REVIEW OF LITERATURE
"Tuberculosis may have been the first-born of the mother of pestilence and disease. It evidently existed in the pre-historic time on the planes of Gangas as manifested by pathological conditions found in preserved bones and joints", (quoted from Tuberculosis - C. Arthur Myers). From the dawn of history, on through the centuries, a glimpse is caught here and thereof the presence of this disease in people and animals. In the long history and amid the large literature of tuberculosis, certain dates and classic publications are outstanding, signifying major advances in knowledge and understanding of this disease.

In the Rig Veda (6000 B.C.) there are observations concerning the behaviour of the tuberculosis in people. This is also true of Egyptian papyri and ancient Chinese writings. Evidence of Pott's disease dates back to Neolithic period (5000 B.C.). A number of bodies mummified between 2000 and 1000 B.C. have been found with evidence of tuberculosis of bones and joints, indicate that man has been affected with tuberculosis since ages. Charak and Sushruta (500 B.C.) had mentioned about the disease in their writings. Aristotle (450 B.C.) recognised its contagious nature.
Hippocrates (470 - 376 B.C.) named it "Phthisis" for the first time, meaning thereby to waste away. It has been given other names such as "captain of the man's death" and the great white plague". Spes Phthisis" referred to the eternal hope of the phthisised person, even though in the last stage of the disease. The word "tuberculosis" was coined for the first time by Scholein (1839). Kortum (1789) apparently, was the first to attempt to transmit the disease from human to human. Klenche (1843) transferred tubercles to a rabbit but apparently did not clearly understand the significance of his experiment.

Farrot (1876) in a short impersonal account, reported his observations on the relationship of the pulmonary tuberculosis in childhood to tuberculosis, of the tracheo bronchial glands. He thought that the tracheo bronchial glands were the mirrors of the lungs and conversely there is no bronchial adenopathy which does not have it's origin in the lungs. But Laennac said that the bronchial glands were often tuberculous at the same time as the lungs were healthy.

Robert Koch announced the discovery of the causative organism Bacillus tuberculosis, on 4th April.
1882. He isolated the bacterium, grew it on artificial media, inoculated into the animals and produced the same disease. Koch later noted the difference in the reaction complex produced by the inoculation of the organism in a healthy animal "previously uninfected" and one in a "previously infected". This is known as 'Koch's phenomenon'. It is the basis of the concept of 'Primary' and reinfection tuberculosis.

In 1890, Koch announced a curative agent against tuberculosis and named it as 'tuberculin'. He noted the symptoms (local and general) produced by a subcutaneous injection in a tuberculous patient, but he paid no importance to the reaction produced at the site of injection. Epstein and Escherich for the first time mentioned diagnostic importance of the reaction produced at the site of injection. With the advent of the phenomenon of allergy it was established that tuberculin allergy means an individual who had experienced a specific body change through infection with the living or dead tubercle bacilli. Soon after this, various tests came into picture for detecting the tuberculous sensitivity.
In 1912, thirteen years after the discovery of tubercle bacillus came Ghon's classic monograph, "Der Primair Lungenherd bei der Tuberculose der Kinder" in which the primary lung lesions and the relationship of the glands were described. Ghon also mentioned extrapulmonary primary lesions on skin and on mucus membranes where the same relationship of primary focus and regional adenitis was demonstrated and he confirmed Parrot's work.

In 1916, Ranke postulated three stages in tuberculous infection associated with different degrees of allergy; the first that of primary infection; the second that of dissemination and hyper-sensitivity and a tendency to exudative lesions, while the third was the stage of increased resistance and lowered sensitivity and localisation of infection. This broadly speaking, indicates the stages of tuberculosis after primary infection.

The next masterly study was the series of papers by Rich and McCordock (1929) in which they considered the relationship of host resistance, virulence of the organism, allergy and the size of the infecting dose in determining the character of the resultant lesions. They advanced and clarified concept of the differences of the progressive adult and childhood tuberculosis.
In France, the "Grancher" system of care for the separation and protection of infants in contact with infective parents was evolved from 1907, and in 1920 B.C.G. vaccine was given its first clinical trials, the results which caused so much controversy that the acceptance of its usefulness was delayed in many countries.

Wallgren and his colleagues (1925) published many studies of tuberculosis in childhood. During this time they also established suspicion of children in contact with infection and used B.C.G. vaccination as a method of protection. They emphasised the relationship of erythema nodosum and primary infection.

Rich (1944) published a book "The Pathogenesis of Tuberculosis" in which he has described a critical survey of contemporary knowledge concerning the relationship of tubercule bacillus and human host and the basis factors which cause variations in that relationship. Brailey and Hardy (1958) published a clinical and epidemiological study of 437 White and 492 Negro children admitted for observation between 1928 and 1944 and supervised until 1950. This dealt with the prognosis of the primary infection and with the risks of developing progressive adult type pulmonary disease before the introduction of the chemo-therapy.
Hardy (1958) has noted "Primary tuberculosis" is the result of primary tuberculous infection in an individual often a child, though sometimes an adult, who having no previous experience with this type of infection has, acquired neither resistance nor previous hypersensitivity towards it. He has further noted "very rarely a second primary infection may occur in an individual, who has lost the immune responses, resulting from the first infection". Mead (1959) has clearly noted "the term primary tuberculosis should be limited to the lesion which arises from the infection that takes place prior to the development of tuberculin sensitivity".

In South India (Saidapet), 30,000 population was tuberculin tested and prevalence of the tuberculosis was found to be 2.3% (Benjamin et al, 1939). A tuberculin survey was conducted in Uttar Pradesh (1949-51) under the auspices of W.H.O. This revealed a higher reaction rate in urban population as compared with rural population. In industrial cities 73% of people were tuberculin positive by 15 years of age.

Sarin et al (1957) carried out histologic studies of hepatic lesions in 100 cases of pulmonary tuberculosis in which they reported tubercle in 15, focal cellular collection in 35, focal necrosis in 47, fatty metamorphosis in 27 and reticulo-endothelial hyperplasia in 57 cases.
Bently and Grybowski (1954) reported that out of 317 children with uncomplicated primary complex 115 had a paranchymal focus and 202 had only lymphadenopathy.

In 1958 Walker, in a study of 538 children, primary complex was seen in 280 cases (mottling with hilar adenitis), primary cavitation in 5, bronchopneumonia in 3 and miliary tuberculosis in 60 cases.

Manchanda et. al. (1966) reported their observation on 225 children, 43 were found to have primary complex, 86 were found to have lymphadenopathy, collapse were in 4 cases, consolidation in 11 cases, bronchopneumonia in 2 cases, pleural effusion in 5 cases, middle lobe syndrome in 2 and cavitation formation in one case.

Dinglay (1966) conducted a study and found that out of 500 cases of primary tuberculosis 260 had glandular enlargement and 28 cases had primary complex, consolidation in 170 cases, collapse in 37 cases, pleural effusion in 56 cases and calcification in 5 cases.

In 1976, A. Govindan and R. Marmada conducted a study on 1500 children, clinically suspected primary tuberculosis. There detailed history was taken. All the children were tested for montoux positivity and their chest X-ray was done.
The various features observed on the chest roentgenograms were numbered and tabulated in table as follows -

**TABLE**

**Showing various features of primary tuberculosis**

*in Chest X-ray & various Roentgen manifestation of primary complex*

1. Primary focus with perifocal inflammation
2. Lymphangitis
3. Hilar adenitis
4. Mediastinal adenitis
5. Inter lobar pleural opacification
6. Primary focus calcification
7. Calcification of lymphatics
8. Glandular calcification
9. Pleural effusion
10. Pleural thickening
11. Pleural calcification
12. Atilectasis
13. Bronchiectasis
14. Miliary tuberculosis
15. Bronchopneumonia
16. Associated carries of thoracic spine
17. Tubercular pneumonia
18. Obstructive emphysema
19. Extra pulmonary calcification
Kotaiah (1958) published his study of primary pulmonary tuberculosis in 195 cases at Visakhapatnam. He observed that highest incidence of tuberculosis in children seem to be in higher age group (10-15 years) than in those published by Western countries and there were only 23 cases (12%) below the age of three year. 98% of his cases were Mantoux positive. In this series, mediastinal lymph node enlargement was seen in 50% cases. Manchanda et al, (1958) reported 215 cases (15.7% of 1366 cases) from Amritsar. They had a history of contact in 30% of the subjects, symptoms were vague but gastrointestinal symptoms were most common. This series had higher incidence in 5 - 10 year age-group.

Udani (1960) in his autopsy report of 100 children between the age of two months and ten years revealed that tuberculosis of various types formed 19.8% of the total cases on clinical side and tuberculosis was major cause of death in 10% of cases, though in 26% of cases, there were various types of tuberculous lesions as associated lesions.

Barucha et al, (1961) reported one case of miliary tuberculosis with granulomatous lesions in the liver. Udani (1962) reported 9 cases with hepatic and splenic lesions in 36 tuberculous children, who were subjected
to autopsy. Three of them had miliary tubercles of liver as a part of generalised miliary tuberculosis, whereas in one case the miliary tubercle in the liver were realised as an abdominal picture of miliary tuberculosis; in others there was a picture of caseating tuberculoma, toxic hepatopathy, cirrhosis and amyloidosis. Balkrishnan and Sharma (1962) reported 6 cases of tuberculosis in whom they studied hepatic lesions.

Prakash et al, (1963) observed 273 tuberculin positive children at Lucknow and found the incidence of 10.8%, 32.6% and 40.7%, among 0-3 years, 3-7 years and 7-12 years age-groups respectively. In their series 35% cases had a positive history of contact and on radiography, 84.2% of the cases showed intra-thoracic abnormalities.

In 1964, Reddi et al studied hepatic lesions in 20 tubercular children and found that 80% (4 out of 5) of miliary granulomatous lesions were seen in cases of miliary tuberculosis, the remaining was seen in tuberculous meningitis and none was seen in other forms of tuberculosis.

In 1966, Ramchandran and Purnayyan from Thanjavur, presented an analytical study of 365 children out of which 321 (87.9%) were P.P.D. positive, but the rest of the cases (44) were also suffering from tuberculosis. They found that
maximum incidence was amongst 0-2 years age-group (46.8%) but only 10% gave the history of contact. In this series, the commonest radiologic finding was mediastinal glandular enlargement, the classic primary complex was seen only in 32 radiographs out of 347.

Classifications: On the basis of the detailed studies of the type and duration of the disease in children and adult, Sekulick (1955) described two types of tubercular processes.

a) Primary tuberculosis: as benign - mostly effecting children.

b) Secondary of reinfection type - mostly affecting the adults.

Primary Tuberculosis:

This is the form of the tuberculous process which develops in an individual who having had no previous exposure with this type of infection has acquired neither resistance nor previous hyper-sensitivity towards it. It is more common in infants and children because they provide a "virgin soil" more frequently than the adults. 'Primary focus' is the structural change or reaction of the tissues brought about by the tubercle bacillus at the side of first recognisable implantations. The term 'Primary adenitis' is
employed for the tuberculous adenitis associat with primary focus. The first affected node is known as 'primary node' and the others as 'satellite node'. The primary focus the lymphangitis and the regional adenitis are known as the primary tuberculous complex. Primary tuberculosis can lead to seriousness and crippling disease in children, it can be fatal and is considered as 'fountain head' of clinical tuberculosis of adult life.

Incidence:

In Vienna, Hamberger and Monti (1909) found that every child before reaching adult life was infected with tuberculosis and it is likely that in all the cities of Europe, the age incidence of the infection at that time was much the same although the comparative incidence in country bred children was less known. The incidence in India has been quite high since long and even today. Ukil (1931) reported it as 11.40% for 0-5 years, 30 percent for 5-10 years and 33.3% among 10-15 years age-groups. Similarly, other surveys done in localised areas have also reported a very high incidence of the disease (Frimodt Moller, 1948; B.C.G. Team, 1951). Chatterjee (1957) and Basu et al (1958) reported moderately high incidence of tuberculous infection in sick children attending the
children hospital and centre. Extensive work done by Frimodt-Moller (1962) at Madanpelle, a rural area in Southern India revealed that 95% of this population react to a dose of 100 T.U. (with reactions of 5 mm. or more) and that young children appeared heavily infected, at the age of 5 years 60% and at 10 years 85% react to 100 T.U. A systemic programme of case finding, X-ray and tuberculin testing and hospitalization of the infectious cases reduced the mortality in that area from 200 to 21 per 100,000 in less than four years duration. Udani (1962) has quoted in his article 'Management of tuberculosis in children that a sample surgery of the population of Bombay city, tuberculous disease was found in 5.5%. The incidence is likely to be higher in the population of low socio-economic groups who live in overcrowded, ill-ventilated one room tenements in the chawls of the city.

However, the incidence is failing in so called better developed countries. Miller (1958) has stated that in 1957 in London, about 2% of children were tuberculin positive in 1st year, 10% at 10 years, 25% at 14 years whereas in 1939 it was 16.6% at 4 years, 26.4% at 8 years and 47.2% at 12 years (Bradshaw). In United States, for
the kindergarten children it has been estimated as 1.5% and 2.6% respectively for White and Negro children (Maha, 1963).

Mode of infection:

There are two main types of primary tuberculous infection, the air borne and the alimentary, and the former is more frequent. It is active in 90% of all infection; in countries with no bovine tuberculosis, the percentage is 100. There is no general agreement as to whether infection by droplets or by dust has the dominant role.

In addition, direct implantation of tubercle bacilli into the skin or mucus membrane may occur in exceptional cases. By congenital tuberculosis is meant a foetal infection passing across placenta and via the umbilical vein, this is a rare phenomenon.

Incubation period:

The end of the incubation period is marked by the appearance of tuberculin sensitivity. The length of pre-allergic period varies considerably in different cases the reasons for this variability include intensity and virulence of the infection, the age of the child and individual variation of reaction. In spontaneous natural infection in man the pre-allergic period has been found
to be not less than 3 weeks and not more than eight weeks and is generally 5-6 weeks (Wallgren, 1941; Waz-Hockert, 1947).

**Tubercle Bacilli and the Host:**

Tubercle bacilli (*Mycobacterium tuberculosis*) are characterised by their capacity to produce the infection or disease known as tuberculosis, in susceptible animals. They are acid fast and an aerobic. There are five types of tubercle bacilli, human, bovine, avian, murine and cold blooded. They are defined by their pathogenicity for different species of animals. *Mycobacterium* contains 25% lipid (dry weight). The polysaccharides of tubercle bacilli have arouse interest by their antigenic activity but the antibodies which have been demonstrated are of little value in diagnosis and have no relationship to immunity. During the growth of tubercle bacilli proteins are liberated which are concerned in tuberculin sensitivity although nothing is known of their function in the organism.

The relationship between the tubercle bacilli and the human host is complex. The outcome of their encounter presenting a wide spectrum of possibilities. There are many examples of nurses caring for patients with advanced open chronic disease over many years, who
never show any clinical or radiological evidence of infection and have a persistently negative tuberculin test. Course of this infection is variable - one person develops only a small limited primary complex which never gives rise to illness, whilst another rapidly develops extensive progressive disease.

**Forms of Primary Tuberculosis:**

It has been estimated that about 95% of the lesions of primary tuberculosis are intra-thoracic, out of which pulmonary form is commonest. The other forms ranking next to pulmonary, in order of frequency, are gastro-intestinal, primary adenitis, tonsils, skin and congenital tuberculosis etc.

**Primary Pulmonary Tuberculosis:**

The lung is the commonest site for primary infection. The primary focus is usually sub-pleural and without any strong predilection for any special segment or lobe. Anatomical distribution with order of frequency are: right upper lobe, right lower lobe, left upper lobe, left lower lobe and finally the middle lobe. This is seen in children without any religion, sex or racial discrimination.
Clinical Features of Primary Pulmonary Tuberculosis:

The manifestations depend on the type of primary disease. In simple primary pulmonary tuberculosis, the cases usually are asymptomatic or show minimal or a non-specific illness. Wallgren (1948) is of the opinion that fever is the most common clinical manifestation of primary tuberculosis infections. Probably all infected children display an increased body temperature, which is in no way characteristic of tuberculosis. The other manifestation like pulmonary pleural effusion, segmental lesions and hematogenous forms are almost always associated with characteristics symptoms in association with the constitutional symptoms i.e. cough anorexia, prostration, malaise, night sweating, loss of body weight, failure to thrive etc. The signs of pulmonary primary infection are largely constitutional and signs or symptoms directly referable to the lungs are uncommon except in young infants and then if pulmonary symptoms are present the condition has usually spread beyond that of the simple primary complex.

Pathogenesis and Course of Primary Complex:

At the site of initial focus, e.g. in the parenchyma of the lung, there is at first an accumulation of polymorphonuclear leukocytes. This reaction is
temporary and is followed by proliferation of epitheloid cells, which surround the tubercle bacilli, creating the typical tubercle formation. The tubercles are usually surrounded by an accumulation of lymphocytes, and giant cells are usually present. Tubercles may remain discrete or may become confluent; central caseous necrosis is commonly present.

In the majority of the people caseation in primary focus rarely exceeds a centimeter in diameter and as the infection is contained by the host, healing begins and capsule develops as fibroblasts and lymphocytes appear at the periphery and collagen fibres are laid down around the focus. The tendency of the primary lesions both in paranchyma and nodes is towards healing in the majority of instances.

The various possibilities are — (i) healing, (ii) indolent lesions persist, (iii) extension of the lesion with progressive destruction of tissues, (iv) bronchial wall erosion with partial or complete occlusion of bronchial lumen with establishment of localised obstructive emphysema or atelectasis and at times with distribution of tubercle bacilli to other parts of the lung and establishment of number of new lesions, (v) erosion of blood vessels with wide spread
distribution of tubercle bacilli (miliary tuberculosis) or with establishment of localised lesions at distant sites, (vi) subsequent reactivity of the lesion or (vii) re-infection, endogenous or exogenous.

One year after infection, the primary focus in the lung may extend to involve the pleural sac and cause an effusion. Biological healing of the primary foci takes a very long time. The bacilli may persist in calcified lesions for many years and perhaps biological healing is never complete in some cases. Anatomically, the calcified foci shrink and show regressive changes in which they first become denser and then by eventful absorption of calcium salts, the density may in turn decrease, finally, after some decades they may disappear. Ninety percent of all cases of tuberculous meningitis and miliary tuberculosis arise in the first few weeks or months after primary infection. Most of bone and joint lesions appear within two to three years of primary infection. Hematogenous skin lesions, papulonecrotic tuberculides appear as a rule, within two years, but isolated lesions of lupus vulgaries or verrucose tuberculides appear many years afterwards. Tuberculosis of the genital tract is uncommon in either sex before puberty.
Common Tuberculous Intrathoracic Lesions:

1. Segmental Lesion:

On occasion the initial lesion in the lung is not confined to a small focal area, but extends into the surrounding tissue or segments. Such lesions may involve several lobules or most of lobe. Though there may be symptoms; not infrequently, extensive pulmonary lesions are detected roentgenographically, in children, who have no complaints and had no physical finding. Average age incidence is 5 to 6 years. The lesions are frequently seen in right upper and middle zones.

A hilar lymphode involvement is an almost constant feature of pulmonary tuberculosis in childhood. The infection of the lymphode undergoes similar changes as that of parenchymal lesion until calcification is complete it has the same dangerous of local extension and hematogenous spread.

There may be intra-luminal extension of the tuberculous process usually in a lymphode, through the bronchial wall with formation of an ulcerative or granulomatous lesion. This may partly or completely obstruct the lumen of the branchous which ultimately would lead to the dissemination of the infection material to the other portions of tracheo-bronchial tree with
establishment of non-caseating broncho-pneumonia. Extraluminal occlusion may be partial or complete, brought about by enlarged, adjacent tuberculous lymphnodes without erosion through the bronchial wall. Partial compression gives rise to emphysema of the segment but in complete obstruction absorption atelectasis occurs. In each instance there may be a tuberculous pneumonitis in all or part of this involved pulmonary area.

Occasionally children with bronchial erosion do not develop either bronchial obstruction or a radiological segmental lesion. Instead, small areas of broncho-pneumonic changes appear diffusely throughout the lung field, these without the chemotherapy would ultimately coalesce to form extensive caseating broncho-pneumonia. It may be localised in one area of the lung or it may be widely disseminated children with this lesion tend to be quite sick.

2. Pleurisy:

Though the pleura is often involved it is less frequently found on clinical or radiological examination. It may occur as a dry fibrinous, pleurisy, as a serious effusion and rarely as a necrotic involvement of pleura, stemming from a contagious caseous focus in the lung.
Most effusions occur during the first few months after primary infection but some times clear evidence, such as glandular calcification may exist to show that the infection has been present since long. It does not occur below the age of 18 months, and rarely below five years. The common age incidence is between 10-12 years. Most authors have found that pleural effusion, like, primary complex itself, is more common on right side, and before the days of chemotherapy about 5% of children developed effusion on the other side also, within a year and occasionally within a few weeks after first infections. All pleural effusions should be regarded tuberculous until proved otherwise. There is a disagreement regarding the route by which the tubercle bacilli reach the pleura. According to Thompson and Land, the bacilli reach the pleura from an active primary lesion of the lung. Pleural effusion arises on the same side as the primary lesion in the lung. It is rarely seen on the contra-lateral side. Sibly believes that it is due to a haemotogenous spread as it is associated with miliary tuberculosis and other extra-pulmonary complications. The diagnosis depends on the tuberculin test, chemistry and cytology of pleural fluid and demonstration of organisms in pleural fluid. Pleural effusion may get complicated with bilateral effusion, meningitis, pericarditis and skeletal tuberculosis.
3. **Miliary Tuberculosis**

It is a blood borne infection characterised by multiple tubercle formations. Tubercle bacilli become lodged in the small capillaries, a lesion develops at each site and necrosis tends to develop rapidly in each of small foci. The symptoms are usually those of the general infection, initially there may be no physical signs. If choroidal tubercles are visible the diagnosis can be made before the results of tuberculin test X-ray are known. The distribution of the lesions may be limited to the lungs or may include other viscera i.e. liver, kidney, spleen and brain.

In miliary tuberculosis three major types of radiological picture are seen and in any of them other radiological evidence of tuberculosis may be present, such as a primary complex, mediastinal lymphadenitis, segmental lesion or pleural effusion.

i) **The "snowstorm" type**: In which innumerable small nodules are scattered equally throughout all the areas of both lungs. They are best seen in the diamond shaped spaces between the ribs; round, or approximately so, they vary in size from a millimetre upwards and when large enough have a centre denser than the edge which tends to be indistinct.
ii) The "hard" chronic type: These lesions are fewer in number and usually larger and give the impression of a lighter and more intermittent spread than that which produces the snowstorm film - the so-called sub-acute or chronic types of miliary lesions fall into this category.

iii) The "mixed" type: Usually found in infants or young toddlers who are ill with extensive disease, some of which is bronchogenic aerogenous spread and may contain areas of cavitating disease but in addition in the less affected areas, evidence of miliary spread may also be present.

There were well authenticated examples of survival after miliary tuberculosis before the introduction of chemotherapy but they were rare, the prognosis was bad for most cases developed fatal meningitis.

4. Tuberculous Empyema:

Generally speaking no child treated with chemotherapy at the onset of a pleural effusion develops an empyema. Miller, Seal and Taylor (1963) suggested that many effusion can become purulent, if sufficient tubercle bacilli are present, if the original cause of the effusion has been, rupture of a caseous lymphnode and occasionally, if there is a double infection with
Mycobacterium tuberculosis and pyogenic organisms. Price, on the other hand, says that serious effusions do not become purulent and that empyema do not occur in children except as a complication of pneumothorax or when haematogenous foci form in the pleura without underlying pulmonary involvement.

The possibility of empyema should be suspected whenever a pleural effusion becomes encysted or takes longer than usual to absorb. The general physical condition of the child is no indication of the character of the encysted effusion and diagnostic aspiration is required. If the empyema is small then aspiration and chemotherapy with careful supervision and adequate physiotherapy are sufficient. With larger collections or if the pleuras have become thickened so that the affected lung is immobilized, the possibility of surgical treatment will arise.

**Extra Thoracic Tuberculous Lesions**:

1. **Glandular Lesions**:

   In cases of extra-thoracic lesions, the cervical, preauricular, mesentrick or orginal lymphnodes are enlarged. Such enlargements are seen in older age group after five years of age.
Infection of the cervical lymphnodes is, in most instances, secondary to tuberculous tonsils or to a pulmonary lesion. Nodes of the both sides are affected frequently, initially discrete, firm and freely mobile, later when they become caseous, there is tendency to erosion of the capsule, matting together with the adjacent nodes forming an irregular mass. The mass becomes attached to other adjacent structures. Subsequently the caseous mass may liquify and not infrequently the overlying skin is perforated forming a sinus. Retropharyngeal lymphnode involvement may give rise to osseous lesion in the cervical vertebrae, and retropharyngeal abscess. In spite of this, most of the nodes undergo resolution and calcification before extensive caseation occurs. Other glandular involvement may occur in axilla, groin and occipital region, usually occurring secondary to tuberculosis of skin, but are of rarer occurrence.

2. **Abdominal Tuberculosis**

Primary abdominal infection may occur alone or simultaneously with primary infection elsewhere, usually in the oropharynx or the lungs. When it does occur the usual sequence of events ensues and a small lesion in the bowel wall is associated with enlargement and caseation in the regional mesenteric nodes. Localised clinical illness
at this stage is almost always due to complications of the mesentric adenitis, which are as follows:

e) A node may rupture and the liberation of caseous material and possibly tubercle bacilli into the peritoneal cavity produces a reaction analogous to that of a pleural effusion.

b) The node may soften and slowly involve neighbouring coils of bowel in a plastic peritonitis causing acute or sub-acute intestinal obstruction.

c) A single loop of bowel may become adherent over the surface of a node and given rise to acute intestinal obstruction.

d) As in peripheral adenitis, healing mesenteric nodes may become active again after nonspecific infection.

Mild constitutional and vague general symptoms are the main features of this disease. The physical findings are rarely supporting. It is suspected on the findings of tuberculin conversion and raised E.S.R. in the absence of active pulmonary disease. The diagnosis is supported by the changes observed during the therapy (Gefel et al, 1963) and is proved after leprotomy. It has been classified in the following types:
i) Intestinal type: It has two forms; ulcerative and hypertrophic, the latter is more common in cases of primary infections.

ii) Glancular type or mesentric adenitis.

iii) Peritontial type: It has got 3 sub-types; ascitic—most common, plastic or fibrinous and miliary (rare).

iv) Combined type.

3. Primary Tonsillar Insfection:

In tonsils, the primary focus is localised in the crypts. It is small and not as a rule demonstrable by examination in vivo, and the tonsil need not be enlarged (Wallgren, 1948). Observations of Miller et al (1963) have shown that unequal tonsillar enlargement associated with regional adenitis should always arouse suspicion of a primary tuberculous infection, and this unequal enlargement was striking in ten of their twelve children. The inequality was noticeable even if both tonsils were large. After removal of the tonsil the tuberculous lesions are easy to detect by histological examination.

4. Tuberculosis of Skin:

Ghon (1912) in his monograph on primary lung infection recognised that primary complex could occur on skin or a mucosal surface. Since then many case reports
of primary skin infection have appeared but almost always have been limited to one or two cases. Tubercle bacilli invade the skin or mucus membrane through abrasions; the common sites are the lip, nose, chin, extremities and genital region. There is an accompanying involvement of regional lymphnodes to complete the primary complex. Its common forms met within childhood are lupus vulgaris, sacrofuloderma, lichen sacrofulosus and tuberculides.

5. **Congenital Tuberculosis**:

The cases of congenital tuberculosis reported up to 1945 have been critically analysed by Hughesdon (1946), who added a few new cases. Infants with truly congenital tuberculosis may give no indication of illness before their sudden death. Attention should be called to the infant by the development of the nasal discharge, cough, dyspnoea, lethargy, anorexia, failure to gain weight or by the passage of bloody stool indicating intestinal ulceration. By the time the disease is suspected, lungs ordinarily show advanced, wide-spread areas of consolidation and hilar gland enlargement, with or without cavitation and miliary spread. The course is long and even in the face of vigorous therapy progression is the rule.
**Allergic Manifestations in Primary Tuberculosis:**

These manifestations are owing to the allergic reaction to the tuberculoprotein circulating in the body of the individual. Such manifestations are as under:

i) **Phylictenular Conjunctivitis**: It is characterised by a lesion seen as a small, grey spot at the limbus in one or both eyes, a single spot with a leash of conjunctival vessels running towards phylactenules. Phylactenules are accumulations of lymphoid cells beneath the epithelium of cornea or conjunctiva. This conjunctivitis is seen most often between 5 and 15 years.

ii) **Erythema Nodosum**: These are believed to result from hyper-sensitivity to tuberculoprotein in cases of tuberculosis. These lesions are characterised by the development of tender, painful indurated, shining, elevated ovoid patches 1 to 3 cms. in diameter, usually symmetrically distributed over the shins, calves, knees, buttocks and occasionally the arms. The indurations decrease after 1-2 weeks. Crops of lesion occur, generally over a period of 3-6 weeks, later recurrences are unusual.

iii) **Allergic Lymphadenitis**: The adenitis appears rather suddenly in association with constitutional symptoms. In case of cervical adenitis it is bilateral. The gland recedes in two to three weeks' time and with it the constitutional symptoms also disappear.
iv) **Allergic Pleural Effusion**: It appears at the time of tuberculin conversion and is similar to other forms described already.

**Diagnosis of Primary Tuberculosis**:

Early diagnosis of primary tuberculous infections is of paramount importance in order to reduce the incidence and prevent the complications of the disease. Besides the positive history of contact, haematological examination, I.E.R. and bacteriological investigations, a positive tuberculin and abnormal roentgenographic appearance are the essential criteria for the absolute confirmation of the diagnosis. Lesions are termed inactive when constitutional disturbances, physical signs, radiologic evidence, bacteriologic examination and other investigations are negative (Sekulick, 1955).

**Tuberculin Test**:

The tuberculin test was discovered by Von Pirquet in 1907 and was established as a principal case-finding agent. Various methods have been employed from time to time by different workers; viz. Von Pirquet Test (scarification of the skin through Cold Tuberculin), the Mantoux Test (intradermal injection of P.P.D.), percutaneous Tests (Vollmer patch test, the jelly test), Heaf's Multiple puncture Test and Tine Test. No other method or no other
material has been as extensively used in this country or perhaps in the whole world as the Mantoux technique using

Mantoux Test:

Charles Mantoux suggested a dose of 1 T.U. (1/20th c.c. of a 1,5000 dilution of C.I.). Tuberculin used for this test is of 2 types - (i) Old tuberculin, prepared by growing tubercle bacilli on artificial mediums, then entire culture products is filtered and the filtrate is concentrated and used, (ii) Purified protein Derivative (P.P.D.).

Around 1934, Seibert and Munday having realised that the anti-genicity of tuberculin was related to its molecular size, produced the substance known as purified protein derivative, in which the proteins were of a relatively small molecular size, this was a major advance. In 1941, the W.H.O. established P.P.D.-S at international standard for Mammalian tuberculosis. In 1958, however, it was found that a new tuberculin P.P.D. R.T. 23 in a solution containing Tween 80 was more potent than P.P.D.-S. P.P.D. is obtained by growing tubercule bacilli of strain RT 23 on protein free culture medium, from which protein (Tubercle bacillus protein) is then precipitated. P.P.D. is thus more purified product than is old tuberculin and
it has been adequately demonstrated that it is an effective skin testing material, and after dilution can be stored in refrigerator for 6 months and marketed in dry state, which is diluted at the time of use.

**Dose of P.P.D.**

The potency of tuberculin (P.P.D.) may be expressed in the terms of international tuberculin unit called T.U. One T.U. is equivalent to .00002 mg. of P.P.D., i.e. 1:50,000 or 0.01 mg. O.T. There is a great controversy regarding dosage of P.P.D. to be employed. Some authors have suggested 10 T.U. for routine check-up, while others suggest 1 T.U. because with 10 T.U. there are chances of severe reaction and false positive results. Agarwal (1962) and Bagle (1962) have advocated 1 T.U. of PPD RT 23 with tween 80, as the ideal testing dose for our country. The standardization of tuberculin reaction, as described by W.H.O. (1959) is as follows:

a) Induration of 10 mm. are more regarded as positive (but drugs and diseases should be kept in mind).

b) Induration between 5-10 mm. is doubtful and in these cases test should be repeated.

c) Induration below 5 mm. is negative.
Agarwal and Nagle (1962), in their separate studies have recommended an induration 8 mm. and above to consider the test as positive.

**Hematological Examination:**

Haemoglobin percentage, total and differential leucocytic counts, agglutination, complement fixation and the haemagglutination tests do not serve as a diagnostic or prognostic purpose.

**Sedimentation Rate:**

Wintrobe has noted that raised sedimentation rate in the presence of the other evidences for the etiologic diagnosis, suggest acute disease process. The rate varies with the extent and nature of this disease. He further emphasized that it is a guide to the process of infection, particularly in tuberculosis. Though a normal sedimentation rate does not necessarily mean that all is well, occasionally specially in cachexia. The importance of sedimentation rate lies more in judging the prognosis and course of the disease process.

**X-Ray Examination:**

"In the diagnosis of pulmonary tuberculosis at any rate Roentgen tube has superseded Laennec's tube", Burton Wood (1930). In fact, it is the only method as has been
shown by results of mass radiography in detection of the cases. The primary focus is seldom shown in recent primary pulmonary tuberculosis, the X-ray changes are usually those of hilar infiltration, which owing to its smallness and its position is obscured by the air content of the lung. The hilar changes consist of enlargement and increased density of the root shadow with streaky or cloudy diffuse limitation. In lateral (oblique) view the retrocardial space is occupied by the shadow of enlarged glands. If the primary focus is visible, it presents itself as a homogeneous, cloudy, rather diffuse shadow, often seemingly in direct connection with the enlarged hilar shadow.

There is nothing characteristic of the X-ray picture of primary tuberculosis. The same changes may encountered in several other disease for example atypical pneumonia, common pneumonic lesions etc. The X-ray changes in cases of pulmonary primary tuberculosis remain for at lease 3-4 months without any sign of decreasing and the hilar shadow become gradually smaller, more distinctly limited and more dense (Wellgren and Wegelius, 1949). After 2-3 years calcification is seen in the necrotic parts of the hilar glands and in the primary focus. If not previously seen in the X-ray film, the primary focus become obvious after calcifications.
Vasantkumar et al (1976) studied 1500 cases of clinically suspected primary complex radiologically. A dynamic radiological classification of primary complex was attempted. The pathological significance of interlobar pleural opacification had been stressed. The various features observed on the chest roentgenogram were numbered and tabulated as:-

1. Primary focus with parifocal inflammation.
2. Lymphangitis.
3. Hilar adenitis.
5. Interlobar pleural opacification.
6. Primary focal calcification.
7. Calcification of Lymphatics –
   a) Hilar.
   b) Mediastinal.
11. Pleural calcification.
15. Bronchopneumonia.
16. Associated caries of thoracic spine.
17. Tubercular pneumonia.

18. Obstructive emphysema.

19. Extrapulmonary calcification.

20. Cavitating lesion.

In his study, it was noted that 76% of cases and 63% cases of Mantoux positive, the minor interlobar pleural fissure was opacified. So this work revealed that the opacification of interlobar pleural fissure in the chest skiagram of the children were of significant value in diagnosis of primary complex.

Ramchandran and Mukundan (1976) reported to approach radiological shadows in tuberculosis. 1. How long after proper treatment, do lymphnodes and/or parenchymal lesions get calcified? 2. How long after treatment do the intrathoracic lymphnode shadow diminish in size of disappear completely?

How often does intracranial calcification occur in children suffering from tuberculous meningitis? Is there a discernible relationship between intrathoracic and intracranial calcification? An attempt was made to answer above questions. The X-ray analysis of the report taken from the children attending T.B. Clinic, in the department of Paediatric, Medical College, Tanjavur. The children were registered as per criteria of Ramchandran...
(1971), namely - suspicious symptoms, mantoux positive, radiological evidence of Tuberculosis and chest X-ray.

Out of 3000 children registered, 630 X-rays only were available for studies.

In the radiological study of 1630 cases, 527 had only lymphnode involvement, 223 had node with parenchymal focus and 86 had multiple infiltration. So lymphnode constitute above 50% of radiological findings.

Payre quoted Miller (1963) reported that 32.7% of children with pulmonary primary infection had radiological evidence as primary complex. In majority of X-ray the infiltration is in the right upper lobe and the lymphnode is often superior mediastinal and sometimes right hilar. In cases with right lower lobe infiltration, the lymphnode are often right interior and posterior bronchial.

**Lymphnode enlargement**

<table>
<thead>
<tr>
<th>Location</th>
<th>No. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superior mediastinal adenites</td>
<td>215</td>
</tr>
<tr>
<td>Right hilar adenites</td>
<td>165</td>
</tr>
<tr>
<td>Left hilar adenites</td>
<td>36</td>
</tr>
<tr>
<td>Bilateral hilar adenites</td>
<td>53</td>
</tr>
<tr>
<td>All glands</td>
<td>58</td>
</tr>
</tbody>
</table>
### Lymphnode with parenchymal infiltration

<table>
<thead>
<tr>
<th>Location</th>
<th>No. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superior mediastinal adenites</td>
<td>72</td>
</tr>
<tr>
<td>Right hilar adenites</td>
<td>96</td>
</tr>
<tr>
<td>Left hilar adenites</td>
<td>32</td>
</tr>
<tr>
<td>Both right and left adenites</td>
<td>23</td>
</tr>
</tbody>
</table>

### Progressive primary complex

<table>
<thead>
<tr>
<th>Location</th>
<th>No. of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lymphnode with multiple infiltration</td>
<td>86</td>
</tr>
<tr>
<td>Consolidation</td>
<td>134</td>
</tr>
<tr>
<td>Effusion</td>
<td>23</td>
</tr>
<tr>
<td>Broncho-pneumonia</td>
<td>74</td>
</tr>
<tr>
<td>Miliary</td>
<td>74</td>
</tr>
</tbody>
</table>

In this series 86 cases had multiple infiltration distribution all over the long fields and in 60% X-rays, they were at right side.
Pleural effusion:

Twenty three cases reported with pleural effusion (Miller, 1963) reported more right sided effusions, almost all pleural effusions are post primary. On X-ray examination more often a horizontal line is seen in children due to fluid in inter-lobe septum or to a co-existing segmental lesion. Quite often the pleural effusion is revealed by vertical line following the attachment of parital pleura to apex of the lung.

The radiological evidence of pleural effusion disappears within 4 weeks on steroid therapy. Miller (1963) reported the clearance of effusion radiologically depends on the duration of effusion prior to therapy.

Miliary:

Miller (1963) miliary tuberculosis can be of 3 types in radiological appearance. Snowstorm, hard chronic type, mixed type.

Lincoln's (1963) criteria divides radiological appearance into classical and sub-miliary. The sub-miliary shadow become evident when film is looked at slanting position. X-ray give impression of granularity. Twenty out of 74 shows miliary lesions, rest associated with either superior mediastinal nodes, hilar adenopathy or parenchymal patches.
Consolidation - 123 cases shows consolidation out of which 99 cases were right sided similar to infiltration. 20% of these cases shows AFB in gastric levage.

Calcification - Calcification of tuberculosis lymphnodes and parenchymal lesion is end resulting the disease of all above cases.

According to Payne quoted by Miller (1963) calcification occurs after 4 years of treatments and 80% calcified after 3 years of treatments.

Tubercular meningitis - 55 skull X-ray children with tubercular meningitis were taken. Only 6 shows calification after 1½ years of treatment. The incidence of intracranial calcification was very low compared to Corber's figure of 48.4% and Miller's (1963) 37.5%. This was attributed due to the use of corticosteroid therapy and poor nutritional status of children.

As study carried out by S. Malik, K.L.Narasimharao (Nov., 1983).

A child with clinical and radiological features of solitary mass lesion in the lung proved to be tuberculoma was reported for its rarity.
Tuberculoma is a localised granulomatous lesion and is manifestation of primary tubercular infection. Large tuberculoma is rare in children and is extremely rare in children under 2 years. Tuberculoma when associated with mediastinal gland enlargement usually have anterior group of glands involved.

A study carried out by N.R. Bhandari and associates, Indian Paediatric J. (July, 1984).

B.C.G. test was positive in 90.9% cases of tuberculosis as compared to tuberculin test which was positive to 47.2%. The effect of malnutrition on the diagnostic sensitivity of BCG test is not significant local complication was observed in 1.2% cases. BCG test is safe and simple diagnostic method for diagnosis of childhood tuberculosis and at the same time provides immunity to those who need it.

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