DISCLAIMER

Portions of this document may be illegible in electronic image products. Images are produced from the best available original document.
CONTENTS

a) Dedications...........................................................................................................(i)
b) Acknowledgements.........................................................................................(ii)
c) Declaration........................................................................................................(iii)
d) List of abbreviations........................................................................................(iv)
e) Abstract............................................................................................................(v)
f) Summary (Arabic) .............................................................................................(vi)

Chapter I: Introduction:

a) Rationale........................................................................................................... 1
b) History of tuberculosis in Sudan.......................................................................... 3
c) Literature review.............................................................................................. 6
   (i) The pathogen.............................................................................................. 6
   (ii) Transmission and pathogenesis................................................................. 6
   (iii) Immunology............................................................................................. 9
   (iv) Pathology................................................................................................. 11
   (v) Clinical presentation.................................................................................. 14
   (vi) Primary pulmonary TB............................................................................. 16
       Complications.............................................................................................. 17
   (vii) Post primary TB...................................................................................... 21
         Clinical features....................................................................................... 21
         Physical signs........................................................................................... 22
         Complications.......................................................................................... 23
   (viii) Tuberculosis, AIDS & diabetes mellitus.................................................... 26
   (ix) Diagnosis of pulmonary TB.......................................................................... 29
      a) Smear examination................................................................................... 29
      b) Tuberculin sensitivity.............................................................................. 33
      c) Role of radiology...................................................................................... 35
         Radiological features of pulmonary TB..................................................... 39
         Primary TB............................................................................................... 40
         Post primary TB....................................................................................... 42
         Radiographic pattern in patients with AIDS............................................. 48
         Radiological classification of pulmonary TB.......................................... 49
      d) Other radiological investigations.............................................................. 51
      e) Blood investigations.................................................................................. 52
      f) Fibroptic bronchoscopy............................................................................ 53
   (x) Differential diagnosis of pulmonary TB....................................................... 54
   (xi) Control and management........................................................................... 57
Chapter II: Objectives ........................................................................................................59

Chapter III: Methods & Design ......................................................................................62

Chapter IV: Results .........................................................................................................( I- XXVIII )
   a) Tables and graphs. .................................................................................................
   b) Illustrations. ...........................................................................................................

Chapter V: Discussion ....................................................................................................63
   a) Epidemiology:
      Age & sex ...........................................................................................................63
      Residence ..............................................................................................................64
      Origin ....................................................................................................................64
      Occupation ..........................................................................................................65
   b) Clinical:
      Presenting complaints .......................................................................................65
      Duration of symptoms .......................................................................................67
      Social habits .........................................................................................................67
      Physical signs .....................................................................................................69
   c) Investigations:
      Sputum ...............................................................................................................72
      Mantoux test .........................................................................................................72
      ESR .......................................................................................................................73
      Chest x-rays .......................................................................................................73

Chapter VI: Conclusions & recommendations ................................................................80

Chapter VII: References ................................................................................................84

Appendages ....................................................................................................................92
   a) Questionaire
   b) X-ray illustrations
DEDICATIONS

To:

My Honoured Late Father,
My Beloved mother,
My Caring Husband,
& My Darling Childern.
ACKNOWLEDGEMENTS

I am indebted to all those who, directly or indirectly, have made it possible for me to write this thesis. I would like to express my deep gratitude to Prof. A/Rahman Ellider, Dr. Asma Elsoni & Dr. Hamad Ali Elturabi for their encouraging supervision & guidance of this thesis. Grateful thanks are due to Prof. M. Elseed for reviewing the X-rays. I would like to acknowledge the help of Dr. A.M. Zaki for his assistance in tuberculosis clinic at Abu Anja Hospital. I am especially indebted to my husband Dr. Khalafalla for his forebearance & support during this tedious work. My heartily gratitude to Mr. Siddig Dongola for the computer facilities that he provided. Special thanks are due to my family & my husband's family who were of great help during the whole study period. Finally, my thanks are extended to Misses lltaf & Hanaa' for their great secretarial help.
DECLARATION

I hereby declare that all this work has been carried out by myself & has not been published before.
**LIST OF ABBREVIATIONS**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>TB</td>
<td>tuberculosis</td>
</tr>
<tr>
<td>RH</td>
<td>right hilar</td>
</tr>
<tr>
<td>RPT</td>
<td>right paratracheal</td>
</tr>
<tr>
<td>LH</td>
<td>left hilar</td>
</tr>
<tr>
<td>LPT</td>
<td>left paratracheal</td>
</tr>
<tr>
<td>No</td>
<td>number</td>
</tr>
<tr>
<td>ZN</td>
<td>Zeihl-Nelson</td>
</tr>
<tr>
<td>AAFB</td>
<td>acid-alcohol fast bacilli</td>
</tr>
<tr>
<td>LN</td>
<td>lymph node</td>
</tr>
</tbody>
</table>
ABSTRACT

160 adult tuberculous patients were selected randomly from Al Shaab & Abu Anja hospital, to: (1) study the clinico-radiological pattern of the disease, (2) to determine the percentage of radiologically-positive patients & (3) to study the frequency of the initial presenting symptoms & to correlate them with their x-ray findings.

A flow-sheet was filled & proper clinical examination was conducted for each patient. Tuberculin test, 3 sputa examinations, ESR & chest x-ray were done for every patient. Three quarters of the patients were males in the young age group & most of the patients were of low socioeconomic status.

The main presenting symptoms were productive cough, chest pain, dyspnoea, fever, weight loss and malaise. Patients presented rather late, with a mean period of four months.

57% of patients were sputum positive, 80% Tuberculin-positive and 98% had a high erythrocyte sedimentation rate (ESR).

15% of patients had radiological features of primary diseases of which hilar lymphadenopathy was the commonest, while consolidation was the commonest parenchymal lesion. In post-primary disease fibrocavitary type was the commonest and together with the exudative lesions constituted 98% of parenchymal lesions.

90% of patients had typical upper or middle zone infiltrates and 10% had lower lung field tuberculosis.

14% of patients had pleural effusion.

60% of patients had moderate disease extent in the chest, 30% far-advanced and 10% minimal lesions in their chest x-rays.
CHAPTER I:

INTRODUCTION
Rationale

Tuberculosis is a disease which is associated with high morbidity and mortality. It has been found in the vertebrae of Neolithic men in Europe and of Egyptian mummies perhaps as early as 3700 BC\textsuperscript{10}. Till now it remains the world's leading cause of death from a single infectious disease\textsuperscript{2}. Today it has become the most important communicable disease in the world with a total of 4-5 million highly infectious cases of pulmonary tuberculosis occurring each year, and at least an equal number of less infectious cases\textsuperscript{3}\textsuperscript{a}. More than three quarters of the cases are in the developing countries\textsuperscript{4}\textsuperscript{b}. It continues to be a major health problem in the Sudan and has been declared by the government as an urgent priority for control\textsuperscript{5}\textsuperscript{a}. The best indicator of the extent of the tuberculosis problem is the average annual risk of infection (ARI) which is the proportion of the population that is likely to be newly infected over a period of one year. ARI in 1986 was 1.8\% in Sudan\textsuperscript{6}\textsuperscript{a}, while in 1992 it was 1.9\%\textsuperscript{5}\textsuperscript{a}. The hospital case fatality rate was 10\%. These statistics does not include the Southern States, where no data were available due to insecure conditions.

The magnitude of the problem has been further complicated by the problems of war\textsuperscript{5}\textsuperscript{a}, displacement and influx of refugees from neighbouring countries, and the emergence of AIDS.

Pulmonary type accounts for about 90.0\% of all cases of reported tuberculosis till 1990 in Sudan, and it is through this type that most of the disease is being spread.

Tuberculosis patients may present with protean clinical manifestations and its diagnosis may prove quite difficult sometimes. Nevertheless one should be cautious before labelling a patient as being tuberculous, bearing in mind the prolonged expensive management for an already poor person, and the social stigma haunting the patient and his family for the rest of their lives. But, since it is a curable disease, accurate diagnosis is mandatory.
The definitive diagnosis of pulmonary tuberculosis lies in the isolation of the tubercle bacilli from the patient's sputum, either by direct microscopy or by culture. But the yield of sputum positive cases ranges between 40 - 80%. Patients with strong clinical suspicion and negative sputum pose a diagnostic problem. Other investigations may help, such as radiology which carries the lion's share, and has been used as an important adjuvant to the diagnosis of tuberculosis, and in mass surveys for detecting disguised cases, although this latter trend is now declining in the developed world. It is also indispensable in showing the distribution, classification, stage, type (primary or post primary), and extent of the disease. It is also important in assessing progress and monitoring response to chemotherapy. Radiology has also been important in the accidental finding of asymptomatic patients, where its yield in tuberculosis detection rate was up to 21% of sputum positive cases. This is specially important in immunocompromised patients.

As mentioned above, only a few studies have been conducted in Sudan concerning tuberculosis inspite of the abundance of the study material.
**History of Tuberculosis in the Sudan**

In the nineteenth century, observations on Tb in Africa were made by a number of laymen as well as doctors, but they were chiefly based on clinical records or recollections of individuals. Pruner (1884) talks of the disease as becoming rarer in proportion as we proceed southwards from the shore of the Mediterranean. In central and upper Egypt it is decidedly uncommon, but increases in Khartoum and Sennar.

It was only at the beginning of this century that serious and critical attention was paid to the problem (Wilcock's, 1962). Von Becker (1904) called attention to the small incidence of Tb amongst Egyptians in Egypt in contrast to the intense susceptibility to the disease shown by the Berbarians (from Berber, the capital of the Northern Province in the Sudan) when they were subjected to Egyptian living conditions. Smith (1909) observed that "The Sudanese soldier's liability to the disease is accompanied by an almost complete absence of it amongst the Sudanese tribes in their natural surroundings, and by a corresponding absence of tuberculosis in the herds of the Sudanese cattle-owning tribes.

Cummius (1912) realized a higher incidence of Tb among Sudanese soldiers as compared to Egyptian soldiers and formulated the theory of virgin soil. This theory had two parts, firstly that primitive tribes are highly susceptible to Tb because in the absence of the tubercle bacillus they have not been obliged to protect themselves against it, and secondly that people living in more commercial or industrial communities are highly protected against Tb because of being in regular contact with the bacillus they have developed protective immunity against it. This theory holds good up to the present day.

Archibald (1922) wrote that there was no doubt that in the Sudan the disease was practically confined to the large towns of the North. But this is no longer the case; with the development of the country and the continuous movement of people the disease is spreading (Fig I).
Studies on infectivity of Tb were conducted in various provinces of the Sudan between 1918 and 1930. Dongola showed the highest infectivity rate of 4.3% and Khartoum province showed almost 3%. 50% of all patients in Khartoum came from other parts of the Sudan, mostly from the Northern Province, and they came to Khartoum seeking treatment which was not available elsewhere.

In Southern Sudan, Tb showed very low incidence before the 1930s [0.2/10000].

In 1932 a total of 421 pulmonary Tb cases were admitted compared to only 260 cases in 1928 and only 140 cases in 1922, with pulmonary infection forming 57% of cases and extrapulmonary infection forming 43%.

In 1940, hospital admissions reached 1036 cases with 56% pulmonary and 44% extrapulmonary, and with the start of the second world war a rapid increase in cases mounted to 1600 cases admitted in 1945 with 60% pulmonary and 40% extrapulmonary.

After the war Tb became one of the most important health problems in the Sudan.

Historically, two prevalence surveys have been conducted in the Sudan. The first was in 1954/1955, and the second 1959/1960. Both were technically and financially assisted by the WHO. The second survey studied only the prevalence of pulmonary Tb in the Blue Nile Province. The overall provincial prevalence was 26.0%.

A tuberculin survey was carried out in 1976 and again in 1986 in children 0-14 years old in Khartoum and in the Central, Northern, Eastern and Kordofan regions. There are no recent data for the Southern regions of Darfur.

In 1976, 3407 subjects of 10.5 years average age were tested and an ARI of 1.8% was found.
In the 10-year interval, the extent of the Tb problem seems substantially unchanged in the sampled areas.

Zaki AM\(^{(39)}\) has recently conducted a community-based study on the Red Sea area, and found an ARI of 3.4% in that region.

Some hospital-based studies were performed in Khartoum in two teaching hospitals in the paediatric wards. They described the general Tb pattern in Sudanese children.\(^{(4)}\) Furthermore some MD studies are being currently conducted in paediatric TB, TB with AIDS, and Tb with diabetes, and results are to be expected.
Route of entry

- 3.7/1000 (TB incidence 1930)
- 2.5% (TB prevalence 1960)
- 50-MA/100,000 people (1980-1994)

Fig. 3.1 History of tuberculosis in the Sudan
The Pathogen

Mycobacteria are aerobic, non-motile non-spore forming rods. They are 2-4um in length and 0.3um in thickness. They live usually best at PO$_2$ of 14.0 mmHg. There are several species of the genus Mycobacterium which may cause disease to the lung or pleura, and by far the most important of these is Mycobacterium tuberculosis, which is responsible for 95-99% of pulmonary mycobacterial infections. Mycobacterium bovis, formerly a common cause of lung disease has declined in prevalence in the industrial countries, but is still prevalent in countries where milk hygiene is still not adequate. Other species like M.kansii and M.avium also cause disease in man very similar to M.tuberculosis and can only be differentiated on culture. Enrichment of the ordinary culture media by potato, eggs, bacon or albumin is required to produce growth on culture. Growth is slow, requiring 2-8 weeks. Colonies are opaque white to cream, and have a wrinkled surface. M.tuberculosis is highly pathogenic for guinea pigs. Tubercle bacilli are difficult to stain, but once stained they strongly retain the dye which is not removed by acid-alcohol solutions. This acid and alcohol fastness can be demonstrated by the Ziehl-Neilsen staining procedure of which there are various modifications. The tubercle bacillus is susceptible to heat, sunlight and U/V radiation, but is fairly resistant to drying and some disinfectants.

Transmission and Pathogenesis

The source of TB infection is the diseased man, in particular the patient with pulmonary disease. The infectiousness of a patient is determined by his bacillary status: patients in whom tubercle bacilli can be demonstrated in the sputum by direct smear examination are highly infectious, whereas in contrast, patients in whom
tubercle bacilli cannot be demonstrated in the sputum by culture only, or who are culture negative are relatively non-infectious. It is known that infection is transmitted by the airborne route, and that the unit of infection is a small particle called a droplet nucleus, which consists of a single bacillus, and the smaller the droplet the more liable it is to deposit in alveolar surfaces, while the larger particles are trapped in larger airways and cleared by mucociliary function.

Not only the size of a droplet but also the dose of the infecting organisms inhaled by someone affects the liability of his developing the disease. Coughing, sneezing, spitting and other respiratory manoeuvres will generate droplet nuclei, due to evaporation of small respiratory droplets. These droplets are dispersed throughout space without settling, and the organisms which they contain can remain viable for extended periods of time. They are neither filtered by simple gauze masks nor prevented from getting out by covering the mouth and nose during coughing.

From these, it is seen that prolonged contact with a highly infectious case is necessary before infection is acquired. At the other extreme, infection may be acquired by a single exposure, for example in laboratories or post-mortem rooms. The above mentioned factors govern the transmission but there are other complex host factors which play a major role in the patient developing the disease, namely:

1. Age and Sex. There is little difference between boys and girls up to puberty. Up to the age of 2, infection is particularly liable to result in the most fatal forms, miliary TB and tuberculous meningitis due to blood stream spread. After one year of age and before puberty, an infected child may develop miliary TB or tuberculous meningitis or one of the more chronic disseminated types of TB, particularly lymph node, bone or joint disease. Before puberty the lung part of the primary lesion usually just affects that focal area. In Europe and North America when TB was common the peak incidence of pulmonary TB was usually in young adults. The
male rate continued fairly high at all ages, but the female rate tended to drop rapidly after the child bearing years. Women often developed pulmonary TB following child birth. In Africa and India the pattern seems to be somewhat different. The prevalence of pulmonary TB seems to increase with age in both sexes. In women the overall prevalence is lower and the rise with age is less steep than in men. In women it reaches a maximum at the age of 40-50 and then falls. In men it goes on rising at least to age 60.

2. **Nutrition.** This is a very good evidence that starvation or malnutrition reduces resistance to the disease. This is a very important factor in poorer communities both in adults and in children. Trendal has shown an association between development of pulmonary TB and body mass index.

3. **Standard of Living.** The prevalence of TB diminishes as social and economic conditions improve. Poor housing with associated overcrowding increases the risk of massive infection or reinfection if one of the occupants suffers from infectious TB. Some studies have shown increased incidence of TB amongst prisoners and common hostel dwellers.

4. **Toxic Factors.** Tobacco smoking and high alcohol intake are important in reducing body defences, as well as corticosteroid drugs and other immunosuppressants used for treating certain diseases.

5. **Other Diseases.** Certain diseases were found to increase the susceptibility to TB, by far the most important of which is AIDS. Others like duodenal ulcer, leukaemia, leprosy, DM, gastrectomy and silicosis were shown also to have a significance. In the tropics chronic malaria and worm infestations may be additional factors. Miliary TB may follow whooping cough, measles or HIV infection.
6. **Endogenous Host Factors.** There is good evidence that isolated populations, for instance Innuits (Eskimos) or American Indians, when they met the disease for the first time they had poor defences and TB spread very rapidly and caused high mortality. In Europe or China where TB had been common for many years, those with congenitally poor defences had probably died early, often before they had any children. The survivors had more natural resistance to the disease. But in populations where the disease was new affected people often died within a few months from the so-called galloping consumption. A study in India has shown an increase in level of HLA DR2 in patients with pulmonary TB more than the control group.

7. **Occupation.** Certain occupations predispose to the development of disease either by increasing exposure to infection, such as hospital workers and mortuary attendants, or by damage to the lungs caused by inhalation of harmful dusts in mining and other dangerous trades.

**Immunology**

The alveolar macrophage and circulating monocytes are the key cells in TB immunity. A network of interactions among the T-lymphocytes, B-lymphocyte and macrophage modulates the host defence. When a tubercle bacillus passes into the distal air spaces of the lung of a previously infected person it is engulfed by a macrophage and enclosed within a phagocytic vacuole (phagosome). The phagosome may fuse with a lysosome, causing the tubercle bacillus to be killed by proteolytic enzymes. With or without intracellular killing the macrophage presents mycobacterial antigens to T-lymphocytes and secretes substances which result in lasting changes in lymphocytes. Activated T-lymphocytes produce several mediators
including macrophage-activating, chemotactic migration inhibitory, and blastogenic factors. The ability of activated macrophages to kill tubercle bacilli is greatly increased. Some of the macrophages change into epithelioid or multinucleated giant cells which are capable of killing ingested tubercle bacilli. Proteolytic enzymes released by the macrophages, epithelioid cells and giant cells produce a type of tissue necrosis referred to as caseation. In presence of a large number of bacilli the necrotic tissue may liquefy, enabling the infection to spread to other parts of the lung.

Apparently tubercle bacilli may survive within activated macrophages for prolonged periods ("persistors") where they divide only occasionally. The immunity transferred by an initial infection is today utilized in the form of vaccination with BCG (Bacille Calmette-Guerin). BCG is a virulent strain of the \textit{M. bovis} initially attentuated by cultivation on a potato-glycerol-bile medium for 230 serial transfers, thus achieving immunogenicity without pathogenicity. BCG confers immunity by activation of macrophages within the reticulo-endothelial organs of the immunized host with resultant limitation of mycobacterial growth on subsequent challenge. The immune host, (whether protected by previous infection or BCG) is not protected from subsequent infection, but in such a host the rate of growth of \textit{M. tuberculosis} is slowed compared to that seen in non-immunized controls.

Acquired immunity is mediated by T-cells which reach the tubercle from the spleen and lymph nodes. On contact with antigen processed by macrophages in the tubercle, lymphokines are released which activate blood-derived monocytes entering the lesion. These activated macrophages undergo structural, enzymatic and metabolic changes and phagocytose and kill tubercle bacilli at a markedly enhanced rate compared with unstimulated cells.
Pathology

In the non-immune subject, tubercle bacilli can gain entrance to the body by several routes: lung, GIT, tonsils, direct cutaneous or percutaneous inoculation (as may occur accidentally at the autopsy table). For practical purposes the most important route is the lung. The majority of the lesions in the early phase are the lower two-thirds of the lung where ventilation is best and deposition of droplet nuclei more likely. Deposition of tubercle bacilli in the alveoli of the lung is followed by vasodilatation and an influx of polymorphonuclear leucocytes and macrophages to the area. After phagocytosis in the non-immune host, the bacilli may remain viable or multiply occasionally within macrophages for an extended period. After several weeks the polymorph numbers diminish and the macrophage predominates. The macrophages develop pale foamy cytoplasm rich in lipid and crowd together. After 2-3 weeks fibroblasts, lymphocytes and plasma cells dispose themselves around the circumference of the lesion which in the centre appears one or two giant Langhan's cells which are multinucleated cells formed by fusion of mononuclear cells. These exceed 100um in diameter and they contain many dark-staining nuclei round their periphery. This lesion now constitutes the microscopic tubercle, the hallmark of tuberculosis infection.

Further growth is by extension or by fusion with neighbouring lesions until a macroscopic tubercle is formed. Its subsequent fate depends on many factors. The centre undergoes caseation necrosis when it follows one of three courses: it may rupture into one of the air passages, discharges its contents and leaves a cavity; it may become encircled by fibrous tissues and undergo healing with calcification; or it may remain dormant, containing virulent mycobacteria and be stirred to renewed activity years later.

Primary infection is usually evident as a subpleural tubercle (the Primary or Ghon focus) which may be in any lung zone and which drains via lymphatics to hilar lymph nodes to form the primary complex (Ghon's complex). Most primary infections heal with or without calcification of the primary complex, but haematogenous spread
probably occurs via lymphatics in the majority of infected subjects, resulting in the seeding of tubercle bacilli to other parts of the lung as well as other organs. The primary lesion may occasionally progress into a massive exudative type, or it may progress to a lesion similar to that seen in the reinfection type. Less commonly the primary infection is in the tonsils or the intestine with lymphadenitis in the cervical or mesenteric nodes. This occurs when organisms are swallowed, usually in infected milk. The infection is usually contained at extrapulmonary sites as it is in the lung, but the potential for reactivation of infection at all sites is always present. Once the primary lesion has healed, the patient will have acquired hypersensitivity to tuberculosis protein and subsequent infection, which is often termed "post-primary" or "reinfection" will follow a course modified by this fact. Reactivated pulmonary tuberculosis is most often seen in the upper lung zones and is limited in extent most frequently to the posterior segment of the upper lobe or the apex of the lower lobe. The high V/Q ratio with alveolar PO\textsubscript{2} elevated relative to other zones is believed to predispose to reactivation at these sites. Proliferation of tubercle bacilli in the caseous centres is followed by softening and liquefaction of the caseous material which may discharge into a bronchus with resultant cavity formation. Whereas 1x10\textsuperscript{4} bacilli per gram are found in caseous tissues, up to 1x10\textsuperscript{9} organisms may be harboured within a single cavitory lesion\textsuperscript{(12)}. Fibrous tissue forms around the periphery of such tuberculous lesions but is usually incapable of limiting extension of the tuberculous process. Haemorrhage may result from extension of the caseous process into vessels within the cavity walls. Spread of caseous and liquefied material through the bronchial tree may disseminate infection to other lung zones with or without development of vigorous inflammatory exudate or tuberculous pneumonia. Rupture of a caseous pulmonary focus into a blood vessel may result in miliary (Latin: milia=seed) tuberculosis with the formation of multiple 0.5-2.0 mm tuberculosis foci in the lung and in other organs of the body. Encroachment on bronchi of pulmonary or lymph
node caseous material may give rise to **tuberculous bronchitis**. Rupture of caseous glands into the trachea or major bronchi causes **collapse** of the lung or even **sudden death** by suffocation in young children.

**Spread of infection in the body**

*M.tuberculosis* is generally an intracellular parasite, although it is capable of extracellular multiplication in pulmonary cavities and similar situations. It commonly spreads by lymphatic dissemination, cells containing tubercle bacilli are carried by lymph vessels to regional lymph nodes which then become the site of active infection. Chains of lymph nodes may be affected in this fashion and material containing tubercle bacilli may enter the thoracic duct and thus the blood stream. Renal tuberculosis or spondylitis complicating pleurisy are often caused by spread along lymph vessels with involvement of paravertebral nodes. Infection may also spread by simple extension to contiguous surfaces.

The third route of spread is by the blood stream itself. Before hypersensitivity develops as a few tubercle bacilli may be discharged into the blood stream either directly from the primary focus or by way of regional lymph nodes and the thoracic duct. Some organisms may die without creating foci; others may remain viable in quiescent lesions and may produce active disease even after many years. Erosion of a blood vessel by a caseating lymph node or other lesions may release large numbers of organisms into the blood stream and give rise to miliary tuberculosis. Repeated seedings produce metastases of varying size manifesting as panadenitis, polyserositis, and osteomyelitis; the spleen is extensively affected. Finally, dissemination may occur by way of the bronchi, alimentary tract or urinary passages.

In the first instance, the contents of a tuberculous cavity may leak into a bronchus and be aspirated into another part of the lung causing tuberculous bronchopneumonia. In the second, infected sputum may cause laryngeal tuberculosis or may be swallowed and give rise to tuberculous enteritis. In the third, bacilli discharged from a tuberculous kidney may infect the ureter and the bladder.
CLINICAL PRESENTATION

The time table of Tuberculosis:

Tuberculin hyper-sensitivity usually follows within 3-4 weeks or at most 6-8 weeks after infection. The time of infection can therefore sometimes be dated quite precisely by the demonstration of tuberculin conversion by repeated tuberculin testing or sometimes by the spontaneous appearance of erythema nodosum or phlectanular conjunctivitis which may denote the onset of tuberculin hyper sensitivity. A phlycten is a small (4-5mm) pearly module usually at the junction of the conjunctiva & cornea with a leash of blood vessels leading to it from the periphery. Rarely this lesion may recur or may ulcerate even more rarely it may lead to ocular destruction.

Tuberculin conversion or the appearance of erythema nodosum together with the demonstration of a positive tuberculin reaction followed by a long period of observation enabled Wallgren to construct a timetable of tuberculosis.

Miliary TB & tuberculous meningitis which is frequently associated with the former occur usually within six months of the primary infection & are especially common in children under five years. Meningitis is sometimes a terminal event in cryptic miliary disease in the elderly. Pleural effusion, presumably due to seeding of the pleura from a lung focus, also occurs early, usually within 6 - 12 months, and is commoner in young adults and unusual in small children. In young adults the primary disease can progress with increasing infiltration & cavitation evident 1-2 years or even later after the primary infection. This type of disease is labelled progressive primary or post primary disease and occurs most commonly at puberty. Also 1-5 years after the primary infection, skeletal tuberculous lesions, most commonly of the spine, may present. Lesion of the genito-urinary tract & the skin make their appearance much later, commonly 5-15 years after the primary infection. The most common type of the disease seen in developed countries today, post primary disease of late middle age or the elderly is due to reactivation of apical pulmonary foci which where seeded at the time of the primary infection, have lain dormant, and then present with progressive
cavitating pulmonary disease many decades after the initial primary infection. Finally, cryptic miliary tuberculosis or anergic tuberculosis of the elderly is an extreme variant of reactivation tuberculosis which also presents late in life and may be associated with an entirely normal chest radiograph\(^\text{12}\).

Further data based on the British Medical Research Council's tuberculosis vaccines trial, were in keeping with Wallgren's timetable.\(^\text{13}\)
Tuberculin test becomes positive. Minority of those infected experience febrile illness and erythema nodosum.

Miliary and meningeal tuberculosis common in children under 5 years; pleural effusion rare in children. Usually within 6-12 months after primary infection.

Adult (post-primary) disease and skeletal disease commonly occurs 1-5 years later.

Genito-urinary and skin lesions are late manifestations - after 5-15 years.

Fig. 13.3. Natural history of untreated primary tuberculosis — the timetable of tuberculosis. See text for explanation.
CLINICAL FEATURES:

Primary pulmonary tuberculosis:

The first infection with the tubercle bacillus is known as primary tuberculosis and usually includes involvement of the draining lymph nodes in addition to the initial lesion. The combination of the primary or Ghon focus and the draining lymph nodes is known as the primary complex. All other tuberculous lesion are regarded as post primary and are not accompanied by major involvement of the draining lymph nodes.

Although primary tuberculosis may occur in the intestine, tonsils or various other unusual sites, in the great majority of cases, the route of infection is by inhalation and consequently the primary lesion is pulmonary.

The great majority of primary TB infections are probably symptomless, at least in young adults and adolescents, the infection being overcome without the individual being aware of it. A proportion may experience a brief febrile illness at the time of tuberculin conversions which is indistinguishable from the many febrile illnesses of childhood. Occasionally, typical primary tuberculosis may occur in elderly people who have lost their tuberculin sensitivity.

When symptoms are present they are likely to be of a general nature with failure to thrive as the outstanding feature. In older children there may be pyrexia and a cough or sensitization reactions such as erythema nodosum and Phlectanular conjunctivitis may draw attention to the underlying disease. Primary infection in adults occurs most commonly between the ages of 16 - 30 years. Glandular component of the primary complex predominates in young children and the air passages are particularly vulnerable to compression by enlarged lymph nodes or obstruction by swollen mucosa or viscid sputum. Wheezes and cough paroxysms are common, but sputum production is rare in children.
In most cases there are no detectable physical signs. With severe disease the child may appear unwell, fretful and debilitated. Auscultation of the chest is usually unrewarding but occasionally crepitations may be heard over an extensive primary focus and wheezes from pressure of glands on bronchi. More extensive physical signs may be expected if complications occur. Complications may occur anywhere within the body and protean clinical manifestations may occur.

But important symptoms and signs that should arouse suspicion of TB in children are:

1- Failed to gain weight or lost weight for more than 4 weeks.
2- Lost energy and possibly some weight over.
3- Wheeze or cough +/- 1&2 above
4- Fever for more than one week.
5- Any of the above + signs of pleural effusion.
6- A swollen abdomen especially if associated with any lumps.
7- Chronic diarrhoea (white stools) especially if not responding to the treatment for giardiasis and worms.
8- A limp on walking or a stiff spine.
9- Spinal lump with or without stiffness on walking.
10- Swelling of a big joint not due to injury.
11- Lymph node enlargement.
12- Cold abscesses.
13- Discharging sinuses.
14- Headache and irritability and occasional vomiting.
15- Slow onset of weakness of one arm or leg or one side of the face.

**Complications of Primary TB:**

**GENERALIZED TUBERCULOUS INFECTION:**

Generalized infection arises through the discharge of infected material from a caseating lesion into the circulation. The lesion in question is often a lymph node which becomes adherent to a vein, finally eroding its wall and pouring its contents
into the blood stream. The picture varies with the site of lesion from which dissemination occurs; when a systemic vein receives the discharge of tuberculous material the lungs may be affected almost exclusively; when a pulmonary vein, the infection is mainly systemic. All intermediate gradations occur. The acuteness of the process also shows variations. The common form is generalized miliary TB in which the bacilli disseminate through the blood stream give rise to innumerable discrete tubercles scattered throughout the lungs and other tissue (so called because they are about the size of a millet seed 2mm).

The classic form is seen most frequently below 10 years of age. Onset may be abrupt or gradual. Initially there is vague ill health, fatigue, exhaustion, headache, anorexia, loss of weight and fever. Anaemia appears early and may be misleading of the problem as a haematological one. Irritability in children esp. after known primary infection or PUO in elderly should arouse suspicion.

In about 90% of patients with miliary disease, TUBERCULOUS MENINGITIS develops. In the sub acute type local lesions such as pleural or pericardial effusion, arthritis or cutaneous tuberculoids may appear in successive crops over weeks or months.

A part from fever, there may be no physical signs and in particular the chest is frequently normal on auscultation although creps may develop in the later stages. Hepatomegaly, nuchal rigidity, lymphadenopathy, & splenomegaly may be found in a proportion of cases.(41,42)

Choroidal tubercles are found in over 90% of children with miliary TB but less commonly in adults. (43,44) Generalized TB was always fatal before the specific antibacterial agents were available, but now the outlook is better.

Cryptic miliary or disseminated TB:

The cryptic form of disseminated disease is increasingly being seen in the elderly where it may be difficult to diagnose since the chest film may be normal, Choroidal tubercles are absent and the tuberculin test may be negative. The most common presentation is with weight loss, malaise and PUO. Anaemia is usual and ESR is
elevated. A variety of blood dyscariasis and abnormal liver function tests and serum salts have been seen.

LUNG COMPLICATIONS

[A] Pleural Effusion:-
We have seen that the primary focus forms just below the surface of the lung. Most do not become larger than 10mm. Sometimes the focus does get bigger still. Then the surface of the lung may rupture allowing caseous material and sometimes bacilli to leak into the pleural space. The fluid of an effusion is usually absorbed without difficulty. But occasionally if many bacilli are present the fluid may become purulent and a tuberculous empyema is the result.

[B] Acute Cavitation of a Focus:-
When resistance is poor as in young or malnourished children, the primary focus may increase in size and instead of opening into the pleural space it opens into a small bronchus and the caseous material is discharged by coughing. Cavitary tuberculosis may result. Distention of the bronchi may lead to obstruction and acute suffocation by caseous material, or also spread to a lung segment or lobe may cause [C] ACUTE TUBERCULOUS PNEUMONIA, or later on sequestration of the bronchi especially beyond a segment with bronchial stenosis may lead later on to

[D] BRONCHIECTETIC CHANGES.

[E] LYMPH NODE ENLARGEMENT may produce several complications:-

1. The enlarged lymph node may compress on a segmental or lobar bronchus leading to atelectasis beyond the obstruction level, with the resultant radiological picture formerly known "EPI-TUBERCULOSIS". This radiographic appearances may be due to collapse, inflammatory exudation, caseous pneumonia or a combination.

2. The lymph node may obstruct a bronchus and act as a ball valve with air entering during inspiration and failing to exit during expiration leading to OBSTRUCTIVE EMPHYSEMA.
(3) The lymph node may empty its contents into a bronchus leading to inflammatory exudate filling that segment and causing **EXUDATE PNEUMONIA**. The site of erosion of the L.N through the wall may heal by fibrosis and scarring leading to **BRONCHOSTENOSIS**.

(4) Mediastinal lymph node may rupture into the pericardium causing **TB PERICARDITIS**, or rarely into the oesophagus.

(F) Material being discharged through the bronchi may be swallowed and reinfect the gut leading to **INTESTINAL TUBERCULOSIS**, this is an alternative way to the other route of infection by bovine tuberculosis.

(G) Calcification in a primary focus, or more commonly in a lymph node may later be extruded into a bronchus as a "**BRONCHOLITH**" which may declare itself with haemoptysis such broncholiths may be seen through the bronchoscope, but are best left well alone.

(H) Primary infection may manifest itself as hypersensitivity reaction in the form of **phlectanular conjunctivitis** (as already mentioned) **erythema nodosum**, or **pleural effusion**.

The characteristic feature of erythema nodosum is the presence of tender, dusty-red slightly nodular lesions on the anterior surfaces of the legs, although lesions are occasionally also found in the anterior surfaces of the thighs, the extensor surfaces of the forearms and rarely on the face and breasts. The nodules are usually 5-20mm in diameter, have ill-defined margins and may become confluent. They usually resolve over a week or two, the red color fading to purple and then brown, the brownish pigment after persisting for several weeks. Recurrent crops of lesions may occur. Associated generalized accompaniments include, fever, high ESR and arthralgia and tuberculin test is virtually always strongly positive.
POST PRIMARY TUBERCULOSIS

CLINICAL FEATURES:

Post Primary infection may arise in different ways:

1. Direct progression of a primary lesion.
2. Reactivation of a quiescent primary or post primary lesion.
3. Haematogenous spread to the lungs.
4. Exogenous reinfection.

In the developed countries, the majority of patients are elderly, while in countries with high prevalence young adults are the ones mostly affected.

Symptoms are most frequently related to the respiratory system, cough being the commonest symptom. It is usually there for more than three weeks since the patients ignore it as being due to smoking or common cold. Sputum may be mucoid, purulent or blood stained.

Haemoptysis is the classical symptom of pulmonary TB and may vary from mere blood-staining of the sputum to torrential amounts of blood which occasionally may be fatal.

Chest Pain is common, it may be dull ache or worsen with breathing (pleuritic). Coughing may cause it and even progress to cough fracture.

Breathlessness is usually due to extensive pulmonary disease or to pleural effusion.

Occasionally the patient may have a localized wheeze. This is due to local tuberculous bronchitis or to pressure of a lymph node on a bronchus.

Sometimes the patient seems to have developed an acute pneumonia which does not respond to routine antibiotics. On close questioning, it is revealed that the patient had mild non-specific symptoms going on for sometime before the pneumonia develops.
Many patients may have no symptoms attributable to the respiratory system but may complain of **Generalized weakness** and **lethargy, malaise, loss of appetite** or **loss of weight**. In some advanced cases **Febrile symptoms** with classical **night sweats** may supervene.

On the other extreme, some patients are **symptomless** and discovered only on routine examination or X-rayed for other purposes.

In women, they may present with **amenorrhea** which is reversible on treatment.

**Physical Signs:-**

Physical examination may not help much. Useful signs are:

1. **General condition** - Sometimes this may be good, in spite of advanced disease. But the patient may look obviously **ill**. He may be very thin and looks emaciated with obvious **loss of weight**. He may be **pale** or have flush or looks sweaty from the **fever**.

2. **Fever** - This may be of any type, but they may have only a slight rise in the evenings. The temperature may be high or irregular, but most often, there is no fever.

3. **Pulse** - It is usually raised in proportion to the fever.

4. **Finger clubbing** - is found occasionally but is usually with extensive disease, especially in Africans.

5. **Chest Examination** - Often there are **no abnormal** signs. The commonest early sign is fine post tussive **crepitations** in the upper parts of one or both lungs. Later there may be **dullness** to percussion or even **bronchial breathing** in the upper parts of both lungs. Occasionally there is a **localized wheeze** due to local bronchitis or pressure by a L.N. on a bronchus. In chronic tuberculosis there may be signs of **fibrosis**. At any stage there may also be signs of **pleural effusion**.

The patient should also be examined for signs of disease else where and for other concomitant diseases.
(3) Pulse: - It is usually raised in proportion to the fever.

(4) Finger clubbing: - is found occasionally but is usually with extensive disease, especially in Africans.

(5) Chest Examination: - Often there are no abnormal signs. The commonest early sign is fine post tussive crepitations in the upper parts of one or both lungs. Later there may be dullness to percussion or even bronchial breathing in the upper parts of both lungs. Occasionally there is a localized wheeze due to local bronchitis or pressure by a L.N. on a bronchus. In chronic tuberculosis there may be signs of fibrosis. At any stage there may also be signs of pleural effusion.

The patient should also be examined for signs of disease else where and for other concomitant diseases.

COMPLICATIONS OF POST PRIMARY TUBERCULOSIS:

(1) Tuberculous Pleural Effusion and empyema:
The pleura may be affected in three different ways:
(i) Effusion which develops within a few month of primary infection in children and young adults.
(ii) Effusion developing as a result of lung disease in older adults rarely this may go to a purulent effusion (Empyema).
(iii) Rupture of a tuberculous cavity and escape of air into the pleural space. The TB from the ruptured cavity produce a purulent effusion (Empyema). The air and pus together constitute pyopneumothorax.
COMPLICATIONS OF POST PRIMARY TUBERCULOSIS:

(1) Tuberculous Pleural Effusion and empyema:
The pleura may be affected in three different ways:
(i) Effusion which develops within a few months of primary infection in children and young adults.
(ii) Effusion developing as a result of lung disease in older adults; rarely this may go to a purulent effusion (Empyema).
(iii) Rupture of a tuberculous cavity and escape of air into the pleural space. The TB from the ruptured cavity produces a purulent effusion (Empyema). The air and pus together constitute pyopneumothorax.

It shows clinically as:
(1) Pleuritic pain, which later becomes dull ache in the lower chest.
(2) Fever which may be mild and short lasting.
(3) Slight irritating cough.
(4) Breathlessness on exertion.
(5) Physical signs of effusion: dullness on percussion, no air entry sounds, pushing of the trachea and mediastinum.
(6) Mantoux is usually positive in young patients.
(7) There may be an abscess in the chest wall, if the effusion spreads through the chest wall.

[2] Spontaneous Pneumothorax:
This occurs when air escapes into the pleural space following rupture of a tuberculous cavity. This causes sudden acute chest pain of that side and breathlessness. Physical signs of pneumothorax if amount is large enough may be detected.
[3] **Tuberculous Laryngitis:**

1. The patient may have cough and sputum.
2. There may be hoarseness of voice, becoming a moist whisper.
3. Pain in the ear.
4. Pain on swallowing, which usually means the epiglottis is involved. The pain may be severe.
5. In very advanced disease, ulcer of the tongue and larynx may be shown.

[4] **Cor Pulmonale:**

(Congestive Cardiac Failure due to back pressure from damaged lungs):

This may occur if there is very extensive destruction of the lungs. This may happen even if the tuberculous disease is no longer active, but has left a lot of scarring.

[5] **Chronic Obstructive Airway Disease**.

6. Well treated and healed tuberculous cavity, sometimes remain open, and can be infected by the fungus *Asperigillus fumigatus*. This may cause severe and even fatal haemoptysis. If bleeding recurs, surgical removal may be the only solution.

(7) **Pulmonary gangrene.**

This is very rare but is usually fatal.

[8] **Bronchiectasis:**

Bronchiectatic changes may occur, but since the disease is mostly apical, there is good drainage and there may be no much disability to the patient, but problems become evident when there is lower lung field disease.

[9] **Respiratory Failure and Acute Respiratory Distress Syndrome:**

This may cause delay in the diagnosis of pulmonary tuberculosis.

[10] **Haemoptysis**:

This may occasionally be massive and life threatening.

[11] **Malignancy**:

A fibrotic scar in the lung may be later site for malignancy.
[12] Bronchopleural Fistula:
This is one of the most dreaded complications of pulmonary TB.

[13] Amyloidosis:
Now becoming rare and diagnosed by biopsy.

[14] Gastro-intestinal TB:
Although GIT system is generally resistant to penetration by tubercle bacilli, but in cavitary pulmonary TB associated with heavy secretions of bacilli, mucosa may be penetrated at the iliocaecal junction.
TUBERCULOSIS, AIDS & DIABETES MELLITUS:

The pandemic of the Acquired Immuno Deficiency Syndrome (AIDS) and the evidence of an association between TB and Human Immunodeficiency Virus (HIV), which causes AIDS, is now a cause of worldwide concern. Since containment of TB infection in an individual depends on intact cellular immunity, HIV due to its ability to destroy the immune system, now has emerged as the most significant risk factor for progression of the dormant TB infection to clinical disease. As a result, the TB problem not only has begun to worsen but also possess an unprecedented medical, social and economic threat, globally specially to developing countries.

In the early 1992, the global program on AIDS (GPA) of the WHO estimated that at least 9-11 million adults and one million children had been infected with HIV worldwide.

Nearly 85% of HIV infection had occurred in developing countries. And the vast majority in the age group 15-49 years. All epidemiological data collected during the 1980s point to continuing large increase in HIV sero prevalence level in Sub Saharan Africa and other developing countries.

The impact of HIV infection on the TB situation is obviously most serious when the prevalence of TB infection in young adults, who are at risk of HIV infection is high. It has been estimated that in early 1992, there had been more than 4 million persons with dual HIV and TB worldwide, a great majority (3-12) million of whom lived in Sub Saharan Africa.

The 'cursed duet' of TB and HIV infection is also responsible for the increase of TB in the Sub Saharan Africa, and the estimated annual risk of break down among those infected with both HIV and TB varies from 5-to 8% with accumulative life time risk of 30% or more.
Tanzania for example has reported 22,544 cases in 1990, with an increase of 86% over the 12,089 cases reported in 1984. Burundi 140%, Malawi 180%, and Zambia 154% over the same period (49).

In summary: The HIV prevalence will worsen the TB situation in developing countries in three ways:

1. By reactivation of latent TB infection among dually infected persons.
2. By new infection with TB bacilli and rapid progression to active disease in HIV infected persons.
3. By increasing the number of cases in the general population whose infection and disease will result from transmission from HIV +ve individuals developing TB either by reactivation or recurrent infection (49).

There is a long period, often several years, between infection with HIV virus and developing AIDS. During this time, the patient may feel quite well (though he/she remain infectious). The development of TB may be the first sign that he has HIV infection.

There is some evidence that TB in an HIV infected patient may speed the development of full clinical AIDS.

The following are differences from the usual ways TB shows in patients without HIV infection:

1. Extra pulmonary disease: Specially in L.N.s is more common. There is often L.N enlargement, which is rare in other forms of TB.
2. Miliary Disease is common. TB may be isolated from blood culture (which never occurs in ordinary TB) (18).
3. X-ray in pulmonary disease there are often large mediastinal L.N masses up to 50%. There is often lower lobe disease, cavitation may be less frequent, though reports from different areas vary. Pleural Effusion is more common. The shadows in the lungs may change rapidly (49-52).

The chest X-ray may be normal in higher proportions (54).
(4) TB may occur at unusual sites e.g. tuberculomas of the brain, abscesses of the chest wall or elsewhere.

(5) Sputum smear may be negative despite considerable changes in the chest X-ray; though some workers in Africa find smears positive as often as in non-HIV patients.

(6) The Tuberculin test is often negative\(^{(11)}\).

**DIABETES MELLITUS:**

In DM it has been suggested that TB tend to occur predominantly in the lower lobes. The right base was more frequently affected with cavitations and fluid levels.\(^{(136)}\).

Spencer et al\(^{(136)}\) found that 6.3% of patients presenting having anterior segment involvement with statistically significant occurrence in diabetics.
DIAGNOSIS OF PULMONARY TB:

In high prevalence areas and when the patient presents with the classical form, diagnosis is easy. But TB can have wide-varied clinical manifestations which sometimes render its diagnosis very difficult.

Delay in the diagnosis may result in increase in patient morbidity & further spread of the disease. In a study of big number city hospitals in the USA 20% of patients with culture proven pulmonary TB either died or were discharged before the diagnosis of TB was made.(84).

4.8% of pulmonary TB was diagnosed at death in the USA between 1985 through 1988 and 18% for extra pulmonary disease.(85).

This picture is now being seen in Europe and USA because of low prevalence rates & thus a lower index of suspicion of the disease. Older patients were misdiagnosed more commonly than younger patients.(82% vs. 48%). Patients without respiratory symptoms were misdiagnosed more frequently (78% vs. 49%) than were those with symptoms. Other reasons for delayed diagnosis and treatment included low use to tuberculin skin test, mis-interpritation of unusual chest roentegenograms and waiting for culture results in patients with negative acid fast smears.

The mean clinical evolution time before diagnosis was made, was of 3 months in young adults.(86).

SMEAR EXAMINATION:

The only absolute proof of tuberculosis is the cultural identification of the tuberculosis from tissue or body fluid; sputum, gastric washing, urine, CSF, serous effusion or pus from an abcess or sinus. A useful preliminary examination however, is to make a smear of material and stain it for standard microscopy or fluorescent microscopy, though less definite.

The smear is not a very sensitive method, but it has the virtue of quickly identifying (1) the patient who is discharging great number of organisms into the environment
and therefore is the source of infection and (2) confirming the presence of TB in that suspected patient. (3) following response to a given treatment.

Whether the staining is by conventional Ziehl-Neelsen (ZN) method or by one of the fluorochrome procedures, the mycobacterium property being examined is the retention of carbol fuchsin and related dyes after exposure to acid alcohol. Acid-fastness is indicated by red stained bacteria against a blue or green background (ZN) or alternatively by yellow fluorescent mycobacteria against a dark background.

The Fluorescence method, though less definitive allows large numbers of specimens to be examined rapidly. The most commonly examined material is sputum. When it can be produced spontaneously (usually in the early morning) it makes the most satisfactory material for both smear and culture. It should be provided under supervision, as the patient should be trained to produce real sputum and not saliva. Inhalation of aerosolized heated saline may help to stimulate production of bronchial secretions.

Bacteriological specimens may also be collected by bronchial washing, bronchial brushing or transbronchial biopsies using fibro-optic bronchoscopy. Alternative methods are laryngeal swabs in gastric aspiration in patients who cannot produce sputum e.g. young, elderly or psychotics or transtracheal aspiration, fine needle aspiration biopsy or mediastinoscopy.

Multiple specimens may be needed for confirmation of diagnosis and according to WHO regulations a minimum of two and a maximum of three sputum specimens should be provided for examination.

Quantitative grading of smears is of value in following prognosis, especially in research.

The amount of bacilli in the smear corresponds fairly closely to the concentration of bacilli in the sputum. It was shown experimentally that below a certain concentration of bacilli in a sputum specimen, the probability that acid-fast bacilli will be transferred from the specimen to the smear and found by microscopy approaches zero. 
Direct smear is occasionally negative in a patient with far advanced disease and rarely cultures are also negative. By contrast, it must be remembered that an unexpected positive smear or culture examination in a patient whose clinical characteristics do not otherwise support TB may be due to some error.

**Causes of a false positive result:**

1. Acid-fast particles other than tubercle bacilli, but showing the same property of acid-fastness.
   
   i. Food particles (e.g. oils, waxes)
   
   ii. Precipitates (from stain)
   
   iii. Other micro-organisms e.g. saprophytic acid-fast bacilli, Mycobacterium Kansassi or Norcardia spp.
   
   vi. Spores of bacillus subtilis.
   
   v. Fibres and pollens.

2. Scratches on the slide.

3. Contamination through transfer of bacilli from one smear to another.

**Causes of False negative results:**

1. Inadequate sputum collection.

2. Inadequate storage of sputum specimens and stained smears.

3. Failure to select suitable sputum particles for smear preparation.

4. Inadequate preparation of smear staining of slides.

5. Inadequate examination of the smear.

6. Administrative and Reading errors.
When adequately performed sputum examination is a reliable method and up to 93% reliability was alleged\(^{(56)}\).

The probability of obtaining a +ve sputum smear result ranged from 43% up to 88% \(^{(12)}\). So there still remains a good amount of patients who has to be diagnosed by other means.

Some studies \(^{(57,58)}\) have shown that several demographics, clinical and roentegenographic factors were associated with an increased risk of being sputum positive, age younger than 50 years, a positive tuberculin test, report of a cough, and a cavitary lesion on Chest X-ray.
TUBERCULIN SENSITIVITY:

The most readily obtained evidence of a past or present infection with tubercle bacilli, is a finding of hypersensitivity to tuberculin, which is a purified protein derivative of the broth in which tubercle bacilli have been grown.

There are several tuberculins, most important of which are: PPD-S and PPD-RT23.

Tuberculin skin testing may be performed by intradermal injection of PPD (the Mantoux test) or the multiple puncture device utilizing Old Tuberculin (the Heaf and Tine tests). The strength of PPD is expressed in terms of international units (IU) the standard dose according to the WHO recommendation is to give 2-5 IU

The mantoux test is performed by intradermal injection of 0.1 ml of PPD into the extensor surface of the forearm using a disposable syringe. The injection should be made just beneath the surface of the skin. The test is read at 84-72 hours. The reaction consists of erythema and induration. The erythema should be disregarded and the diameter of the induration measured in millimeter. Induration of greater than 10 mm is virtually diagnostic of past or present infection. The heaf test is carried out with a gun with fixed six short steal needles. Penetration of the tested skin take place through a thin film of applied solution. The test is best read between 3-7 days and is graded into 4 grades (I - IV). Grade III and IV are indicative of past or present infection. Grade I and II may be due to other mycobacteria or past vaccination with BCG.

As mentioned above the positivity of Tuberculin testing merely indicates previous infection with M. Tuberculosis and it should be differentiated clearly between infection and active disease.

Although, with proper attention to careful technique, tuberculin testing is very useful in measuring the prevalence of TB in a community, in many poorer countries it is much less valuable as a tool of diagnosis*

Using the Mantoux technique with 5 TU of PPD-S or its equivalent, surveys of TB patients throughout the world have shown remarkable degree of concordance in the
size of skin reaction. The distribution of reactions is normal with a mean ranging from 12.8 mm in South India to 18.8 mm in Republic of Sudan. A strongly positive test is, of course, a point in favour of TB, but a negative test does not exclude TB.

As it is the first marker of infection, Tuberculin conversion was used to detect recent infection (when done periodically) in persons at high risk e.g. hospital employers. The M. T. is particularly helpful in children suspected of TB who are less than 5 years old and have not received BCG vaccination. However a recent study in India conducted in children 2-12 years showed that the M.T. helped in diagnosis of TB in almost 50% of the cases.

Non-reaction to Tuberculin does not exclude active disease and causes of false negatives and anergy include:

1- Malnutrition (e.g. Kwashiorkor)
2- For a variable period of time during and after certain vaccination e.g. live virus vaccination such as measles.)
3- Overwhelming tuberculous disease (e.g. Miliary TB or Cryptogenic miliary disease and tuberculous meningitis)
4- Drugs. (e.g. Corticosteriod therapy)
5- During the third trimester of pregnancy.
6- HIV infection.
7- Viral infection (e.g. chicken pox and glandular fever).
8- Age (e.g. newborns and elderly).
9- Metabolic diseases (e.g. Chronic Renal Failure).
10- Malignancy.
11- Technical failure with the tuberculin e.g. in storage, administration, reading, etc.

Tuberculin skin anergy is associated with 8% of patients with TB. Infection with other mycobacteria (usually non-pathogenic) & previous BCG vaccination may give a false positive result, but usually a weakly positive result.
ROLE OF RADIOLOGY:

Historically, radiological examination had had an important role in the diagnosis and treatment of pulmonary TB in the past nine decades. Very soon after the discovery of the X-rays in 1895, it became apparent that chest X-ray would be a help in identifying pulmonary lesions. As the quality of examination improved, the benefits become significantly better. Earlier lesions were diagnosed. Follow up studies after medical or surgical treatment were increasing helpful. Newly developed techniques allowed demonstration of extra pulmonary complications. Radiological monitoring of collapse therapy played a critical role in the success achieved even after the medical conquest of active tuberculous lesion by drug therapy, identification of new lesions and follow up of treated patients remains a significant role for radiology. Control of this once fatal and widely feared disease depends on continued early recognition and appropriate treatment.

In an Italian study, the authors summarized the role of radiologist towards the problem of TB as follows:

"During the treatment of pulmonary TB, the radiologist is often asked the following questions:

1- Is it really TB?
2- Is there any improvement?
3- When is the X-ray check up required?"

In our opinion, the radiologist is in a position:

1- To confirm the diagnosis of TB.
2- To give a radiological diagnosis of "abnormality" but without prognostic opinion.
3- To suggest, case by case, when the X-ray check up is required.

After some months or years, when radiological, clinical sequelae are present, the radiologist is often asked equally difficult questions:

1- Is it still TB?
2- Is it still active?
3- Could it have caused the haemoptysis?
Mass roentgenography for active case-finding in TB was once very popular all through the modern world and the yield from it was very rewarding. Mass roentgenography for TB case-finding has been reported to be responsible for the detection of 10-30% of active cases of the disease(66,67,76).

But as the prevalence of TB declined in Europe and the United States, it is now being evaluated as not cost-effective as "the actual efficiency of the radiographic chest screening is thus very low" (68), or more quantitatively "Results suggest inadvisability of carrying out mass chest X-rays examination if the tuberculosis morbidity is less than 30/100,000(69)."

"The predictive value of positivity are likely to be very low at the current case prevalence rate in the community, being 2-8 per thousand(70)."

But it still finds its place in places with higher prevalence rates. Many years observations carried out in different territories of Kazakhstan have demonstrated that repeated total flurographic examination of the population result in decrease of TB morbidity, in the proportion of destructive forms and fibrocavernous tuberculosis (71,80).

Mass miniature X-ray screening for TB in immigrants from high prevalence countries entering Switzerland still detects a majority of asymptomatic cases & seems an easy means of preventing the transmission of TB to members of the same community(68). The above situation proves it necessary to conduct preventive flourographic examination of the chest at least once every two years(83).

Although sputum examination is the investigation of choice to detect TB cases, but radiology is equally sensitive in detecting cases. It is well acceptable by the population, and a chest radiogram taken by a transportable chest X-ray apparatus or examination of two sputum specimens might be equally successful at detecting all cases of active pulmonary TB within the time required for sputum culture(70).

If chest X-ray is the first investigation to be done, it can pick easily those who are likely to be contagious. Of 58 patients whose chief complaints were unrelated to pulmonary TB, the chest X-ray suggested TB in 52(90%). Among patients whose
roentgenograms showed cavitation or extensive alveolar infiltrate, sputum smear showed AFB in 98% of cases. If alveolar infiltration is absent, or if the roentgenograms pattern was not that of adult reactivation disease, sputum smears revealed AFB in only one half of the cases.

The only serious hazard from radiation is the development of superficial malignancy when repeated low-dose chest fluoroscopy was done especially when collapse therapy was adopted as treatment of TB in (1925-1954). Among 2,573 women examined by X-ray-fluoroscopy an average of 88 times during lung collapse therapy and followed for an average of 30 years, 147 breast cancers occurred in contrast to 113.6 expected [O/E = 1.29, 95% confidence interval]. Increased rates for breast cancer were not apparent until about 10-15 years after the initial fluoroscopic examination. Age at exposure strongly influenced the risk of radiation-induced breast cancer with young women being at highest risk and those over 40 years of age being at lowest. The breast is one of the most sensitive tissues to the carcinogenic force of radiation, and that fractionated exposures are similar to single exposure of the same total dose in their ability to induce breast cancer.

Nowadays when such repetitive fluorographic examination are not performed, the breast doses are too low, 8.27 G on average, to expect to detect a significant elevation in breast cancer risk overall.

The standard technique of examination is the standard posterio-Anterior film. Lateral films may be needed for more accurate localization of lesions. Apical lardotic views may also be asked for better visualization of the apices.

For small especially sub pulmonic effusion lateral decubitus films may be requested to displace the fluid against the lateral chest wall. Tomography may also be needed to evaluate the boundaries and walls of a suspected cavity.

In any technique image quality has to be high enough that even subtle pulmonary abnormalities can be easily visualized. Conventional screen-film radiographs obtained with 200-400 speed systems fulfill this requirement well enough. Large screen and slit beam intensifier system that have replaced fluoroscopy screen, yield equivalently good
results. The amber system and storage phosphor digital radiograph system are efficient alternatives.

Radiation exposure is generally low and decrease further if image intensifiers are used. Conventional techniques with highly sensitive film/screen combination has been approved for diagnosis of pulmonary TB. So radiology is important in the management of TB for:

1- Active and passive case finding.
2- Confirmation of diagnosis in symptomatic patients.
3- Showing the extent and pattern of the disease.
4- Prediction and assessment of complications.
5- Monitoring response to therapy (whether medical or surgical).
RADIOLOGICAL FEATURES OF PULMONARY TUBERCULOSIS:

Pulmonary TB can mimic almost all other pulmonary diseases in much the same way as syphilis may mimic most neurologic diseases(12) and there may be considerable amount of disagreement between radiologists on individual X-ray as has been dedicated from the comparative studies done by the IUAT on reading-interpretation of chest X-ray films(79). They concluded that purely radiologic criteria can not give really satisfactory evidence of TB in the individual patients.

As we have earlier seen that TB is divided into primary and post primary or reinfection TB. The main radiological criterion that differentiates both is the presence or absence of hilar or paratracheal L.N. enlargement(13,87,88). Distinction between both types necessitates that the chest X-ray should be the first step of the work up of the patients(82).

In a minority of patients, primary disease may progress directly to a chronic destructive form of reinfection TB, this transition occurs particularly in infants under the age of one, is uncommon between the age 2-12 years and has been estimated to occur in about 10% of cases in adolescence and young adults(80). And these do show both signs of primary and post primary disease on the same radiograph(89). Some researchers(81) do not agree to this and they believe that although lymphadenopathy is more common with primary disease, it is not specific for it, particularly in adults and they found lymphadenopathy in post primary disease in 5% of cases.
PRIMARY PULMONARY TB:

Primary infection with the tubercle bacilli within the thorax usually affects one or a combination of four structures:

1. The pulmonary parenchyma.
2. The mediastinal and hilar L.N.
3. The tracheo-bronchial tree.
4. The pleura.

**Parenchymal involvement:**

The upper lobes were slightly more frequently involved than the lower, especially the anterior segment of the upper lobe, although there were no significant differences between left and right lungs or between anterior and posterior segments. Some found that the right lobe is slightly more affected and specially the right middle lobe, and mostly affected is the apex of the lower lobe.

The Parenchymal reaction typically is that of air space consolidation, usually homogenous in density and have ill-defined margins, except when it abuts on a fissure. Lobar involvement probably is due to a combination of parenchymal consolidation (due to direct invasion by the organism) and atelectasis resulting from bronchial obstruction due to enlarged L.N or endobronchial disease.

It occurred in about 50-64% in patients with primary disease and there were no significant differences between consolidation occurring in children or adults.

**Cavitary Disease:**

This shows in about 10% of children and is more common in older children and adolescence where it may amount up to 30%.

Miliary disease may be found in up to 6% in patients with primary disease.
**Lymph Node Involvement:**
This shows clear-cut radiological evidence in up to 97% of cases. In the remaining it was obscured by contiguous pulmonary disease. In 10% L.N enlargement was bilateral and hilar and in 42% it was predominantly unilateral and hilar and in other 42% it was unilateral and both hilar and Parenchymal\(^{13}\). L.N involvement is less commonly found in adult onset primary disease \((35-43%)^{63,60}\), and here the right paratracheal were the commonest to be affected.

**Air Way Involvement:**
Tracheobronchial disease is common and usually is the result of the compression of the bronchi by enlarged L.N. And less commonly tuberculous granulation tissue may accumulate in the mucosa and can be identified bronchoscopically\(^{13}\).

Atelectasis was identified in up to 30% of cases\(^{95}\), and more commonly it affected the right lung\(^{95}\).

**Pleural Involvement:**
Pleural effusion as a manifestation of primary TB occurs more commonly in adults than in children. It occurs in about 10-30% of patients and is usually mild to moderate in degree.

It is usually accompanied by radiological evidence of pulmonary disease\(^{13}\).

**Calcification:**
Whether in pulmonary or L.N. is rather rare in primary TB and when it occurs it is usually small less than 5mm in diameter.

Patients with primary disease may show an entirely normal chest X-ray. Here, the disease may be entirely endobronchial. These patients may diagnosed by sputum smears or bronchoscopy.
POST PRIMARY OR REINFECTION TB:

Several roentegenographic patterns may be identified in reinfection TB and any individual case may show one or more of these patterns:

(1) **Local exudative TB:**

The roentegenographic pattern of exudative pulmonary TB is one of air space (acinar) consolidation, either patchy or confluent in nature, situated in the specific anatomic regions - the apical and posterior segments of an upper lobe or the superior segment of a lower lobe.

Individual shadows commonly are homogenous and indistinctly defined. There is frequently an accumulation of the drainage markings towards the ipsilateral hilum. Cavitation may or may not be present. L.N enlargement is not a feature.

The shadows may disappear following the treatment, or if not treated may either progress and become confluent to form acute tuberculous pneumonia or regress into fibroproductive lesion.

(2) **Local Fibroproductive TB:**

The relatively poor definition of the exudative lesion is replaced by a more sharply circumscribed shadow, usually somewhat irregular and angular in contour. Although the shadow may be homogenous its density is no greater than that of exudative lesion.

Cavitation may be present, but not evident on the X-ray or it may be clearly shown. Healing occurs by replacement of the tuberculous granulation tissue by fibrous tissue, the resultant cicatrazation may result in considerable loss of volume. Bullae formation may also occur. Signs of loss of lung volume are:

1. Change in site of one of the hila.
2. Diaphragmatic elevation.
4. Fissural displacement.
5. Compensatory emphysema in adjacent lobes.
(3) Cavitation :-
When liquefied caseous material is expelled from the center of the lesion into the bronchial tree, a resultant cavity forms. The wall of an untreated tuberculous cavity is moderately thick and its inner surface usually is fairly smooth. And air-fluid level is seldom seen. Sometimes cavities may not be visible in routine chest films and 8.8% may need tomography for their visualization.

(4) Bronchogenic Spread and Acute TB Pneumonia :-
When liquefied material is expelled through the bronchial tree it might be either expectorated with sputum or spread to other segments of the same or opposite lung. Characteristically such dissemination leads to the formation of multiple small (4-6mm) shadows, which have characteristics typical of acinar shadows. This appearance is almost pathognomonic of bronchogenic spread of TB. Extension of the disease through surrounding of air spaces sometimes leads to acute confluent air space pneumonia. Clues to TB aetiology may be provided by the identification of an open cavity somewhere else and by the presence of fairly discrete acinar shadows in parts of the lung remote from the massive consolidation. This later feature was not seen in air space pneumonia of other aetiology.

(5) Miliary TB :-
If large number of tubercle bacilli enter the blood stream, specially in an immunocompromized host, there is wide spread invasion of tissues and organs. In the lung, this is manifested by the appearance of tiny discrete foci widely and uniformly distributed throughout both lungs. The time interval between dissemination and the development of a roentegenographic evidence of disease probably is 6 weeks or more, during which time, the foci are so small for roentegenographic visualization. When they do become visible (preferably on low Kv exposure) their appearance is distinctive - tiny, absolutely discrete, pin-point shadows evenly distributed.
throughout both lungs. When first visible they measure little more than 1mm in diameter and in absence of adequate therapy they may reach 2-3mm in diameter before the patient dies. By this time, they may have become almost confluent, presenting what has being termed "Snow storm" appearance. Rarely, patients die from miliary TB (proved pathologically) without demonstrable abnormality on the chest X-ray.

Roentgenographic miliary shadows may be seen accidentally in asymptomatic patients in X-ray done for other purposes(37).

(6) Tuberculous Bronchiectasis:

Two mechanisms may be involved in the pathogenesis of tuberculous bronchiectasis:

1- The bronchial wall is infected during the active phase of the disease and the fibrosis and cicaterization which occurs during healing leads to irreversible bronchial dilatation.

2- A segmental bronchus is obstructed by a compression of enlarged L.N in primary TB or by bronchostenosis secondary to endobrochial disease and the results is obstructive pneumonitis and subsequent bronchiectasis.

Cystic bronchiectasis may manifests itself as cavitation or honeycombing, curvilinear shadows or cystic changes in the lungs, while "tram-line" or tubular shadows appearance may be seen in a cylindrical type(37).

The same appearance is seen in C/T cuts which proved that bronchiectasis was quite common with TB. Since the vast majority of post primary disease is in the upper lobe, thereby facilitating drainage, bronchiectasis is usually asymptomatic though haemoptysis may occur.

Tuberculous Bronchostenosis:

In primary TB, the tracheobronchial tree is involved chiefly as a result of pressure from enlarged L.N. In reinfection TB endobronchial involvement is fairly frequent and occurs particularly in air ways that drain a pulmonary cavity. Ulceration of the
bronchial mucosa leads eventually to fibrosis and cicatrical bronchostenosis. This may occur without any radiological evidence. And if untreated may lead to obstructive atelectasis, pneumonitis or bronchiectasis, visible radiologically. The patients are suspected clinically by a persistent wheeze and they are usually sputum positive.

Tuberculoma:-
This may be a manifestation of either primary or reinfection TB. It is usually round or oval lesion situated most commonly in the upper lobes; the right more often than the left.

Tuberculomas range in size from 0.5 - 4 cm or more in diameter and typically are smooth and sharply circumscribed, but up to 25% may be smooth and lobulated.

Small discrete shadows in the immediate vicinity of the main lesion "satellite" lesions may be identified in as many as 80% of cases.

Tuberculomas may be seen in 7% of cases. The majority of these lesions remain stable for a long time and many calcify. The larger the mass, the more likely it is to be active.
ANATOMIC DISTRIBUTION :-

A characteristic manifestation of reinfection TB is a tendency to be localized in the apical and posterior segment of the upper lobes (85%) and the superior segment of the lower lobe (9.5%), with only 4.5% in other locations\(^{100}\).

Recent studies were done to see if there are any changes in the classical distribution sites. It was found that no much change occurred \(^{93, 101, 102}\) in the recent years.

The lower lobes are seldomly involved in other than the superior segments. Lower Lung Field TB (LLF-TB) together with affection of the anterior segment of the upper lobe was found in 6.3% \(^{103}\) & occurs predominantly in patients with predisposing factor for the disease e.g. Alcoholics, diabetics, and immunocompromised and they also show the presence of multiple cavities within any given lesion\(^{104}\).

The presence of local exudative or fibroproductive lesion of the chest X-ray of newly diagnosed post primary TB patients was found in up to 100% of cases\(^{93}\).

Cavitation may be seen in 45% up to 53% of cases \(^{106}\) & bronchogenic spread in 21%. Marked fibrosis 29%, pleural effusion 18%, empyema 4% and pleural thickening 4%, pneumothorax in 5%\(^{93}\).

Pleural effusion may also occur due to reinfection disease and it was shown that 15% of the pleural effusion was due to reinfection TB\(^{105}\).

Pleural effusion altogether occurred in 18% of cases of post primary disease\(^{93}\).

A chest X-ray may seem normal in a patient with bacteriologically proven pulmonary TB; There are two provisos:

It is possible for a patient to have localized post primary endobronchial TB with a positive sputum and a normal chest film. This picture may be seen in 1% up to 15% of cases of post primary pulmonary TB\(^{93, 107, 108}\).
missed «». Such observer error lesions may be reduced by double recording: by two independent observers or even by the same observer on two separate occasions.

Common causes of a missed diagnosis of TB are:

1- Failure to recognize hilar and mediastinal lymphadenopathy as a manifestation of primary disease in adults.
2- Exclusion of TB because the disease predominates in or is limited to the anterior segments of an upper lobe or the basilar segment of the lower lobe.
3- Overlooking of minimal fibroproductive lesions or reporting them as inactive.
4- Failure to recognize that an upper lobe mass surrounded by satellite fibroproductive lesions might be tuberculous, and
5- Failure to consider healed sequelae of primary disease or a positive PPD skin test as contributory to identifying the patient pulmonary disease⁹⁳. 
RADIOGRAPHIC PATTERN IN PATIENTS WITH AIDS :-

Patients with AIDS present with a slightly different radiological pattern from TB in normal hosts:

The major diagnostic findings are:

1- Frequent isolated involvement of hilar L.N as observed in primary TB (14.3%). The infiltrates in 80% are most frequently located in the middle lobe and basal lobes, excavations are uncommon (21%).

Pleural effusions are unusual, and in some patients (28.6%) the chest X-ray was normal[100,110,113].

Although some reporters report pleural effusion as being commoner in the HIV seropositive groups specially in negros[110]. The miliary pattern was most common in seropositive group[112].
RADIOLOGIC CLASSIFICATION OF PULMONARY TB:

Bearing in mind that many diseases may mimic TB roentogengraphically and that activity may be difficult to assess accurately in all cases, nevertheless, an objective approach to the description of the roentogengraphic shadows in the pulmonary TB is favored and it is the pathological classification that has been used earlier, in which pathological terms were borrowed from the pathologists but are more comprehensive. For clinical and research purposes the classification of the National Tuberculosis Association of the USA has proved useful. It is based mainly on the anatomical extent of the disease:

1- Minimal: Minimal lesions include those which are of slight to moderate density but which do not contain demonstrable cavitation. They may involve a small part of one or both lungs, but the total extent, regardless of distribution, should not exceed the volume of the lung on one side which is present above the second chondrosternal junction and the spine of the fourth or the body of the fifth thoracic vertebra.

2- Moderately advanced: May be present in one or both sides, but the total extent should not exceed the following limits: Disseminated lesion of slight to moderate density which may extend throughout the total volume of one lung or the equivalent in both lungs, dense and confluent lesions which are limited in extent to 1/3 the volume of one lung, total diameter of cavitation, if present, must be less than 4cm.

3- Far advanced: Lesions more extensive than moderately advanced.

Recently the American Lung Association has presented a new classification of pulmonary TB which rests primarily on bacteriology and the chemotherapeutic status. Roentog-engraphic findings are regarded as necessary only in certain circumstances.

Roentogengraphic findings are subdivided as follows:

1- Normal.

2- Abnormal.

(a) Cavitary or non Cavitary

(b) Stable, worsening or improving.
Assessment of activity: It is very difficult to make a clear cut statement on this on radiologic basis alone, and therefore bacteriological & clinical data should be used in conjunction with radiology to reach a decision.
OTHER RADIOLOGIC INVESTIGATIONS

Ordinary tomography is useful in defining the wall of cavities & rounded mass lesions & their contents.

With the advent of computerized tomography, it became more fashionable. It is more accurate & can detect lesions not detected on the normal chest x-ray: - miliary disease, cavities, bronchogenic spread, nodules & adenopathies.116. It is also more accurate in defining disease activity.117,118

Ultrasonography examination of peripheral lung lesions was alleged to better delineate the more complex nature of the lesion better than chest x-ray. U/S guided aspiration biopsy provided the diagnosis in 90% of cases, with no major complication. U/S can direct the needle to the suitable part of a lesion to obtain the relevant specimen. It is especially helpful in patients with negative result of sputum & bronchoscopic examination.119

Ultrasonography of the abdomen may also be helpful in diagnosis of abdominal TB.
BLOOD INVESTIGATIONS:

White cell count is usually normal or rather low. It may be sometimes raised in tuberculous pneumonia\(^{(18,24)}\) or in miliary T.B where a leucomoid reaction may sometimes suggest other diagnoses.

Anaemia (usually normochromic normocytic) is common in pulmonary T.B, but the more bizarre blood dyscrasia characteristic of miliary T.B are unusual and if present, most likely imply some disease with cryptic miliary spread\(^{(12)}\).

Erythrocyte Sedimentation Rate (ESR) :-

This is almost always raised in active TB \(^{(11)}\). A result in the order of three figures is quite common. But a normal result does not exclude active TB\(^{(12)}\).

Liver Function Tests (LFT):-

It is not uncommon to find abnormalities of liver function tests in moderate or advanced disease.
FIBROPTIC BRONCHOSCOPY:

Using fibroptic bronchoscopy allows not only substantial meaningful assessment of endobronchial tuberculosis, but also relieve atelectasis, eventually resulting in successful treatment of T.B\(^{(120)}\).

This investigation is especially important in patients with negative smear examination who traditionally present a considerable problem in diagnosis by providing early confirmation of diagnosis & material for culture\(^{(121)}\).
DIFFERENTIAL DIAGNOSIS OF PULMONARY TUBERCULOSIS:

In reference to differential diagnosis of TB, it has been said that to know syphilis is to know medicine, the same could be said for tuberculosis. In 1948, Gatland confined himself to the chest roentogengraphic manifestations of TB and produced a list of 89 alternate diagnoses.

Despite the long list of differential possibilities, the diagnosis of TB is simple when there is positive sputum microscopy, compatible chest X-ray and positive skin test results. When any of these are lacking, consideration of pertinent differential diagnosis is necessary. The alternative possibilities will depend on the clinical appearance of the individual patient:

**Bacterial infection:** a frequent problem in differential diagnosis is the patient who has acute symptoms of fever, leukocytosis and pulmonary infiltrates and a decision must be made as to appropriate antimicrobial treatment. If AFB are identified, a presumptive diagnosis of TB is made. If not, specially of the non cavitating type, the clues of a tuberculous cause include the appearance and the location of the infiltrate, evidence of bronchogenic spread and the clinical appearance.

The infiltrates detected in areas of lung part from major consolidation are evidence in favor of TB. Clinically patients with TB usually appears to have a less toxic condition than patients with bacterial infections, though similarly febrile, also TB patients are less dyspnoeic and tachypnoeic and exhibit less blood gas abnormality.

When cavitation is present, they can be confused with lung abscess and necrotizing pneumonia. TB cavities lack large air fluid levels and are usually associated with surrounding infiltrates. Lung abcesses are rarely found in an apical segment of an upper lobe. Production of large quantities of putrid sputum is pathognomic of anaerobic infection. Miliary TB may be confused with many diseases including bacterial infection e.g. haemolytic streptococci, staph. and mycoplasma pneumonieae.

When the clinical picture suggest chronic infection:
Ting et al (128) offer several roentgenographic criteria as suggestive of associated Ca:

(1) Progression of pulmonary infiltrate during treatment with anti TB drugs.

(2) Atypical location of the infiltrate.

(3) An atypical appearance of the infiltrate e.g. dense & homogenous without air bronchograms.

(4) Development of pleural densities during therapy.

(5) Hilar adenopathy.

(6) Presence of solitary nodule greater than 3 cm in diameter.

(7) Cavity with thick irregular nodular wall.

(8) Mass impression in a displaced lobular fissure.
MANAGEMENT & CONTROL

The following control measures are important in the achievement of the goal of eradication of the disease:-

1. **Improvement in socio-economic conditions:** mainly in respect of adequate housing ventilation & nutrition may still be the most important control measure of all.

2. **Case-finding:** The highest yield by far is from patients referred by general practitioners because of symptoms. Open access to chest radiography for general practitioners & minimum waiting time for patients are essential for success. Other important groups are persons in prisons, borstals, mental hospitals etc.... & people in high risk groups mentioned earlier.
   Sputum smear examination is an important and inexpensive method of case-finding in developing countries. The provision of a microscope and the training of a health worker in the examination of smears can be readily organised.
   Contact examination achieves a high yield case-finding. Efforts should be concentrated on the immediate examination of household contacts of sputum-smear positive patients especially amongst contacts under 25 years of age.

3. **Chemotherapy:** The proper use of modern highly-effective chemotherapy, by rendering patients non-infectious rapidly, makes a very important contribution to the control of the disease. The most important medications for the treatment of TB are isoniazid, rifampicin, pyrazinamide, ethambutol, streptomycin and thiacetazone.
   Thiacetazone, which is cheap, is widely used in developing countries. Pyrazinamide is particularly useful in the treatment of tuberculous meningitis because it diffuses well into the cerebrospinal fluid. Apart from minor a few minor variations in dose and duration of treatment, the policy governing the use of anti-tuberculous drugs is the same for all forms of the disease. The drugs are used in combinations and delivered in 2 phases: an initial phase (usually of two months period) in which three or four drugs are used & a continuation phase (of four to ten months duration) where two drugs are employed. The trend now is for using a larger number of more potent drugs for a shorter period of time.
4. **Isolation of patients:** is rarely considered necessary nowadays even in smear positive patients except where very young children are at risk, provided the source is being properly treated by chemotherapy.

5. **BCG Vaccination:** This is carried out by the administration of freeze-dried vaccine, reconstructed at the time of use, by the intradermal route (0.1 ml) injected at the junction of the upper & middle thirds of the upper arm. BCG vaccination should not be given in the presence of immunodeficiency. The duration of protection is from 3 to 7 years. Vaccination reduces the incidence of pulmonary tuberculosis in young adults by 80% and minimises the risk of serious disseminated disease: miliary tuberculosis and tuberculous meningitis.

6. **Chemoprophylaxis:** using isoniazid is indicated in: (a) non-BCG vaccinated tuberculin positive children under three years of age, as this is a vulnerable group in respect of miliary TB and tuberculous meningitis; (b) un-vaccinated individuals who have recently become tuberculin-positive; (c) patients on immunosuppressive drugs; (d) tuberculin-positive adolescents with a high level of tuberculin sensitivity; (e) infants of highly infectious parents, when isoniazid-resistant BCG vaccine may be administered, INH being given for 6 weeks thereafter to prevent infection until vaccination exerts its protective effects.

7. **Elimination of Bovine infection:** Although it is now becoming rare, vigilance will be required to ensure that it remains so.
CHAPTER II:

OBJECTIVES
OBJECTIVES:

1. To study the radiological pattern of pulmonary tuberculosis in patients in the sample studied.

2. To determine the percentage of radiologically positive patients in the sample studied.

3. To study the frequency of the initial presenting symptoms & correlate their severity with the chest x-ray findings.
CHAPTER III:

METHODS & DESIGN
METHODS & DESIGN

The study was conducted in the period form December 1994 up to August 1995 in El Shaab teaching hospital & Abu Anja tuberculosis Hospital. Cases were collected from the out patient clinics & the wards. Only newly diagnosed cases, who did not receive any anti-tuberculosis therapy were entered into the study.

160 Adult patients were selected randomly according to WHO criteria for diagnosing tuberculosis:-

1. Cases with two positive sputum smear results, e.g. over night & spot sample i.e. in two or more different occasions.

2. Has only one sputum positive smear, but with strong clinical suspicion.

3. Have 3 negative sputum smear results, but with strong clinical & radiological evidence of tuberculosis & the medical officer decides on proceeding to treatment.

Strong clinical suspicion was raised on patients presenting with the following symptoms either separately or in combination:-

1. Cough for more than 3 weeks.

2. Haemoptysis

3. Fever

4. Sweating

5. Malaise

6. Loss of weight

7. Chest pain

8. Breathlessness
EXCLUSION CRITERIA :-

1. Any patient with concomitant cardio-vascular or chest disease that might affect his chest X-ray will be excluded from the study.

2. Patients below 15 years of age.

METHODS :-

Each patient had a sputum examination done on 3 different occasions, sputum, being collected according to WHO recommendation & stained by Zeihl-Nelson stain for AFB.

b- Each patient had a tuberculin test done. Most patient had Mono test done & the reaction so obtained was recorded.

c- Each patient had a standard postero-anterior chest X-ray film taken with the appropriate facors. Some patient had a laterel film when necessary. All xrays were interpreted by at least two radiologists to reduce the in-between observer error.

d- Each patient had an ESR examination done.

A flow-sheet was filled in for each patient. Every patient was personally interviewed by the investigator. Physical examination was conducted by the chest physician or the medical registerar of the unit.

Collected data was analysed by the computer using EPI. INFO software programme. A 95% confidence limit was taken as significant.

A Medline search for comparable literature using MESH :- Tuberculosis-Pulmonary-radiography was done & supplemented by manual search of the library for articles outside the Medline range.

h- Data collected was compared with world-wide literature.
Difficulties & Constraints :-

Although patients with coexistent disease were excluded from the study some patients with certain chest diseases, may present with typical tuberculous picture & may have even a positive sputum smear, tuberculosis occurring as coexistent infection. Such patients might have been unknowingly included in the study.

X-rays are rather expensive & tuberculosis is the disease of the poor. As aimed earlier was to a PA & lateral view for each patient but this was not financially feasible for each patient. Other views could not be done either.

Shortage of x-ray films at times, tuberculin test equipments & some other technical obstacles had somewhat delayed the study.
CHAPTER IV:

RESULTS
SEX INCIDENCE OF PATIENTS IN THE STUDIED SAMPLE (DEC. 94 - AUG. 95)

- MALE: 117
- FEMALE: 43
AGE DISTRIBUTION IN THE STUDIED
SAMPLE (DEC. 94 - AUG. 95)

No. OF PTS

AGE

15-25 26-35 36-45 46-55 56-65 66-75
AGE AND SEX DISTRIBUTION OF THE PATIENT IN THE STUDIED SAMPLE (DEC.94 - AUG.95)

<table>
<thead>
<tr>
<th>Age Range</th>
<th>No. ofPts</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>15-25</td>
<td>50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>26-35</td>
<td>40</td>
<td></td>
<td></td>
</tr>
<tr>
<td>36-45</td>
<td>30</td>
<td></td>
<td></td>
</tr>
<tr>
<td>46-55</td>
<td>20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>56-65</td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>66-75</td>
<td>0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
RESIDENCE OF PATIENTS IN THE STUDIED SAMPLE (DEC. 94 - AUG. 95)

KHARTOUM: 58
KRT NORTH: 26
OUTSIDE KRT: 11
OMDURMAN: 65
ORIGIN OF PATIENTS IN THE STUDIED SAMPLE (DEC. 94 - AUG. 95)

- North: 7
- West: 64
- South: 41
- Central: 25
- Ethiopia: 4
- KRT State: 17
OCCUPATIONS OF PATIENTS IN THE STUDIED SAMPLE (DEC. 94 - AUG. 95)

<table>
<thead>
<tr>
<th>OCCUPATION GROUP</th>
<th>No. OF PTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-Skilled Lab.</td>
<td></td>
</tr>
<tr>
<td>Skilled Lab.</td>
<td></td>
</tr>
<tr>
<td>Students</td>
<td></td>
</tr>
<tr>
<td>House Wives</td>
<td></td>
</tr>
<tr>
<td>Non-Occupied</td>
<td></td>
</tr>
</tbody>
</table>

No. OF PTS
PRESENTING COMPLAINTS IN THE STUDIED SAMPLE (DEC. 94 - AUG. 95)

PRESENTING COMPLAINT

- COUGH
- SPUTUM
- HAEMOPTYSIS
- CHEST PAIN
- BREATHLESSNESS
- FEVER
- SWEATING
- MALAISE
- WEIGHT LOSS
- OTHERS

No. OF PTS
### TABLE 1

**DURATION OF SYMPTOMS IN THE SAMPLE STUDIED**
*(DEC. 94 - AUG. 95)*

<table>
<thead>
<tr>
<th>Duration of Symptoms</th>
<th>No. of Patients</th>
<th>% of Total Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>4-8 weeks</td>
<td>41</td>
<td>25.6</td>
</tr>
<tr>
<td>8-12 weeks</td>
<td>31</td>
<td>19.3</td>
</tr>
<tr>
<td>&gt; 12 weeks</td>
<td>60</td>
<td>37.5</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>160</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>

Max : ‡ 770 days  
Min : 21 days  
Mean : ‡ 120 days = 4 months  
Mode : 84 days ≥ 2.3 months = 12 weeks  
Median : 84 days ≥ 2.3 months = 12 weeks
TABLE 2

SOCIAL HABITS OF PATIENTS
IN THE SAMPLE STUDIED
(DEC.94 - AUG.95)

<table>
<thead>
<tr>
<th>TYPE OF HABIT</th>
<th>NO. OF PATIENTS</th>
<th>% OF TOTAL PATIENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking</td>
<td>45</td>
<td>28%</td>
</tr>
<tr>
<td>Alcohol</td>
<td>31</td>
<td>19.5%</td>
</tr>
<tr>
<td>Drug Addict</td>
<td>6</td>
<td>4%</td>
</tr>
<tr>
<td>SOCIAL HABIT</td>
<td>FAR-ADVANCED STAGE ON X-RAY</td>
<td>NOT FAR ADVANCED</td>
</tr>
<tr>
<td>---------------</td>
<td>-----------------------------</td>
<td>-----------------</td>
</tr>
<tr>
<td>Smokers</td>
<td>15</td>
<td>30</td>
</tr>
<tr>
<td>Non-smokers</td>
<td>32</td>
<td>83</td>
</tr>
<tr>
<td>TOTAL</td>
<td>47</td>
<td>113</td>
</tr>
</tbody>
</table>

Chi square: 0.47  P-value : 0.49
Taking 95% confidence limit
## TABLE 4

### RELATION BETWEEN HEAVY SMOKING AND DEVELOPMENT OF FAR-ADVANCED LESIONS IN THE CHEST X-RAY IN THE SAMPLE STUDIED (DEC. 94 - AUG. 95)

<table>
<thead>
<tr>
<th>SMOKING</th>
<th>FAR-ADVANCED</th>
<th>NOT FAR-ADVANCED</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heavy Smokers</td>
<td>7</td>
<td>6</td>
<td>13</td>
</tr>
<tr>
<td>Non Smokers</td>
<td>32</td>
<td>83</td>
<td>115</td>
</tr>
<tr>
<td>Total</td>
<td>39</td>
<td>89</td>
<td>128</td>
</tr>
</tbody>
</table>

Chi square : 3.73  
P-value : 0.05  

Taking 95% confidence limit.
<table>
<thead>
<tr>
<th>FINDINGS</th>
<th>NO. OF PATIENTS</th>
<th>% OF TOTAL PATIENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>ILL-LOOKING</td>
<td>51</td>
<td>32%</td>
</tr>
<tr>
<td>WASTING</td>
<td>74</td>
<td>46.5%</td>
</tr>
<tr>
<td>PALLOR</td>
<td>59</td>
<td>37%</td>
</tr>
<tr>
<td>CLUBBING</td>
<td>9</td>
<td>5.7%</td>
</tr>
<tr>
<td>PERIPHERAL Lymphadenopathy</td>
<td>8</td>
<td>5%</td>
</tr>
</tbody>
</table>
### TABLE 6

**EXTRA-PULMONARY SIGNS IN THE STUDIED PATIENTS**
*(DEC. 94-AUG. 95)*

<table>
<thead>
<tr>
<th>SYSTEM</th>
<th>NO. OF PATIENTS</th>
<th>% OF TOTAL PATIENTS</th>
<th>ABNORMALITIES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular system</td>
<td>1</td>
<td>0.6%</td>
<td>Hypertension</td>
</tr>
<tr>
<td>Central nervous system</td>
<td>3</td>
<td>2%</td>
<td>Lower Limb weakness</td>
</tr>
<tr>
<td>Abdomen</td>
<td>4</td>
<td>2.4%</td>
<td>Pregnancy, aly</td>
</tr>
<tr>
<td>Skin</td>
<td>6</td>
<td>3.6%</td>
<td>Scrofula, Vitiligo, Impetigo, Tinea Versicolor</td>
</tr>
</tbody>
</table>
## TABLE 7
CHEST SIGNS
OF PATIENTS IN THE
SAMPLE STUDIED
(DEC. 94-AUG. 95)

<table>
<thead>
<tr>
<th>SIGNS OF</th>
<th>NO. OF PATIENTS</th>
<th>% FROM TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>No signs</td>
<td>16</td>
<td>10%</td>
</tr>
<tr>
<td>Fibrosis</td>
<td>19</td>
<td>12%</td>
</tr>
<tr>
<td>Cavitation</td>
<td>57</td>
<td>36%</td>
</tr>
<tr>
<td>Collapse</td>
<td>9</td>
<td>6%</td>
</tr>
<tr>
<td>Pleural Effusion</td>
<td>14</td>
<td>9%</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>5</td>
<td>3%</td>
</tr>
</tbody>
</table>
THE RATIO OF SUPTUM +ve TO -ve IN THE STUDIED SAMPLE (DEC. 94 - AUG. 95)

+ve FOR AFB
90

-ve FOR AFB
70
THE RATIO OF MANTUX +ve TO -ve IN THE STUDIED SAMPLE (DEC. 94 - AUG. 95)

MANTOUX +ve 117

MANTOUX -ve 30
TABLE 8

ESR RESULTS
IN THE STUDIED SAMPLE
(DEC. 94-AUG. 95)

<table>
<thead>
<tr>
<th>ESR (mm/hr)</th>
<th>NO. OF PATIENTS</th>
<th>PERCENTAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 - 49</td>
<td>3</td>
<td>2%</td>
</tr>
<tr>
<td>50-99</td>
<td>48</td>
<td>32%</td>
</tr>
<tr>
<td>≥ 100</td>
<td>109</td>
<td>66%</td>
</tr>
<tr>
<td>TOTAL</td>
<td>160</td>
<td>100%</td>
</tr>
</tbody>
</table>

Mean 104
Minimum 5
Maximum 150
Mode 120
Median 110
TABLE 9
POSITIVITY OF X-RAYS
IN THE SAMPLE STUDIED
(DEC. 94-AUG. 95)

<table>
<thead>
<tr>
<th>X-RAY FINDINGS</th>
<th>NO. OF PATIENTS</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>+ VE</td>
<td>158</td>
<td>98.75</td>
</tr>
<tr>
<td>- VE</td>
<td>2</td>
<td>1.25</td>
</tr>
<tr>
<td>TOTAL</td>
<td>160</td>
<td>100</td>
</tr>
</tbody>
</table>
DISTRIBUTION OF FORM OF TUBERCULOSIS IN THE STUDIED SAMPLE (DEC.94 - AUG.95)

No. OF PTS

140
120
100
80
60
40
20
0

PRIMARY POST PRIMARY PROGRESSIVE STAGE OF TB
TABLE 10

RADIOLOGICAL DISTRIBUTION OF LYMPH NODES IN PRIMARY TB IN THE SAMPLE STUDIED
(DEC. 94-AUG. 95)

<table>
<thead>
<tr>
<th>LYMPH NODES SITE</th>
<th>NO. OF PATIENTS</th>
<th>PERCENTAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left hilum. (LH)</td>
<td>2</td>
<td>4.7%</td>
</tr>
<tr>
<td>Right hilum (RH)</td>
<td>11</td>
<td>25.6%</td>
</tr>
<tr>
<td>Both Hila</td>
<td>12</td>
<td>27.9%</td>
</tr>
<tr>
<td>Right paratracheal (RPT)</td>
<td>1</td>
<td>2.3%</td>
</tr>
<tr>
<td>Bilateral Paratracheal</td>
<td>1</td>
<td>2.3%</td>
</tr>
<tr>
<td>RPT+LPT+LH</td>
<td>1</td>
<td>2.3%</td>
</tr>
<tr>
<td>RPT+LPT+RH+LH</td>
<td>1</td>
<td>2.3%</td>
</tr>
<tr>
<td>RPT+RH</td>
<td>4</td>
<td>9.3%</td>
</tr>
<tr>
<td>RPT+RH+LH</td>
<td>10</td>
<td>23.3%</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>43</strong></td>
<td><strong>100%</strong></td>
</tr>
</tbody>
</table>
### TABLE 11

**RADIOLOGICAL DISTRIBUTION OF PARENCHYMAL DISEASE IN POST PRIMARY TYPE IN THE SAMPLE STUDIED**
*(DEC. 94 - AUG. 95)*

<table>
<thead>
<tr>
<th>SITE</th>
<th>RIGHT SIDE</th>
<th>LEFT SIDE</th>
<th>BOTH SIDES</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Classical upper or middle zone disease</td>
<td>14 (12%)</td>
<td>20 (17%)</td>
<td>71 (60.6%)</td>
<td>105 (90%)</td>
</tr>
<tr>
<td>Lower zone disease</td>
<td>4 (3.4%)</td>
<td>3 (2.5%)</td>
<td>5 (4.2%)</td>
<td>12 (10%)</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>18 (15.4%)</strong></td>
<td><strong>23 (19.5%)</strong></td>
<td><strong>76 (64.8%)</strong></td>
<td><strong>117 (100%)</strong></td>
</tr>
</tbody>
</table>
# TABLE 12

**RADIOLOGICAL DISTRIBUTION OF PARENCHYMAL LESIONS IN PRIMARY TYPE IN THE STUDIED SAMPLE (DEC. 94 - AUG. 95)**

<table>
<thead>
<tr>
<th>SITE</th>
<th>RIGHT SIDE</th>
<th>LEFT SIDE</th>
<th>BOTH SIDES</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper + Middle Zone</td>
<td>6</td>
<td>5</td>
<td>2</td>
<td>13 (54%)</td>
</tr>
<tr>
<td>Lower Zone</td>
<td>5</td>
<td>5</td>
<td>1</td>
<td>11 (46%)</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>11</strong></td>
<td><strong>10</strong></td>
<td><strong>3</strong></td>
<td><strong>24 (100%)</strong></td>
</tr>
</tbody>
</table>
TABLE 13

PATTERN OF PARENCHYMAL DISEASE
OF THE CHEST X-RAYS
IN THE SAMPLE STUDIED
(DEC.94 - AUG.95)

<table>
<thead>
<tr>
<th>PARENCHYMAL LESION</th>
<th>NO. OF PATIENTS WITH PRIMARY DISEASE</th>
<th>NO. OF PATIENTS WITH PROGRESSIVE DISEASE</th>
<th>NO. OF PATIENTS WITH POST PRIMARY DISEASE</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consolidation</td>
<td>8 (33%)</td>
<td>6 (33%)</td>
<td>44 (38%)</td>
<td>58 (37%)</td>
</tr>
<tr>
<td>Collapse</td>
<td>2 (8%)</td>
<td>3 (11%)</td>
<td>17 (15%)</td>
<td>22 (14%)</td>
</tr>
<tr>
<td>Nodular Shadowing</td>
<td>4 (16%)</td>
<td>8 (44%)</td>
<td>40 (35%)</td>
<td>52 (32%)</td>
</tr>
<tr>
<td>Exudative</td>
<td>10 (41%)</td>
<td>13 (71%)</td>
<td>52 (45%)</td>
<td>75 (47%)</td>
</tr>
<tr>
<td>Fibroproductive</td>
<td>2 (8%)</td>
<td>8 (44%)</td>
<td>81 (69%)</td>
<td>91 (57%)</td>
</tr>
<tr>
<td>Cavitation</td>
<td>7 (29%)</td>
<td>15 (83%)</td>
<td>103 (88%)</td>
<td>125 (78%)</td>
</tr>
<tr>
<td>Bronchiectasis</td>
<td>0</td>
<td>3 (17%)</td>
<td>35 (30%)</td>
<td>38 (24%)</td>
</tr>
<tr>
<td>Bullae</td>
<td>0</td>
<td>0</td>
<td>15 (13%)</td>
<td>15 (9%)</td>
</tr>
<tr>
<td>Calcification</td>
<td>3 (25%)</td>
<td>1 (8%)</td>
<td>7 (6%)</td>
<td>11 (14%)</td>
</tr>
<tr>
<td>Miliary Shadowing</td>
<td>1 (4%)</td>
<td>0</td>
<td>1 (0.9%)</td>
<td>2 (1.3%)</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>24</strong></td>
<td><strong>19</strong></td>
<td><strong>117</strong></td>
<td><strong>160</strong></td>
</tr>
<tr>
<td>CAVITY CHARACTER</td>
<td>NUMBER</td>
<td>%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-------------------</td>
<td>--------</td>
<td>----</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thick-walled</td>
<td>76</td>
<td>61</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thin walled</td>
<td>49</td>
<td>49</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fluid - level</td>
<td>17</td>
<td>13</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### TABLE 15

**PLEURAL INVOLVEMENT IN THE SAMPLE STUDIED**

*(DEC. 94 - AUG. 95)*

<table>
<thead>
<tr>
<th>TYPE OF INVOLVEMENT</th>
<th>RIGHT SIDE NO. OF PATIENTS</th>
<th>LEFT SIDE NO. OF PATIENTS</th>
<th>BOTH SIDES</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Effusion (post-primary)</td>
<td>7</td>
<td>6</td>
<td>0</td>
<td>13</td>
</tr>
<tr>
<td>Effusion (Primary)</td>
<td>5</td>
<td>4</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>Thickening</td>
<td>22</td>
<td>27</td>
<td>5</td>
<td>54</td>
</tr>
<tr>
<td>Calcification</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>EXTENT OF PARENCHYMAL DISEASE</td>
<td>NO. OF PATIENTS</td>
<td>%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----------------------------</td>
<td>----------------</td>
<td>-----</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Minimal</td>
<td>17</td>
<td>10%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>95</td>
<td>60%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Far advanced</td>
<td>48</td>
<td>30%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TOTAL</td>
<td>160</td>
<td>100%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
EXTENT OF PARANCHYMAL DISEASE IN THE STUDIED SAMPLE (DEC. 94 - AUG. 95)

No. OF PTS

EXTENT OF PARANCHYMAL DIS.

MINIMAL  MODERATE  FAR-ADVANCED

100  90  80  70  60  50  40  30  20  10  0
TABLE 17

CORRELATION BETWEEN DEGREE OF ILLNESS & DISEASE EXTENT IN THE CHEST X-RAY OF THE SAMPLE STUDIED (DEC. 94 - AUG. 95)

<table>
<thead>
<tr>
<th>GENERAL CONDITION</th>
<th>FAR ADVANCED</th>
<th>NOT FAR ADVANCED</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>VERY-ILL</td>
<td>26</td>
<td>26</td>
<td>52</td>
</tr>
<tr>
<td>NOT VERY-ILL</td>
<td>21</td>
<td>87</td>
<td>108</td>
</tr>
<tr>
<td>TOTAL</td>
<td>47</td>
<td>113</td>
<td>160</td>
</tr>
</tbody>
</table>

95% Confidence Limit
Chisquare = 16.55
P-Value = 0.000047
CHAPTER V:

DISCUSSION
DISCUSSION:

The results obtained from the study were represented in the previous chapter.

SEX & AGE: -

The number of male patients were 117 out of 160 total patients. Thus males represented 73% of all patients. Male preponderance was noticed in similar studies in Sudan & elsewhere.\(^{(29,229)}\)

Most of the patients were in the young age group 15-35 (76%) with mean of 31 years. As has already been mentioned tuberculosis in developing countries occurs most frequently in the young age group of productive mature\(^{(68)}\) who did not acquire natural immunity against the disease. Young males represent the active sector of the community who move around especially from rural to urban areas & thus get more in contact with the disease. They may live in big cities seeking employment, education etc... & usually live in crowded common lodging hostels, most probably with poor housing & under-nutrition conditions which render them easy victims to the disease. Most probably such young males get exposed to the infection for the first time during their lives in such situations, especially if they come from virgin communities where the prevalence of the disease is low. The risk of direct progression from simple infection to overt disease varies with the age when first contracting the infection. It is greatest when infection begins in the first years of life & next in adolescence.\(^{(69)}\)

However, in developed countries, TB used to affect predominantly the young people in the community, but with development of natural resistance against the disease, the age & sex incidence has changed. Twenty years ago, 80% of notifications were of patients under 45 years of age, but nowadays 60% are over that age, males
predominating. The high incidence of active pulmonary tuberculosis in this old group may be explained partly by the fact that they represent the survivors of a child population which was heavily infected early in the twentieth century & which is now showing endogenous reactivation of the disease. Thus the preponderance amongst the young age group reflects the high prevalence rate of the disease in the community. The age distribution of the disease in the community is important in cohort studies & is used to evaluate the overall tuberculosis situation in a country.

RESIDENCE:
Most of the patients live either in Khartoum or Omdurman provinces & this just reflects the catchment area of the hospitals in which the study has been conducted. Some of the patients live outside Khartoum (7%) & came only to seek treatment for their conditions. However, it is worth mentioning that most of the patients lived in the poorer parts of these towns.

ORIGIN:
Many patients were collected from Abu Anja hospital which lies in Omdurman province. To the west of this hospital are the premises the displaced westerners who moved to Omdurman during the time of draught & dissertation & settled in relatively unfavorable conditions. The next major group are those coming from the South also moving north escaping the war in their homelands. Few patients also came from Ethiopia & are among refugees entering Sudan escaping from the war in their countries. This stresses the fact that tuberculosis problem is aggravated by the war conditions.

The origin of these patients gives a clue as to the ethnic variations which might affect the development of the disease, as in Cummins's virgin soil theory. This is divided
into two parts, firstly: that primitive tribes are highly susceptible to TB, because in the absence of tubercle bacilli they have not been obliged to protect themselves against it, secondly that people living in more commercial or industrial communities are highly protected against TB, because being in regular contact with the bacillus, they have developed protective immunity against it.

OCCUPATION :-

The majority of the patients came from the low socio-economic groups of population, which was expected.

We see also that students occupy a considerable percentage of the sample. Since young age groups are the ones mostly affected we expect them to be students. Nevertheless most of those at school or university age has left the study field & became labourers or such to earn their living in view of the poor socioeconomic conditions in which they survive. Some patients (11%) are even not occupied.

The epidemiological distribution of the disease in this sample is in keeping with the general pattern for developing countries with high disease prevalence. Such distribution highlights the high risk groups, which is important for control programmes in case-finding.

PRESENTING COMPLAINTS :-

The major presenting symptom in nearly all patients was cough. By the WHO recommendation major suspects for pulmonary tuberculosis are patients complaining of cough for more than three weeks.

In Zaki’s study patients presenting with cough amounted to 97% & 81% in other studies. This cough was usually associated with sputum expectoration & dry cough was only noticed in 8% of patients. 28% admitted recognizing haemoptasis as
part of the sputum. Massive haemoptasis was not encountered in any of the patients, as all the patients were newly diagnosed cases and many of them were uncomplicated. The general symptoms complained of were consistent with the general literature,

10 patients, (16%) also complained of non classical symptoms such as headache, lower limb weakness & hoarseness of voice which is usually associated with tuberculous tracheolaryngitis. All these may indicate extrapulmonary tuberculous involvement.

From this we conclude that the cardinal complaints in our patients were productive cough, chest pain, fever & weight loss.
DURATION OF SYMPTOMS:

Table 1 showed that only 17.5% of patients presented within a month of starting their illness & that more than half presented after 2 months, with a mean period of 4 months. This reflects the chronicity of the disease. Most of the patients ignore the disease, thinking of it as just mild cigarette cough or due to common cold & go on taking simple medicines or antibiotics & when all these are no good, they seek proper medical care. Moreover, finding no specific signs of the disease at an early stage, some medical practitioners may treat them as cases of pneumonia, flue, etc... & when later on specific signs appear they refer them to specialized hospitals. The mean clinical evolution time before diagnosis was made was found to be 3 months in some studies. 

SOCIAL HABITS:

28% of the study sample were smokers. We can not tell whether this is a high or low percentage because we do not have an accurate statistics of number of smokers in a matched age & sex group. But it is known that smoking & alcohol intake do assist the development of tuberculosis especially if accompanied by the socioeconomic conditions mentioned earlier. Tuberculous alcoholic patients as opposed to non-alcoholic showed more frequent & prevalent larger cavities with non-typical radiological distribution.

We have realized in this study also that smokers although presenting in a fairly well general condition showed more far-advanced changes in their chest x-rays compared with non-smokers. i.e smokers did not show a parallel correlation between their general condition & their chest x-ray appearances.
Far advanced changes among smokers was 34% while only 27% among non-smokers, there was no significant association between smoking as such & developing far advanced disease changes in the xary. But nevertheless, there was significant association between heavy smoking & development of far advanced lesions in the chest xary. Therefore, we can predict that heavy smoking may assist progression of the disease within the lung parenchyma. Another suggested cause was the high incidence of alcoholics among smokers.\(^3\)
PHYSICAL SIGNS:

GENERAL EXAMINATION:-
One third of the patients presented in a relatively ill condition, this may reflect the chronicity of the condition & the delay in presenting to the medical services. About half the patients showed evidence of muscle wasting & this can be explained by the fact that the disease already has a predilection for persons with low body mass index. On top of this, the disease causes anorexia & vomiting in some patients & this may lead to further wasting. This also applies to the pallor seen in 37% of the patients. Pallor may be also due to the associated anemia, or to the hypovolaemia initiated by the vomiting & anorexia.

Clubbing was seen in 9 patients (5%). Clubbing is associated mainly with chronic suppurative lung diseases, such as bronchiectasis & empyema or lung malignancy e.g. Ca bronchus. It was not described as a feature of tuberculosis, unless complicated or associated with such conditions.

Generalized lymphadenopathy is not generally seen in adults with tuberculosis & is mainly seen in patients with primary tuberculosis as part of the general dissemination of the disease. 8 patients (5%) only in the present series showed this sign.

OTHER SYSTEMS:

Of systems affected other than the pulmonary, 3 patients showed lower limb weakness, representing vertebral column involvement by the disease process. From
The time table of tuberculosis it was shown that bone usually is concomitantly affected as post primary lung disease.

2 patients were pregnant at the time of presentation.

Another 2 patients showed hepatosplenomegaly. This may be due to the associated anemia or due to concomitant tropical infections.

Disseminated tuberculosis is known to cause hepatosplenomegaly but these two patients particularly were not suffering from disseminated TB.

On the skin manifestations; 2 patients showed scrofuloderma on their neck skin & this may indicate that they had primary tuberculous lymphadenitis earlier in their lives.

2 patients also showed vitiligo on their skin which the author thinks was a coincident finding.

One patient had impetigo contagiousum & another Tinea- versicolor. Such bacterial & fungal infections are expected in tuberculous patients who are already immunocompromised & live in rather poor non hygienic conditions.

One patient only had essential hypertension. A high percentage of hypertensives was not expected, since the age group for essential hypertension is older than that seen in the present study. Furthermore, the risk factors for hypertension such as obesity, hyperlipidaemia etc ... were not prevalent in the present study sample.

CHEST SIGNS :

16 patients (10%) showed no chest signs on physical examination. It is well known that some patients may not show any physical signs, especially in milliary disease & early non-complicated pulmonary tuberculosis.

The remainder 90% of patients showed different patterns of chest signs, the most common of which was consolidation (69%). Many cases when examined may show
clinical signs of consolidation, but on viewing their chest x-rays other types of parenchymal lesion may be seen. This explains the discrepancy between this number (110, 69%) here & patients showing consolidation radiologically (table 8) who were only 58 patients (37%).

Such differences were also noticed in other physical signs, which suggests that clinical examination may underestimate the true extent of the pathology in the affected person.
INVESTIGATIONS:

SPUTUM:

90 patients (57%) showed positive sputum smear result. This is a fairly reasonable result compared with studies elsewhere where sputum positivity ranged from 44% to 88% \(^{(12)}\). The variations in sputum smear results may be due to: (1) extent of the disease \(^{(12)}\), (2) technical errors, (3) presence of cavitary disease & (4) number of bacilli excreted per ml of sputum \(^{(6)}\). Zaki in his study in Eastern Sudan \(^{(9)}\) attributed the low sputum positivity (31%) to technical errors & inadequacy of sputum sample collection. Since most of the expected tuberculous patients may have a negative sputum result, other means of diagnosis should be sought to confirm the diagnosis.

MANTOUX TEST:-

Only 147 out of 160 patients had a tuberculosis skin test done. 117 (80%) patients had a significant skin reaction (>10 mm induration) to tuberculin test. The rest 30 (20%) either had a reaction below 10 mm or a totally negative result (anergy) which is usually expected in 8% of patients with post primary tuberculosis \(^{(6)}\). This occurred in patients with overwhelming infection, hypoproteinaemia, associated debilitating diseases, or compromised immunity \(^{(10)}\).
ERYTHROCYTE SEDIMENTATION RATE (ESR):

98% of patients showed a high ESR result & 66% of patients had an ESR in the order of three figures. It has been stated that the ESR is always raised in active tuberculosis.

This suggests that ESR is apparently a good measure of activity but it is not sensitive.

CHEST X-RAYS:

2 patients showed totally clear chest x-rays although they had 3 sputum positive results. These two patients presented with barking productive cough together with hoarseness of voice & one showed physical signs of bronchostenosis on clinical examination. A clear chest x-ray in bacteriologically-proven tuberculous patients was attributed to (1) pure endobronchial involvement, (2) parenchymal lesions may not have developed or are so minimal as not to appear on the chest x-ray.

DISEASE FORM:

The presence of lymph nodes in the chest x-ray film was used as differentiation point between primary and post primary tuberculosis.

43 (27%) of cases showed radiologically-demonstrable thoracic lymphadenopathy. 24(15%) of which showed associated parenchymal features typical of primary disease. Primary tuberculosis is not common in adults especially in endemic countries, where most of the people in the community would have contracted the infection in their childhood (even if in the sub-clinical form), and later on they develop the reinfection type, which was the commonest form in the sample (73%).
In those who were attacked by the primary infection & their immune responses did not check the disease at an early stage (due to a variety of conditions) the disease progressed to the post-primary or reinfection type & these patients showed radiological features of both types & are thus termed as progressive type. Patients with such features amounted to 12% of the study sample.

Comparable results were seen in similar studies.  

\[ \text{(Equation)} \]
LYMPHADENOPATHY:

Pure hilar lymphadenopathy was the commonest (25, 60%) either unilateral or bilateral. If unilateral lymphadenopathy occurred, the right side was more commonly affected (11 versus 2) than the left side. Pure paratracheal lymphadenopathy was not common (2, 4.6%), but it was more commonly affected together with hilar nodes and here again it was the right group which was predominantly affected. This right-sided and hilar predominance were noted also by others [650].

PARENCHYMAL LESIONS:

Primary Disease:

Consolidation and exudative processes were the commonest parenchymal lesions (18 cases, 74%) in primary disease. Some contained cavities inside the consolidation mass (7 patients, 29%). Lobar collapse was seen in 2 cases (8%) most probably due to compression by lymph nodes on the major bronchi. Nodular shadowing suggested labor spread via an attacked bronchus. Rarely signs of fibrotic process appeared in the primary lesion (2 cases, 8%). Miliary shadowing occurred in one patient directly from the primary disease. Calcified lesions were seen in 3 cases (25%) most probably as part of healing of the primary complex. Brochiectasis and bullae formation were not seen in primary disease, and are usually found in the post primary form [650].

Progressive:

The progressive type showed similar distribution of results to that of primary disease, but with more prevalence of fibrosis (44% versus 8%), cavitation (83% vs 29%) and
nodular shadowing (44% vs 16%). Here bronchiectasis appeared as the fibrotic process usually invites its formation. Similar results were seen in other studies.

**Post Primary:**

In the post primary disease, fibro-cavitatory changes were the principle parenchymal lesions, and together with the preceding exudative type, they constituted the majority (98%) of the parenchymal reactions. Here again bronchiectasis and bullae formation were more commonly seen as they are usually in close accompaniment with fibrosis. Consolidation as such is not usually seen in post primary disease, but in this group we saw its frequency to be similar to that seen in the primary group. These may represent cases of tuberculous pneumonia or cases of primary TB in whom lymphadenopathy was not quite evident radiologically. This fact is augmented by the high percentage of lobar or segmental collapse (15%), which is not usually common in post primary TB. They may also represent the presence of endobronchial TB which led to cicatrization of the major bronchi, and the formation of the collapse-consolidation complex.

Calcification was more commonly seen in the post primary group because of the chronicity of the condition, and may be due to some delay in the presentation of patients to the medical services. Calcification may suggest early signs of healing. Comparing with the literature we find that we have higher incidence of cavitation (88% in our series compared to 45% and 53%). Again due to chronicity and delay in seeking treatment.
76 (61%) of the cavities were thick-walled and 17 (13%) showed fluid levels; both of these criteria were taken as signs of activity\(^{(13)}\). Fluid levels were seen in 21% of cavities in a study in Nigeria\(^{(132)}\).

Presence of cavitation was postulated to give a higher yield of sputum positivity\(^{(26)}\). We see from the results that in our sample \(P = 0.000037\), and thus agreeing with the known fact, i.e. when cavitation is present, it is more likely to have a positive sputum result.

ANATOMICAL DISTRIBUTION OF PARENCHYMAL LESIONS:

Primary disease:

The Upper lobes were slightly more frequently involved than the lower although there were no significant differences between left & right lungs. This tendency to predominant involvement of the upper lobes was noted by others\(^{(133)}\).

Post Primary Disease:

Post-primary disease was reported to affect predominantly the apical and posterior segments in 85% and superior segment of the lower lobe in 5%\(^{(133)}\).

On the frontal chest radiograph, the superior segment of the lower lobe is usually seen in the middle zone, rendering lower lung field lesions rather rare (5%).

In the present study upper and mid zone involvement were seen in 90% of cases. This is more or less consistent with other studies\(^{(133,33)}\). Bilateral lung involvement was seen in 60% of cases and was thus commoner than unilateral disease.

However, in the present study unilateral involvement of the left lung was more commonly seen than in the right lung.
Lower-Lung Field TB:

While lower zone disease was reported in about 5% of cases, in the present study, it was seen in 10% of cases. This may be explained by variations in different ethnic groups, and other associated factors, e.g. alcohol consumption, smoking, immune-deficiency, etc. Lower lung field TB was postulated to be commoner in women, negroes, and diabetics.

PLEURAL DISEASE:

Pleural effusion:

22 patients had plural effusion all being unilateral, & the right side was slightly more affected than the left side, (12 versus 10). Nearly more than half (59%) were due to post primary disease & these constituted 13% of all patients with reinfection TB. In literature, 18% of patients with post primary disease was shown to have pleural effusion. While in patients with primary disease pleural effusion occurred in 37% of patients.

Pleural thickening:

This was quite common & was seen in 54 patients (34%) all TB patients. 43 of these (i.e. 80%) were in post primary type & only 2 (1%) were in the primary type.

Pleural calcification:

It was rare & seen in only one patient. This was because it is rather a late event in the process of healing of TB & all the patients in the study were newly diagnosed ones & took no treatment before.
EXTENT OF PARENCHYMAL DISEASE:

The commonest group were those with moderate extent of parenchymal lesions (60%). While minimal lesions amounted to 11% of cases. Far-advanced lesions were found in 29% of cases. This may indicate the delay in initiating treatment. Also as mentioned earlier the far-advanced type was more commonly seen in smokers & alcoholics.

The disease grew unchecked in weak, malnourished debilitated patients & many of the candidates in the present study were belonging to this group. A similar distribution was noted in other studies.

It was realized that there was significant association (P = 0.00005) between patients presenting in a generally ill condition & having far-advanced lesions in the chest x-ray.

Thus x-ray findings paralleled the general clinical condition of the patients.
CHAPTER VI:

CONCLUSIONS & RECOMMENDATIONS
CONCLUSIONS & RECOMMENDATIONS

From this study we concluded that:

1. Pulmonary tuberculosis is a disease affecting predominantly males of the young productive age group.
2. The disease affected mainly those living in unfavorable socio-economic conditions particularly those displaced by war & other environmental disasters.
3. The main presenting symptoms were productive cough chest pain, breathlessness in the specific symptoms. In the general symptoms fever, weight less & malaise were the commonest.
4. Patients present to the medical services rather late. Mean period of duration of illness before initiating treatment was 4 months.
5. There was 28% prevalence of smokers, 20% of alcoholics in the study sample. There was a significant association between heavy smoking & development of far-advanced lesions in the chest x-ray.
6. One third of the patients presented in a quite ill condition & nearly half were wasted. Finger-clubbing was not of salient feature of TB.
7. Physical examination alone may underestimate the actual extent of the disease. There may be no findings on clinical examination & this should not preclude further assessment of the patient, looking for TB by other means of investigations.
8. 57% of patients were sputum positive. & there is a high percentage of tuberculin test positivity (80%). There was a significant association between presence of cavities & sputum positivity.
9. ESR is a sensitive test for tuberculosis activity, although not specific. Nearly 2/3 rds of the patients had an ESR for 3 figures.

10. 3/4 of the patients in the study sample had typical post primary TB. 15% had primary & 12% had features of both (progressive). Therefore, adults are mainly affected by the post-primary type.

11. In the primary type bilateral hilar lymphadenopathy was the commonest followed by unilateral Rt hilar. Paratracheal alone was rare & mainly associated with hilar lymphadenopathy.

12. In the primary disease exudation & consolidation were the main parenchymal lesions. The upper lobes were slightly more frequently involved than the lower, although there were no significant differences between left & right lungs.

13. In post primary disease exudative & fibro-cavitory lesions were the salient features in the chest x-rays (98%). The distribution of the lesions was mainly of the classical upper & midzonal lesions. In the present study a slightly higher percentage of lower lung field TB was seen.

14. 14% of all the patients in the sample had pleural effusion, nearly half of which was due to primary & the other half due to post primary type. Pleural thickening was a common finding in pulmonary TB, especially in the post primary type.

15. Xray findings paralleled the general condition of the patient.

16. Only 10% of patients had minimal lesions on the xray & the remainder 90% had either moderate (60%) or far-advanced (30%) indicating the chronicity of the disease & the late presentation of the patients.
17. X-rays are effective means in diagnosis of pulmonary TB & are very important in delineating extent & complications of the disease.
RECOMMENDATIONS:

1. Health education of the community, so that any patient who complains of cough for more than three weeks should present to the medical services immediately & should have sputum examination done properly.

2. Any such patient having sputum negative examination should have an xray done, if possible, immediately to exclude possible tuberculous lesions, & another xary after any given course of antibiotics to monitor response of lesions & try to differentiate from other infections.

3. If the patient had sputum positive result & a chest xary should be preferably be done initially to make a base-line for the original lesions. Subsequent xrays should be done one at 6 months & one at the end of treatment to monitor response to chemotherapy & detect complications as early as possible.

4. Xrays should be done properly & with adequate radiographic factors, so as not to miss early or minimal lesions.

5. Tuberculous patients should be advised to stop smoking as this may worsen the condition of their lungs.

6. Screening high risk groups e.g. in displaced camps may be done for active case finding.

7. Further studies are recommended to: a) follow up patients with pulmonary TB radiologically & monitor progress of the disease, & b) to verify the relationship between the different risk factors including smoking etc.... & the development of pulmonary TB.
CHAPTER VII :

REFERENCES
REFERENCES:


13. Fraser RG, Pare JAP (1970) *Diagnosis of diseases of the chest*. WB SAUNDERS. PHILADELPHIA, USA.


44. Illingworth RS (1956) Tubercles of the choroid. *Arch Dis Childh,* **31**:467.


88. Leung AN, Muller NL, Pineda PR, FitzGerald JM (1992) Primary tuberculo-
89. Donald PR, Ball JB, Burger PJ, (1985) Bacteriologically-confirmed pul-
monary tuberculosis in childhood: Clinical & radiological features. *S Afr
Radiol, 33*:430.
N Amer, 1*:411.
95. Weber, Alfred L, Bird KT, Janower ML (1968) Primary TB in childhood with
particular changes affecting the tracheo-bronchial tree. *Amer J Roentgenol,
103*:123.
96. Frostad, Simon (1959) Segmental atelectasis in children with primary
Livingstone, London, UK.
Pulmonary tuberculoma and indications for surgery: radiographic & clinico-
100. Adler, Hugo (1959) Phthisiogenetic studies by means of tomography in cases
of localized pulmonary tuberculosis in adults. *Acta Tuberc Scand Suppl,
47*:13.
101. Farmon DP, Speir WA, (1986) Initial roentgenographic manifestations of
bacteriologically proven Mycobacterium TB: Typical or atypical ?. *Chest ,
89*:75.
unusual roetgen manifestations of childhood tuberculosis. *Clin Imaging,
18*:149.


91
CLINICO-RADIOLOGICAL PATTERN OF PULMONARY TUBERCULOSIS
IN NTP CLINICS IN KHARTOUM

<table>
<thead>
<tr>
<th>Patient Number:</th>
<th>Date:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Name:</td>
<td>Sex:</td>
</tr>
<tr>
<td>Age:</td>
<td>Residence:</td>
</tr>
<tr>
<td>Origin:</td>
<td>Occupation:</td>
</tr>
</tbody>
</table>

**Presenting Complaints:**
- Cough
- Haemoptysis
- Breathlessness
- Sweating
- Weight loss

<table>
<thead>
<tr>
<th>Sputum</th>
<th>Chest Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever</td>
<td>Malaise</td>
</tr>
<tr>
<td>Others (specify)</td>
<td></td>
</tr>
</tbody>
</table>

**Duration of Symptoms:**

**Past History:**
- Tuberculosis:...
- Diabetes Mellitus:...
- Others (specify):...

**Social History:**
- Smoking: No. per day...
- Alcohol: Consumption per day...

**Drug History:**
- Steroids: Others (specify)...

**Examination Findings:**

(i) General:
- Wasting: Pallor:...
- Clubbing: Lymphadenopathy:...
- Site: Others (specify):...
(ii) Chest

* Signs: Site
  a) Consolidation: ..................
  b) Fibrosis: ..................
  c) Cavitation: .................
  d) Collapse: ...................
  e) Pleural effusion: ...........
  f) Pneumothorax: .............

(iii) CVS:

(iv) Abdomen:

(v) CNS:

(vi) Skin:

(vii) Others (specify):

INVESTIGATIONS:

* Sputum for AFB: .......... No of times: ....
  Date: ..................
* Mono tests: Grades ....
* Haemoglobin: ESR:
* X-Ray Done: PA, Lateral, Apical lordotic.
  Tomography: Others (specify): ..
* CXR findings: tuberculosis: Post primary: Date: ....
  Site: Size
  - Consolidation: ..................
  - Nodular Shadowing: ..............
  - Local exudative: ................
  - Local fibro productive: ..........
  - Cavitation: .....................
  - Miliary spread: .................
  - Bronchietasis: ..................
  - Lymph node involvement: ........
  - Pleural involvement: Thickening: effusion: Calci
  - Extent of parenchymal disease: Minimal: Moderate: Far advanced: ..................
1. Primary pulmonary tuberculosis
   Prominent lymphadenopathy, but minimal parenchymal lesions.

2. Same as (1), two weeks later
   Parenchymal lesions now more prominent in right lower zone.
3. Primary right-sided pleural effusion

4. Primary Miliary tuberculosis
5. Exudative lesions in right upper and left middle zones. Thick-walled cavities also seen in the (R) upper zone.

6. Fibro-productive lesions in left upper and mid zones (slight loss of volume). The lesions are smaller, more dense and defined than in the exudative phase.

8. Nodular shadowing in both mid zones denoting bronchogenic spread.
9. Fibro-cavitary disease, with thick-walled cavities and bronchiectetic changes in left upper zone.

10. Bulla formation in right upper zone, compressing the lung tissue beneath.
11. Multiple modular shadows, simulating cannon-ball secondaries.