

Fifteen of our patients had total serum protein tested with a range of between 37.0gm/dl to 95gm/dl with a mean 70.4 gm/l. The serum albumin ranged between 4.1gm/l and 43.0gm/l with a mean of 25.3gm/l. Normal range of total serum protein is between 65gm/dl and 80gm/dl and that of albumin is between 38gm/dl and 51gm/dl. The total serum protein in our patients was within the normal range. But the serum albumin was below normal range. MacLaughlin (59) found that patients with abdominal tuberculosis have a normal total serum protein level, predominantly gama globulinaemia. This explains the normal total serum protein (59). The predominant chronic form of abdominal tuberculosis implies that the patients are in poor nutritional status. This is reflected by the low albumin serum levels which is a good biochemical indicator of nutritional status of a patient with a chronic illness as in abdominal tuberculosis.

Sixty patients (89.5%) in the study had their diagnosis confirmed by histological examination of biopsy specimen. Twenty specimens (33.3%) were obtained by blind percutaneous needle biopsy and 40 specimens (66.7%) were obtained by laparotomy. Six specimen biopsies and aspirated material (10%) were positive for AFBs after ZN staining which confirmed the diagnosis. Only 1 patients (1.5%) had a positive culture of aspirated fluid for AFBs.

Tissue biopsy for histology is the mainstay procedure in diagnosis of TB of the abdomen (53). This has been reflected in this study. But at Kenyatta National Hospital, most of the biopsies (66.7%) were obtained at laparotomy. In other centres, laparoscopy is the diagnostic procedure of choice (54) and can be performed under local anaesthesia if necessary. This procedure is not well established in the general surgical unit at Kenyatta National Hospital. This explains why most patients have to undergo laparotomy to obtain a biopsy specimen.

Blind percutaneous needle biopsy is an attempt to bridge the gap between laparotomy and laparoscopy. This procedure can be made safer by introducing echo-guided biopsy which is less invasive and can replace diagnostic laparoscopy or laparotomy (68). Studies also suggest that echo guided biopsy is effective in diagnosis of abdominal tuberculosis. Echo guided puncture can replace diagnostic laparoscopy or laparotomy (67).

Ten percent of the patients had positive ZN staining for AFBs of biopsy or aspirated material and only 1.5% of the patients had a positive culture of aspirated material.

Other studies have shown that ZN-staining of ascitic fluid is only positive in 3.0% of the patients and culture of ascitic fluid is positive in 20.0% of the patients (62,63,64). Our figure for ZN staining is high because it included not only ascitic fluid and FNAC but also biopsy specimens. Our culture results fall short of what has been observed elsewhere. Tubercle bacilli are strict aerobes with a rather narrow range of growth temperature around 37°C, are exacting in their nutritional requirements and will not grow on ordinary media. Lowenstein-Jensen medium is widely used. Even on optimal media growth is very slow, and colonies take ten days at least and more commonly several weeks, to become visible.

A total of 23 patients had a plain abdominal x-ray done. The most common finding on a plain abdominal x-ray film was dilated intestinal loops in 8 (34.0%) of patients followed by air fluid levels and ascites each in 4 (17.4%) patients and calcifications is only one patient (4.3%). Six (26.9%) x-ray films showed no significant findings. Prakash A. in his series of 300 patients (23) found that many x-rays may show dilated intestinal loops and air fluids obstruction. These results have been reflected in this study with 34.0% showing dilated intestinals and 17.4% of showing air fluid levels (table 20).

No barium enema or colonoscopy was performed in this study. Abdominal ultrasonography was performed in 49 patients and revealed ascites in 27 (55.1%) patients, hypertrophic intestines in 8 (16.0%) patients, enlarged lymph nodes in 5 (10.5%) patients and cystic abdominal mass in 7 (14.3%) patients. Other features occured at varying frequencies ranging between 1 and 7. Ultrasonography is advocated in abdominal tuberculosis although it is non specific. It may show ascites and lymphadenopathy with bowel wall thickening as shown by Sheikh et al (67). This study to some degree has demonstrated similar findings in Sheikh et al study.

Fourteen patients in this study had CT-scan performed. In two patients (14.4%) adherent bowel loops were found. Other features such as thickened omentum and irregular soft tissue density, thickened bowel wall and massive ascites occurred only in one (7.1%) patient each. Like ultrasound, CT-scan is advocated although it is also non specific. Most patients (64.3%) had no significant findings. Very few patients in our study had CT scan performed because it is an expensive investigation that is only performed when the patient can afford. As indicated earlier (table 5), most of the patients in this study are of low socio economic status and perhaps this is why few of them could have CT scan done. However, the features demonstrated on CT-scan in this study have been seen in another study (67).

Laparotomy.

Laparotomy was performed in 40 patients (table 22). The most frequent finding at laparotomy was the presence of widespread tubercular nodules in 25 (62.5%) patients followed by widespread adhesions 22 (55.0%) patients, thickened peritoneum and omentum 17 (42.5%) patients, ascites in 14 (35%) patients, abdominal mass in 10 (25.0%) patients, dilated loops of intestines in 2 (5.0%) patients and pus in the peritoneum cavity in 4 (10%) patients in that order. These observations are similar to those found by Calder et al (12) that scattered tubercules and thickened omentum forming transverse band and ascites as important findings at laparotomy. In the same study, the presence of purulent effusion was extremely rare. In my study, pus was only found in 4 (10%) patients.

CONCLUSIONS.

1. Abdominal tuberculosis, though rare, occurs in both paediatric and adult Kenyan population as observed at Kenyatta National Hospital, but it occurs predominantly between 21 years and 41 years (71.4%)
2. Both acute and chronic forms of abdominal tuberculosis occur in our population, but it is predominantly a chronic illness as observed in 84.1% of the patients in this study.
3. A long standing history of abdominal distension (78.3%), abdominal pain/discomfort (60.9%), with clinical findings of abdominal distension (42.2%) and demonstrable ascites (59.3%) should give the clinician a high index of suspicion of the likelihood of a diagnosis of abdominal tuberculosis.
4. Not all investigations performed on these patients before diagnosis is confirmed were necessary. Investigations done should be result oriented.
5. Tissue biopsy and histology is the most reliable method of confirming the diagnosis.
6. Laparotomy is the most common method of obtaining a biopsy specimen. At laparotomy, the presence of widespread tubercular nodules (62.5%) and thickened peritoneum and omentum (42.5%) and ascites (35%) are the tell tale signs of abdominal tuberculosis.
7. Tuberculous peritonitis is the most common pathological entity in our set up than gastrointestinal and the glandular types.

RECOMMENDATIONS.

1. Abdominal tuberculosis should be considered in a patient with a long standing history of swelling of the abdomen, abdominal pain, weight loss and fever, with abdominal distension and ascites demonstrated on physical examination.

2. Tissue biopsy should be obtained to confirm diagnosis. The biopsy can be obtained by echo-guided biopsy or by diagnostic laparoscopy. These two methods should be established as the most appropriate methods of obtaining tissue specimen for histology.

3. Other investigation should include:
   a. Full haemogram and E.S.R
   b. Serum albumin
   c. Biochemical analysis, cytology and microbiological analysis of ascitic fluid.
   d. Montoux test in patients who have not been immunised with BCG

REFERENCES.

1. Palmar K.R., Patel DH., Bhasran GS., Riodan JF., Sui D.B.A.
Abdominal tuberculosis in urban Britain: A common disease
Gut 1985; 26:1296-305

2. Gulth A.A., Kim U.
The reappearance of abdominal tuberculosis
Surgery, Gynae, Obstet. 1991; 172:432-6.

3. Veen J., Kalisvaart N.A.,
Index tuberculosis 1994, Nederland
Koninklijke Nederlandse Centrale Vereniging tot Bestrijding der tuberculos 1996

4. Edington G.M., Gilles H.M.
Pathology in the tropics
2nd edition. London: Wdward Arbold, 1976:396

5. Gupta A.D., Sharma V.P., Rathi G.L.
Anorectal tuberculosis simulating carcinoma
Am. J. Proctology 1976; 27:33-8

6. Hodgson T.J., Duncan J.L., Rogers K
Tuberculosis: Surgical view point
Ann. R. Coll. Surg. Engl. 1988; 70:117-19.

7. Klimach O.E., Ormerod L.P.
Gastrointestinal tuberculosis: A retrospective review of 109 cases in a district general hospital
QJ Med 1985; 56:569-78.

8. Glinsky N.H., Marks I.N., Kotter R.E., Price S.K.
Abdominal tuberculosis. A 10 year review
S. Afr. Med. J. 1983; 64:849-57.

9. Joseph L., Tromba M.D. et al
Primary gastric tuberculosis presenting as pyloric outlet obstruction.
The American Journal of Gastroenterology 1991; 86:1820-22.

10. Addison N.V.
Abdominal tuberculosis - a disease revived
Ann R. Coll. Surg. Engl. 1983; 65:105-11

11. Paustina F.F.
Tuberculosis of the intestines. In: Bockus H. ed.
Gastroenterology, Philadelphia Saunders, 1976; 750-74.

12. Hunter J.
From: Works of John Hunter vol. 1.: Lectures on surgery 1835:567

13. Shafer R.W., Kim D.S., Weiss J.P., Quale J.M.
Extra pulmonary tuberculosis in patients with human immunodeficiency virus infection. Medicine Baltimore 1991; 70:384-97.

14. Rosengart T.K., Coppa J.F.
Abdominal mycobacterial infection in immuno compromised patients
Am. J. Surg. 1990; 159:125-31.

15. Bastani B., Shariatzadeh M.R., Dehdastiti F.
Tuberculous peritonitis - report of 30 cases and review of the literature
QJ.Med. 1985; 56:549-57

16. Francis T.I.
Abdominal tuberculosis in Nigerians. A clinicopathological study. Trop. Geogr. Med,.1972; 24:232-9

17. Nwokolo C.
Ascitis in Africa
Br. Med. J. 1967; 1:33-7

18. V.K. Kappor
Abdominal tuberculosis
Post. grad. Med. J. 1998; 74:459-467.

19. O.G. Ajao., A.Ajao., J.K., Ladipo., A.A. Al-Saigh and T. Malatani
"A silent" abdominal tuberculosis: Case report of six cases
The East African Medical Journal 1993; 70:606-8.

20. Bhansali S.K.
Abdominal tuberculosis: Experiences with 300 cases
A.M.J., gastroenterology 1977; 67:324-37.

21. Das P., Shukla H.S.
Clinical diagnosis of abdominal tuberculosis
Br. Journal Surg. 1976; 63:941-6.

22. Al-Hadeedi & Walia H.S., Al-Sayer H.M.
Abdominal tuberculosis
Can, J. Surg., 1990; 33:233-7

23. Prokash A.
Ulcero-coustrictive tuberculosis of the bowel
Int. Surg. 1978; 63:23-9.

24. Takahashi T., Herrera M.F., Onuma et al
Diagnostic laparotomy in fever of unknown origin
Rev. Invest. Clin. 1991; 43:25-30.

25. Perich J., Ayuso M.C., Vilana R., Ayso J.R., Gardinal C., Mallofre C.
Disseminated lymphatic tuberculosis in acquired immunodeficiency syndrome
Can Associate Radiol J. 1990; 41:353-7.

26. Chan W.S., Leu S.Y., Lin J.K., Lin T.C.
Trend of large bowel tuberculosis and relation with pulmonary tuberculosis
Dis. Colon Rectum 1992; 35:189-92.

27. Essop A.R., Posen J.A., Hodkinson J.H., Segal I
Tuberculosis hepatitis: A clinical review of 96 cases
Q.J. Med. 1984; 212:465-77.

28. Darney W.W., O'Donoghe J.M., Ostrow J.H., Homes K.K., Beaty H.N.
The spectrum of tuberculous peritonitis
Chest 1977; 72:310-15.

29. Addison N.V., Findly J.M.
Abdominal tuberculosis. In: Had field J. Hobsley M.
Current surgical practice. London: Edward Arnold, 1983; 3:48-61.

30. Hill G.S., Tabrisky J., Peer M.E.
Tuberculous enteritis
West J. Med. 1976; 124:440-5.

31. Sinh M.K., Arunabh, Kapoor V.K.
Tuberculosis of the appendix - a report of 17 cases and a suggested aetio pathological classification. Postgrad. Med. J. 1987; 63:855-7.

32. Maharaj B., Leary W.P., Pudifin D.J.
A prospective study of hepatic tuberculosis in Al black patients
QJ. Med. 1987; 63:517-22.

33. Levine C.
Primary macronodular hepatic tuberculosis: US and CT appearances.
Gastrointest. Radiol 1990; 15:307-9.

34. Desai DC., Swaroop VS., Mohandas KM et al
Tuberculosis of the pancrease: A report of three cases.
Am J. Gastroenterol 1991; 86:761-3.

35. Barson B.D., Mendelson D.S., Janus C.L.
Tuberculousabscess of the pancreas in AIDS: CT findings. Mt. Sinai J. Med. (NY) 1989; 56:297-9.

36. Calder J.F., Norredam K.
Chylous ascitis due to tuberculosis
East Afr. Med. J. 1972; 49:684-6.

37. Palmer K.R., Patil D.M., Basran G.S., Rioan J.F., Silk DBA.
Abdominal tuberculosis
J. Infect Dis 1988; 158:687-92.

38. Tandon R.K., Bansal R., Kapur B.M.L., Shriniwas
A study of malabsorption in intestinal tuberculosis: Stagnant loop syndrome
Am. J., Clin. Nutr. 1980; 33:244-50.

39. Walia H.S., Khafagy A.R., Al Sayer H.M et al
Unusual presentation of abdominal tuberculosis
Can J. Surg. 1994; 34:300-6.

40. Anonymous
Abdominal tuberculosis in Britain
B.M.J. 1977; 1:1557.

41. Underwood M.J., Thompson M.M., Sayer R.D., Hall A.W.
Presentation of abdominal tuberculosis to general surgeons
Br. J. Surg. 1992; 79:1077-9.

42. Puri A.S., Vij J.C., Cahudry A. et al
Diagnosis and outcome of isolated rectal tuberculosis diseases of colon and rectum 1996; 91:1126-9.

43. Shulka H.S., Gupta S.C., Serigh G., Serigh P.A.
Tubercular fistula-in-ano
Brit. J. Surg. 1988; 75:38-9.

44. Myer S.R.
Tuberculous fissure-in-ano
Jr. Soc. Med. 1994; 87:46.

45. Katariya R.N., Sood S., Rao P.G., Rao L.N.G.
Stricture-plasty for tubercular stricture of the gastrointestinal tract. Br. J. Surg 1977; 64:496-8.

46. Aston N.O., DeCosta A.M.,
Tuberculous perforation of small bowel.
Postgrad Med. J. 1985; 6:251-2.

47. Mukerjee P., Chopra I.
Gynaecological tuberculosis in relation to abdominal tuberculosis
Proc. Assoc. Surg. E. Africa, 1979;2:81-4.

48. Singh M.M., Bhargava A.N., Jain K.P.
Tuberculous peritonitis: An evaluation of pathogenic mechanism, diagnostic procedures and therapeutic measures.
N. Engl. J. Med. 1969; 281:1091-4.

49. Manohar A., Simjee A.E., Hofejee A.A., Pellengell K.E.
Symptoms and investigative findings in 145 patients with tuberculous peritonitis diagnosed by peritoneoscopy and biopsy over a five year period
Gut 1990; 31:1130-32.

50. Shukla B.S., Huges L.E.
Abdominal tuberculosis in the 1970s: A continuing problem
Br. J. Surg. 1978; 65:403-5.

51. Tukahasli T., Harrasa M.F., Oruma L. et al
Diagnostic laparotomy in fever of unknown origin
Rev. Invest. Chn 1991; 43:25-30.

52. Surveillance of notifiable infectious diseases in Victoria 1995. Publication number 95/0139 infectious diseases unit, public health division, Victoria Government Department of Human Services.

53. Fitzgerald J.M., Menzies R.I., Elwood R.K.
Abdominal tuberculosis
Critical review Dig. 1991; 9:269-81.

54. Hossain J., Al-Aska A.K., Al-Mofleh I.
Laparoscopy in tuberculous peritonitis
J.R. Soc. Med. 1992; 85:89-91.

55. Watlers D.A.K.
Surgery for tuberculosis before and after human immunodeficiency virus infection. A tropical perspective. Br. J. Surg. 1997; 84:8-14.

56. M.E. Ahmed, M.A. Hassan
Abdominal tuberculosis
Ann R. Coll. Surg. Engl. 1994; 76:75-79.

57. Klimach O.E., Ormerald L.P.
Gastrointestinal tuberculosis: A retrospective review of 109 cases in 19 district general hospitals
QJ. Med. 1985; 56:569-78.

58. Jakubawski A., Elwod R.K., Enerson D.A.
Clinical features of abdominal tuberculosis
J. Infect Dis. 1988; 158:687-92.

59. S.McLaughlin, T.Jones, M. Pitcher, P. Evans.
Laparoscopic diagnosis of abdominal tuberculosis
Akst. N.Z.J. Surgery (1998) 68, 599-601.

60. Bhargare D.K., Dasarothy S., Shriniwas M.D., Kushwalia A.K., Duphase H., Kapur B.M
Evaluation of enzyme-lined immunosorbent assay using mycobacterial saline extracted antigen for the sero diagnosis of abdominal tuberculosis
Am., J. Gastroenterol 1992; 87:105-8.

61. B.J. Bouma, K.M.A.J. Tytgat, H.G.Schipper, P.A. Kager
Beware of abdominal tuberculosis
Netherlands Journal of Medicine 51(1997) 119-122.

62. Marshall J.B.
Tuberculosis of the gastrointestinal tract and peritoneum
Am. J. Gastroenterol 1993; 88:989-997.

63. Hibbs R.G., Kamal M., Farid Z.
Abdominal tuberculosis in Cairo, Egypt.
Trans R. Soc. Trop. Med. Hyg. 1994; 88:317-318.

64. Shakil A.O., Korula J., Kanel G.C., Murray N.G.B., Reynolds S.T.B
Diagnostic features of tuberculous peritonitis in the absence and presence of chronic liver disease: A case control study.
Am. J. Med. 1996; 100:179-185.

65. Menzies R.I., Fitzgerald J.M., Mulpeter K.
Laparoscopic diagnosis of ascites in Lesotho
Br. Med. 1985; 291:473-475.

66. Martinez-Vazquez J.M., Ocana I., Ribera E., Segura R., Pascula C.
Adenosine deaminase activity in the diagnosis of tuberculous peritonitis
Gut 1986; 27:1049-53.

67. Sheikh M., Abu-Zidan F., Al-Hilaly M., Benbehani A.
Abdominal tuberculosis: Comparison of sonography and computerised tomography
J. Clin Ultrasound, 1995; 23:413-17.

68. Radhika S., Rajwanski A., Kochhars Dey P., Roy P
Abdominal tuberculosis: Diagnosis by fine needle aspiration cytology
Acta Cytol 1993; 37:673-8.

69. Shali S., Thomas V., Mathan M. et al
Colonoscopic study of 50 patients with colonic tuberculosis
GUT 1992; 33:347-51.

70. Kochhar R., Jajwanshi A., Goenka M.K. et al
Colonoscopic fine needle aspiration cytology in the diagnosis of ileocaecal tuberculosis
Am. J. Gastroenterol 1991; 86:102-4.

71. Singh V., Kumar P., Kanal J., Prakash V., Vaiphei K., Singh K.
Clinico-colonoscopic profile of colonic tuberculosis
Am. J. Gastroenterol 1996; 91:565-8.

72. Tandon H.D., Prakash A.
Pathology of intestinal tuberculosis and its distinction f9orm Crohon's disease
Gut 1972; 13:360-9.

73. Cooke N.J
Treatment of tuberculosis
BMJ 1985; 291:497-8\

74. Balasubiamarian R., Ramachandran R., Joseph P., et al
Interim results of a clinical study of abdominal tuberculosis
Indian J. Tubercul 1989; 36:117-21.

75. Kapoor V.K., Sharma L.K.
Abdominal tuberculosis
Br. J. Surg. 1988; 75:2-3

76. Katanga R.N., Sood S., Rao P.H., Rao L.N.G.
Stricture-plasty for tubercular stricture of the gastrointestinal tract
Br. J. Surg. 1977; 64:496-8.

77. Kapoor V.K., Kriplani T.K., Challopadhyay T.K., Sharma L.K.
Tuberculous perforations of the small intestines
Ind. J. Tuberculosis 1986; 33:188-9.

78. Gilinisky N.H., Voigt M.D., Bass D.H., Marks I.N.
Tuberculous perforation of the bowel. A report of 8 cases
S. Afri. Med. J. 1986; 70:44-6.

79. Lavis E.A., Abioje A.A.
Tuberculosis in the abdomen in Ibadaan: A clinicopathological review
Tubercle 1975; 56:144-55.

APPENDIX 1.

DATA COLLECTION FORM

A. **DEMOGRAPHIC**

Hospital number.....

1. Study number ☐☐
2. Age (years) ☐☐
3. Sex (male=1, female=2) ☐
4. Occupation (specify).....
5. Tribe.....

B. **HISTORY.**

Complaints (code, 0=N0, 1=Yes).

1. Anorexia ☐
2. Weight loss ☐
3. Abdominal distension ☐
4. Fever ☐
5. Night sweats ☐
6. Lethargy ☐
7. Other (specify).....

C. **PRESENTATION.**

1=Acute abdominal condition
2=Chronic abdominal condition ☐

D. **IF ACUTE ABDOMINAL CONDITION**

1=Intestinal obstruction
2=Peritonitis
3=Acute mesenteric lymphadenitis ☐
4=Acute tubercular appendicitis
5=Others (specify).....

E. **IF CHRONIC ABDOMINAL CONDITION]**

Presentation (Code 0=Absent, 1=Present).

1. Abdominal mass ☐
2. Ascites ☐
3. Chronic diarrhoea(malabsorption) ☐
4. Chronic obstruction(adhesions) ☐
5. Rectal distension ☐
6. Others (specify).....

F. **LABORATORY INVESTIGATIONS**

i. **HAEMOGRAM**

1. Haemoglobin gm%.....
2. Red cell count No^{12}/l.....
3. WBC x 10^9/l.....
4. Lymphocyte count 10^9/l.....
5. ESR Erythrocyte Sedimentation Rate MM/hr....

ii 1. Tuberculin test - mm.....

2. FNAC - ZN Staining
(code 0=Negative, 1=positive) ☐

iii. **ASCITIC FLUID**

Features (Code 0=Absent, 1=Present).

1. Straw coloured ☐
2. Protein level > 30gm/l ☐
3. Cell count > 1000/ul
Predominantly lymphocytes ☐
4. Others (specify).....

iv Elisa test for HIV.....

V. a. Total serum protein.....

b. Serum albumin.....

vi. **BIOPSY RESULTS**

Features (Code 0=Absent, 1=Present).

1. Histologically epithelioid cells with central caseation ☐
2. A.F.Bs seen on ZN staining ☐
3. A.F.Bs on culture ☐

G. **IMAGING RADIOLOGICAL INVESTIGATIONS**

1. (PLAIN ABDOMINAL X-RAY)

Features (Code 0=Absent, 1=Present).

1. Dilated intestinal loops ☐
2. Air fluid levels ☐
3. Calcified lymphnodes ☐
4. Ascites ☐

2. (BARIUM ENEMA)

Features (Code 0=Absent, 1=Present).

1. Mucosal irregularity ☐
2. Dilated loops and stricture ☐
3. Displaced loops(enlarged lymphnodes ☐
4. Adherent fixed loops (Adhesive peritoneal disease) ☐

3. (ABDOMINAL ULTRASOUND)

Features (Code 0=Absent, 1=Present).

1. Ascites ☐
2. Enlarged lymphnodes ☐
3. Hypertrophic intestinal lesions ☐
4. Others (specify).....

4. (CT SCAN)

Features (Code 0=Absent, 1=Present).

1. Adherent bowel loops ☐
2. Thickened omentum with irregular soft tissue density ☐
3. Caseated lymphnodes (low density centre with high density rim) ☐
4. Others (specify).....

5. (ENDOSCOPIC APPEARANCE - COLONSCOPY)

Features (Code 0=Absent, 1=Present).

1. Hyperaemic nodular friable mucosa ☐
2. Irregular ulcers with sharply defined margins & undermined edges ☐
3. Pseudopolyps and cobblestonning ☐
4. Others (specify).....

6. Important laparotomy findings

1. Ascitic fluid ☐
2. Pus in the peritoneal cavity
3. Thickened peritonium + omentum ☐
4. Tuberculous nodules all over the ☐
5. peritoneal cavity ☐
6. Intestinal perforations ☐
7. Widespread adhesions of intestines ☐
8. Abdominal masses ☐
9. Dilated intestines ☐
10. Strictures of the intestines ☐
11. Others ☐