"A STUDY OF CLINICAL AND RADIOLOGICAL PROFILE OF SPUTUM POSITIVE TUBERCULOSIS AMONG ELDERLY PATIENTS"

By

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Dissertation submitted to BLDE(DU) University, Vijayapura

In partial fulfilment of the requirements for the award of the degree of

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

Under the guidance of

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Dr. PRANAY KUMAR R. P.

LIST OF ABBREVIATIONS USED

1. TB	–	tuberculosis
2. TAI	–	tuberculosis association of India
3. DOTS	–	directly observed treatment short coarse
4. PAS	–	para amino salicylic acid
5. WHO	–	world health organisation
6. RNTCP	–	revised national tuberculosis control programme
7. AFB	–	acid fast bacilli
8. HIV	–	human immunodeficiency virus
9. AIDS	–	acquired immune deficiency syndrome
10. TST	–	tuberculin skin test
11. ZN	–	Zeehl- Neelson stain
12. CT	-	computerized tomography
13. MRI	–	magnetic resonance imaging
14. NAAT	–	nucleic acid amplification test
15. H	–	isoniazid
16. R	–	rifampicin
17. Z	–	pyrazinamide
18. E	–	ethambutol
19. S	–	streptomycin
20. Km	–	kanamycin

21. Lfx – levofloxacin
22. Eto – ethionamide
23. Cs – cycloserine
24. Cm – capreomycin
25. Mfx – moxifloxacin
26. Lzd – linezolid
27. Amx/Clv – amoxicillin/clavulanic acid
28. Cfz – clofazimine

ABSTRACT

NEED FOR THE STUDY:

Tuberculosis has forever been known to mankind to cause morbidity and mortality. The disease has been extensively studied in the younger population to present with typical symptoms and radiological features, but has rather not got the importance it deserves in the elderly. Hence, through this study we wish to study the various patterns of presentation of the geriatric population with tuberculosis and also the various radiological lesions that these patients demonstrate, so that earlier diagnosis is aimed and the definitive diagnosis of tuberculosis is done in the geriatric population despite the lack of typical features.

METHODS:

A cross sectional, descriptive study was conducted by studying 70 patients who attended the outpatient department or those who were admitted in the wards of Shri B. M. Patil Medical College and Hospital. The patients who were considered into the study were to satisfy the inclusion and exclusion criteria. The patients once admitted were submitted for sputum sampling by staining techniques for detection of tuberculosis bacilli. The patients that tested positive for tuberculosis by the staining techniques were to undergo chest X-ray for studying the radiological presentations of these patients. Detailed history was taken and the patients were examined clinically for various patterns. The study was conducted between December 2018 and June 2020.

RESULTS:

Our study showed that the various symptoms that the geriatric population presented with were cough, dyspnoea, chest pain and hemoptysis. While a significant

number of the patients presented with atypical presentation, the most common feature was weight loss followed by fever and night sweats.

The radiological features present in these patients were miliary mottling followed by pleural effusion, middle and lower lobe infiltrates, calcification, cavitation, fibrosis, collapse, hydropneumothorax, synpneumonic effusion.

CONCLUSION:

Geriatric patients with tuberculosis do not have typical features like the young. Therefore there needs to be a very high grade of suspicion in the elderly to look for tuberculosis as the symptoms are often masked or atypical in nature. Radiological studies do aid in diagnosing tuberculosis, but it should be kept in mind that even the chest X-ray findings are not the usual and demonstrate different lesions in different areas of the lung. A good idea about these presentations can guide the clinician to diagnose tuberculosis in the elderly early and also initiate treatment early.

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INTRODUCTION

Tuberculosis(TB) is an infectious disease caused by the bacteria – Mycobacterium Tuberculosis(M.tuberculosis). TB intimidates the human race since time immemorial due to its effect on the social and economic welfare and as a medical tragedy. Even with the advent of modern diagnostic techniques we, as a generation are still to witness the myriad of maladies this sagacious disease is subjecting us to.

It continues to be a very important communicable disease in the world, especially developing countries like India contributing to a major burden of it. Historically, TB was considered to be a disease of the younger population. But as the years pass by, there is a changing trend in the distribution of the disease with respect to epidemiological and clinical characters. One such major change is the transition of cases towards the older people, aged greater than 60 years. The disease can present as a focal infection alone or can progress to multisystem involvement thereby producing significant morbidity and mortality. This contagious infection usually spreads via air borne transmission(inhalation of droplet nuclei – each airborne particle measuring 1 to 5 microns in diameter).

The most constant and distinctive demographic event since the origin is ageing. Initially visualised by the developing countries, but is now being observed globally. In gross terminologies, the number of elderly have tripled over the last fifty years and is expected to grow in the same or greater proportion over the next fifty years.

The geriatric population profile is dramatically changing over the past few years with the average life expectancy at birth extending from 47 years in 1900 to nearly 79 years in 2014 globally. Worldwide, the number of adults over 60 years of

age will rise to 2 billion by 2050 and will constitute over 20 percent of the world's population[1]. Projections based on the current statistics indicate that the vast majority of these older adults reside in developed countries.

TB in the geriatric population usually present with atypical symptoms, thereby leading to a delay in the diagnosis and thereby the delay in initiation of treatment. Hence, higher rates of mortality and morbidity is noted in this subset of the population. Underlying illnesses, age related immune deficiencies, higher rates of adverse drug reactions and prolonged hospital stays can complicate the overall outcome in elderly patients with TB[2]. Acute or chronic diseases, malnutrition, disruption of the protective barriers with aging, impairment of the microbial clearance mechanisms also contribute for development of TB in the geriatric population[3].

There is definitely a deficiency of data regarding the burden of this infection in the elderly from India. With the increase in the geriatric population and a concomitant increase in the number of diagnosed cases, it is evident that the problem of tuberculosis in elderly is yet to seek the importance it deserves. Owing to the reason of misdiagnosis of TB in elderly as bronchiectasis, pneumonia, it may well be given the name "The Grey Plague".

OBJECTIVES

The study was undertaken with the following aims:

1) To study the various patterns of presentation of sputum positive pulmonary tuberculosis in geriatric population.

To study the radiological features in these patients.

REVIEW OF LITERATURE

EPIDEMIOLOGY:

The pattern of epidemiological variation in geriatric TB shows that there is a predilection to younger population in high incidence countries and elderly population is affected mainly in low incidence countries. TB was found to be higher in the geriatric population of nursing homes owing to the cluster gatherings[51]. TB also lead to the increased mortality in elderly not only due to the disease process itself, but also the adverse effects of the drugs used to treat TB. TB in the elderly needs to be given increased concentration due to the fact that in the elderly, the disease is usually misdiagnosed as bronchitis, asthma, bronchiectasis or pneumonia. Studies done in 1800 prophesized the elderly would compromised the single largest group of patients with active tuberculosis and increasing proportion of deaths attributed to TB were in the elderly[9].

More than 1.7 billion people, about 25% of the world population are estimated to be infected with M.tuberculosis[4]. The global incidence of TB peaked around 2003 and appears to be declining slowly. According to the World Health Organisation (WHO), in 2018, 10 million individuals became ill with TB and 1.5 million died[5].

In the United States, the case rate has decreased from 10.5 per 1 lakh population in 1992 to 5.8 per 1 lakh population in 2000, which is a 45% decrease[7]. Although the case rate among the elderly has decreased significantly, from 18.7 per 1 lakh population in 1992 to 11.7 per 1 lakh population in 1999, the elderly still have the highest case rate among all age groups[8].

This is a 16% increase as compared to 2017[6]. According to the national figures in 1953, among those with newly reported TB, the proportion of patient who were

aged above 60 years was 13.8%. By 1979, the figure rose to 28.6%, a rise not explained by an increase in the elderly people from 8.7% to 11.2%.

The RNTCP states that among the population aged above 65 years, 8% were sputum positive for pulmonary tuberculosis. The estimated TB incidence in India is 27lakh. In 2018, RNTCP was able to achieve a notification of 21.5lakh.

REGION	1950	2000	2050
WORLD	205475	605785	1963767
MORE DEVELOPED REGIONS	95473	231442	395106
LESS DEVELOPED REGIONS	110003	374343	1568660
LEAST DEVELOPED REGIONS	10773	32167	173222

The table depicts the estimated rates of TB as per 1000 population and shows the wide variation in the case load between the various regions of the world.

HISTORICAL ASPECTS:

Johann Schonlein coined the term "tuberculosis" in 1834[11]. But according to estimates TB has dated to as long as 3 million years.

TB was called as "phthisis" in ancient Greece, "tabes" in Rome, "schachepheth" in ancient Hebrew.

In 1700, the term "white plague" was designated due to pale appearance of most of the patients with TB. It was also termed "captain of all these men of death".

TB of the neck and lymph nodes was given the term "scofula". In 1793, the caseous, cheese like necrosis in the abscesses was termed "tubercles" by the pathologist Matthew Baille.

In 1810, the French physician Gaspard-Laurent Bayle described the disseminated "miliary" TB.

Extra-pulmonary tubercles were recognised in the intestines, liver, meninges and other organs.

The term “Pott’s disease” was described by the British surgeon Sir Percivall Pott as the vertebral collapse and spinal cord paralysis caused by TB.

On March 24th 1882, Dr. Robert Koch announced the discovery of Mycobacterium tuberculosis, the bacteria responsible for TB. During the time of discovery, the case fatality was one in every 7 cases. The discovery was one of the most crucial steps towards the control and elimination of the disease. A century later, March 24th was designated as the World TB Day- a day dedicated to educate the public on various modalities of TB. Robert Koch was awarded the nobel prize in physiology or medicine in the year 1905 for his discoveries and inventions in relation to TB.

In India, TB had existed for several thousand years and had been extensively written about since 1500 BC where it was attributed to be a disease consisting of fatigue and hunger. Around 500 BC, the Sanskrit manuscripts had written about this disease and further the ayurvedic texts were derived for the same, where they referred to the disease as SOSHA.

It was given the term KSHAYA ROGA in Sanskrit. They claimed the disease to be characterised by wasting and coughing blood. Towards the middle of the nineteenth century is when people and revolutionaries began to recognize TB as a highly infective disease and began to plan strategies for the control and elimination of the disease. The TB Association of India(TAI), is one of the oldest and the longest serving voluntary organisation for curbing the disease throughout the country by means of multiple associates. It was set up in February, 1939 by using the then famous anti- TB fund and her highness, the Marchioness of Linlithgow was its first

president. Since then to date, the organisation has been working selflessly for developing novel strategies aiming at early recognition and effective treatment of TB.

It has been very efficient in publishing various journals and also conducting training programmes for training the health care workers to work in the right direction. It has also aided in the development of the Revised National TB Control Programme(RNTCP) and Daily Observed Therapy Short course(DOTS) programmes as essential government services. Schemes like incentives to the patient and the doctor who report the case of TB have made the sense of responsibility higher in the general population and has helped in reducing the lack of awareness towards the disease that people had.

The discovery of the drugs used in TB also dates back to the times of the world war II. Streptomysin was the first antimicrobial agent developed after penicillin and was also the first effective antibiotic in treating TB. It was discovered by Selman Waksman, Albert Schatz and Elizabeth Bugie in 1943. With the passage of time resistance of the bacilli was noticed to streptomycin, hence seeking the development of drugs apart from streptomysin. Thereby came the origin of para-aminosalicylic acid(PAS) and isoniazid. These drugs used either single or as a combination reduced the resistance and improved the outlook of the infection of TB.

A rather simplified timeline can be deduced as follows regarding the various events that were of paramount importance in developing the drugs for TB :

1946 – clinical use of streptomycin as monotherapy and resistance to it.

1948 – double drug regimen like streptomycin with PAS, prevents drug resistance emergence.

1952-55 – introduction isoniazid along with double drug regimen.

1958 – direct observation treatment is essential.

1958-67 – daily administration of three drug regimens(streptomycin, thioacetazone, isoniazid, rifampicin).

1959 – sanitorium treatment is not required and ambulatory chemotherapy is sufficient.

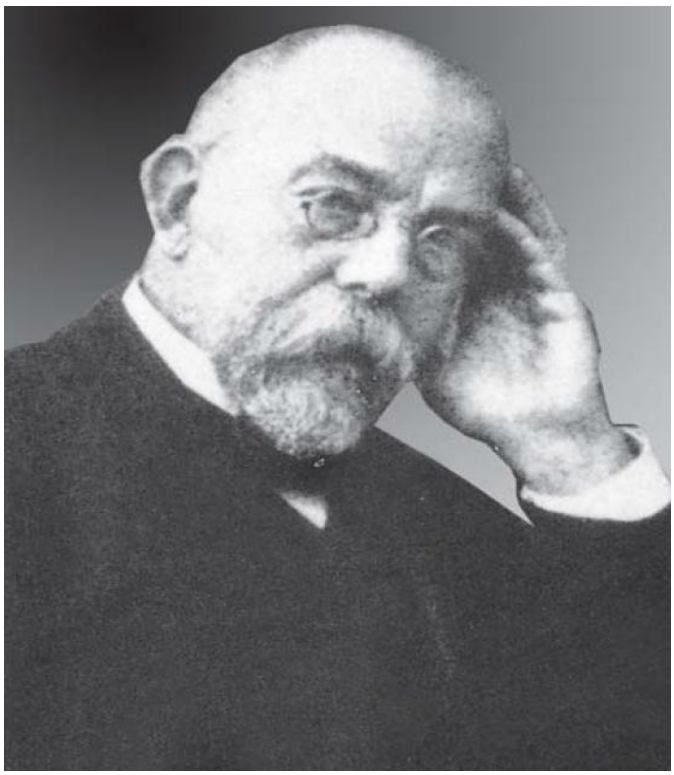
1961- peripheral neuropathy is a side effect of isoniazid.

1964 – efficacy of twice weekly regimen of streptomycin, isoniazid noted.

1969 - twice weekly PAS, isoniazid dosage is sufficient.

1974 – effectiveness of short course daily regimens

ROBERT KOCH



PATHOLOGY :

Although controversial, the pathology of tuberculosis is somewhat similar in many aspects to the other infective diseases , involving the interplay between the bacterium and the host immunity. TB is most predominantly spread by aerosols, hence pulmonary TB is the most dominant form of the disease. The lung is indeed the primary site for infection and the extra-pulmonary TB is either a consequence or an accompaniment of the primary disease.

TB is classified into the following types based on multiple factors :

1. Primary TB : caused by mycobacterium tuberculosis in a person with no prior exposure.
2. Progressive Primary TB : the usually self-limiting primary disease progresses further with larger lesions.
3. Post-primary TB : due to endogenous reactivation(with prior exposure to mycobacterium tuberculosis) or exogenous infection.
4. Based on location : Pulmonary TB

   Extra-pulmonary TB

   Disseminated TB – involving more than 2 non-contiguous sites.

HISTOPATHOLOGY:

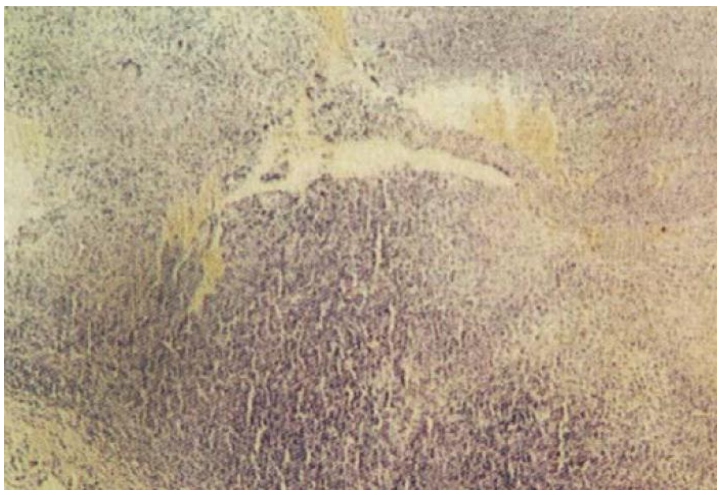
The hallmark of TB is a granuloma. Granuloma is derived from the Latin term granulum, meaning grain. Primarily used by Rudolf Virchow to enunciate lesions that ulcerate and produce granulation tissue[10,11]. It can be defined as a focal collection of inflammatory cells, predominated by mononuclear cells arranged in a compact manner. They are usually a product of either non degradable organisms or hypersensitivity reaction or both, thereby indicating an immunological background in the formation of granulomas. It is usually an amalgamation of factors such as the

bacterium, macrophages, T-cell and B-cell activity along with circulating immune complexes.

Granulomas invariably show organization. Centrally, macrophages predominate, which are essential because this is where the infecting organism is phagocytosed. Activated macrophages have a pale, large, eosinophilic cytoplasm giving a foamy appearance. These are epithelioid cells and the granuloma is termed as epithelioid cell granuloma. Epithelioid cells fuse together to form multinucleated giant cells. Two types of giant cells are identified in TB. In one type, peripheral nuclei form a rosette around the central cytoplasm. This is the Langhans' giant cell, named after Theodore Langhans. The other type of giant cells are the foreign-body giant cells, which do not demonstrate such a regular nuclear arrangement.

Macrophages in the granuloma are surrounded by lymphocytes and eosinophils. Healing, old granulomas demonstrate fibrosis. Finally, the entire granuloma undergoes fibrosis, calcification, hyalinization and ossification. The presence of a central area of necrosis separates a necrotising granuloma from a non-necrotising granuloma. The presence of a necrotising epitheloid cell granuloma is the hallmark of TB.

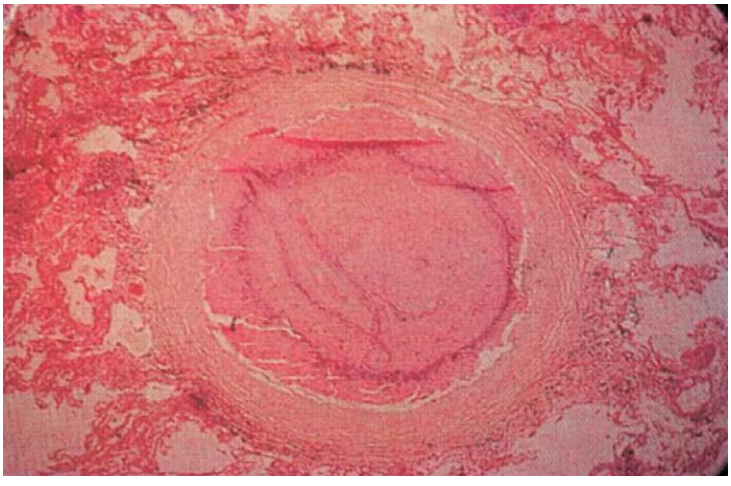
GRANULOMATOUS TUBERCULAR LYMPHADENITIS



GRANUOMATOUS INFLAMMATION IN TUBERCULOSIS :

Microanatomical lesions in TB are divided into exudative and proliferative lesions. Exudative lesions are less demarcated, comprising of lymphocytes, neutrophils, macrophages and epithelioid histiocytes with little fibroblastic activity. These are soft granulomas and are likely to contain Acid Fast Bacilli(AFB). On the other hand, proliferative lesions are well circumscribed with epithelioid histiocytes surrounded by lymphocytes. Fibroblastic activity is high and these are the hard granulomas.

Mathew Ballie and Alois Rudolph Vetter compared the lesions in TB to cheese[11]. This “cheese-like” necrosis on gross TB lesions is termed as caseous necrosis. Caseating granulomas are indeed characteristically but not exclusively present in TB. The caseum has low oxygen content, low pH and accumulation of fatty acids that inhibit bacterial replication. The caseum when encapsulated by fibrous tissue is termed as a fibro-caseous granuloma. The caseous focus can undergo calcification, ossification, liquefaction and cavitation. Caseation necrosis generally indicates that the lesion is active.



NODULAR TB OF LUNG WITH CASEATION, CALCIFICATION AND CIRCUMFERENTIAL FIBROSIS.

PATHOGENESIS :

The predominant site for primary TB infection is the lung. Mycobacterium tuberculosis is the most common pathogen. Mycobacterium bovis was an important pathogen but infection is now rare due to the knowledge regarding pasteurization of milk and relative rarity in bovine rearing.

The conditions responsible for rendering individuals at a relatively high risk for TB are[12] :

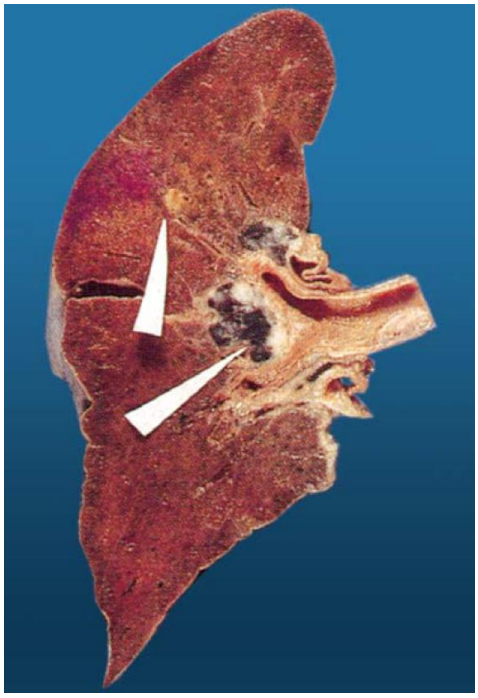
1. Cell mediated immunodeficiency including HIV infection and AIDS
2. Immunosuppressive therapy
3. Immunomodulator drugs
4. Malignant neoplasm
5. Silicosis
6. High dose, long-term corticosteroid therapy
7. Ill controlled diabetes mellitus
8. Chronic renal failure and haemodialysis
9. Connective tissue disorders
10. Organ transplantation
11. Intravenous drug abuse, heroin addiction
12. Tobacco smoking

PRIMARY TUBERCULOSIS :

Initially, primary TB in children was explained by the Parrot's Law which said: "pulmonary TB does not exist in the child without involvement of the tracheobronchial gland"[13]. It meant that lymphatic involvement was essential. Later

this concept was studied even in adults. On the basis of these studies, Anton Ghon formulated his hypothesis[14].

The infection is lugged via the lymphatics to the draining tracheobronchial lymph nodes that eventually enlarge. Through the lymphatic and hematogenous routes, the infection is spread to various tissues, a stage termed as bacillaemia[15,16]. Initial infection is unrecognized despite the tubercle bacilli disseminated in the body. Primary TB infections recover spontaneously with occasional calcifications. Repair is characterised by resorption of caseous material, fibrosis, dystrophic calcification. The typical primary parenchymal lesion of TB of lung is seen as a nodular, subpleural area of necrosis surrounded by fibrosis which eventually calcifies. Calcification is seen on microscopy by two months, but radiologically visible by one year. Primary TB implies that the bacilli remain dormant for a variable period of time. Ultimately resorption of the calcium occurs in the lung and lymph node lesions and these sequence of events remains the central dogma in most of the organs affected by TB.



PRIMARY COMPLEX IN THE LUNG. ARROWS DEPICT THE PRIMARY FOCUS AND ENLARGED TRACHEO-BRONCHIAL LYMPH NODES.

PRIMARY COMPLEX :

The initial unit of pulmonary TB is composed of primary focus and the infected lymph nodes. This is termed as the primary complex of Ranke which inevitably involves the intervening lymphatics between the infected lesion and surrounding lymph nodes.

The terms primary complex of lung and Ghon's complex are synonymous. The primary focus in lung subpleural, in the middle portion [the lower portion of the middle lobe when on the right side or upper region of the lower lobe] and is termed as the Ghon's focus. Hence, the unit of Ghon's focus and the draining tracheobronchial lymph nodes is the Ghon's complex.

Following the lung, the gastrointestinal mucosa is another favourable site for development of TB. The other sites are relatively less involved[17].

- SKIN : As for the other infections, even TB bacilli require a breach in the surface of the skin for their entry. The exposed areas of the skin, especially in certain occupations like pathologists, microbiologists, necropsy attendants are more prone for cutaneous TB. As the primary TB foci in skin is similar to syphilitic skin lesions, cutaneous TB has also been termed as "tubercular chancre". It is also associated with lymph node enlargement[18]. Cutaneous TB has been demonstrated following a subcutaneous, intramuscular injections and venepuncture[19].
- GASTROINTESTINAL TRACT, LIVER : With the significant knowledge regarding unhygienic milk intake, the number of cases of TB in gastrointestinal tract have reduced drastically. Rarely, the buccal mucosa are sites for TB secondary to dental caries, associated with submandibular lymph node enlargement[20,21]. The tongue is also a site for TB[22]. TB of oesophagus,

stomach, ileum, colon, vermiform appendix have also been reported[23]. TB in the liver is invariably congenital.

- HEAD AND NECK : TB of the pharynx, larynx, uvula have been seen as primary sites associated with tonsillar and cervical lymph nodal spread[24-27]. It can also involve the nose, nasopharynx, middle ear cavity and parotid glands.
- GENITOURINARY TRACT : The skin of over the penis is a rare site for TB. It usually follows circumcision or sexual transmission[28]. The vagina and vulva are also rare sites for TB following sexual transmission.
- EYE : TB of the lacrimal sac, conjunctiva and the retina have been reported[29].
- DISSEMINATED TB : It is defined as the presence of wide spread visceral tubercles secondary to haematogenous transmission of infective TB bacilli from an active caseous focus[30]. The prototype finding of miliary TB are small(1 to 2 mm in diameter), discrete nodules, greyish red on cut surface, distributed evenly throughout the involved organ. The lung, spleen, liver and bone marrow are most commonly affected. Pleural and pericardial involvement is also noted, evident as effusions. Miliary tubercles may be found studding other organs such as the intestine, kidney, fallopian tube, prostate, epididymis, adrenals, bone, brain, meninges, eye, skin and lymph nodes. Despite the presence of disseminated disease, the patients present with involvement of one particular organ itself.

PRIMARY PULMONARY TUBERCULOSIS :

Ghon's complexes are small, parenchymal lesions coupled with enlarged, ipsilateral hilar and rarely paratracheal nodes. The involved lymph node is much larger to the parenchymal focus. Certain other sites such as the posterior and apical segments of upper lobe, apical segment of lower lobe or upper part of right middle

lobe are also likely sites of Ghon's complex. Majority of the primary foci undergo calcification and few have caseous necrosis.

PROGRESSION OF TB :

The natural coarse of TB is influenced by age, sex, bacterial virulence, infective dose, natural and acquired immunity, few host factors aiding the disease to follow a scheme of progression according to Wallgren's timetable[31]. In the initial phase of the disease, the tuberculin conversion might produce a mild illness. In the initial years there is increased predilection to miliary spread and meningitis. The infection spreads from the primary focus by different routes namely direct extension into the surrounding tissue, endobronchial, vascular, lymphatic an disseminated spread. Ipsilateral or contralateral acinar pneumonia results from endobronchial spread. Implantation of bacilli in the upper airway mucosa can result in oral, laryngotracheal or middle ear TB. An enlarged caseous lymph node which perforates a patent bronchus, leads to massive lobar or segmental TB due to endobronchial spread. Visceral spread occurs via lymphatic dissemination. Haematogenous dissemination can result through the thoracic duct following lymph node involvement or by direct extension into branches of the pulmonary vein.

- HEALING : The dictum of primary lesions is healing. Reticulin and collagen deposition eventually replaces the calcific foci. Finally hyalinization occurs and demonstration of the lesions at this stage is difficult. A small proportion may display radiologically a residual hyalinized scar or calcified lesion at the primary lesion, in the lung parenchyma and in the paratracheal or hilar lymph nodes —a combination termed as the healed primary complex.
- EARLY GENERALISATION : Dissemination usually follows the primary infection. Lymphohematogenous spread occurs within hours to days following

the implantation of bacilli at the primary site[32]. Similar to the Simon foci in adults, which are just exaggerated calcific forms of the smaller primary foci, the Huebschmann lesions occur in children, observed as a group of nodular lesions in apex of the lung[33].

- LIQUIFACTION: Liquefaction is understood to be a result of delayed hypersensitivity to the caseous foci, accompanied by the activation of macrophages and hydrolysing enzymes[34]. The liquified area contained the active and dormant bacilli and hence are potential sources for transmission of the disease. Following the necrosis, a cavity may be formed which eventually communicates with a patent bronchus and thereby spreads to the larynx, alimentary canal and lungs. The liquified material can seep into the adjacent pleura causing pleural effusions and pneumothorax.
- LOBAR AND SEGMENTAL LESIONS : Due to spread along the submucosal lymphatics of bronchi, bronchi can undergo ulceration and rarely complete necrosis. A cold abscess may develop (endobronchial), which may be visualised radiologically. The lumen of the bronchus is narrowed either due to endobronchial lesion or due to compression by an infected lymph node. The lobe enfolded by the obstruction may lead to obstructive hyperinflation, atelectasis, secondary pneumonia, and disseminated intraalveolar epithelioid cell granulomas.

POST PRIMARY PULMONARY TB :

The areas involved in post primary TB are apical or sub-apical. This area has been called the ‘vulnerable region’ by Medlar[35]. The area is known to have relatively high oxygen tension due to gravitational forces on the ventilation perfusion ratio and the fact that the bacilli survive better in the areas with high oxygen due to limited

macrophage activity. Therefore progressive primary disease that is more common in the apical and posterior segments of the upper lobe. The bacilli multiply at other sites also such as ends of long bones, vertebrae and renal cortex. These areas are prone for high oxygen tension secondary to rich blood supply.

Reinfection in TB may be endogenous or exogenous. Endogenous route is due to activation of the seedling of infection within the apices of the lung whereas the exogenous route is due to bronchial spread from the index foci.

The pathological lesions in post primary TB are[35,36] :

1. Pulmonary lesions :
    - Lobar pleumonia
    - Nodular TB – small, large, healed nodules
    - Fibrocaseous TB – with or without cavitation
    - TB broncopneumonia
2. Bronchial lesions :
    - Bronchial inflammation
    - Endobronchial TB
    - Bronchiectasis
3. Whole lung TB
4. Miliary TB
5. Complications :
    - Haemoptysis
    - Aspergilloma
    - Amyloidosis
    - Carcinoma
    - Oral and respiratory tract TB

6. Pleural lesions

REACTIVATION AND REINFECTION: TB in elderly can be either due to exogenous infection or reinfection or endogenous reactivation, the most common being endogenous reactivation, especially when the community spread is low[52]. Exogenous infection and reinfection are acquired usually due to exposure to a sputum smear-positive case, while endogenous reactivation occurs from lesions that have remained silent either in the lung or other parts of the body. The change in immune pattern of the body as a part of ageing includes reduction in the T-cell populations leading to ineffective control of infection.

The factors that contribute to the reduction in immunity are[53]:

1. malnutrition
2. comorbidities like cancer or diabetes mellitus
3. gastrointestinal surgery [gastrectomy, jejunoileal bypass]
4. immunosuppressive therapy
5. tobacco smoking
6. human immunodeficiency virus [HIV] infection.

Studies have also suggested that tumour necrosis factor(TNF)-α blocking drugs [e.g., infliximab] reduce the T-cell related immunity and exaggerate the reactivation of latent TB[54]. It has also been observed that higher the rates of incidence in younger population, higher the prevalence of TB in elderly[55].

LATENT TB INFECTION IN ELDERLY: A positive tuberculin skin test(TST), indicates an infection with mycobacterium TB and is indicated by an induration of greater than 10 millimetres with an intradermal injection of five tuberculin units of purified protein derivative. The test is not reliable in the elderly due to a slowly fading

immune response in this population, hence the positivity in the elderly is a risk factor for development of active TB.

THE MYCOBACTERIUM:

The generic name Mycobacterium meant mold like (myco: fungus; bacterium: bacteria) and was introduced by Lehmann and Neumann in 1896[37]. The genus Mycobacterium contains more than 50 species, of which several are non-pathogenic bacteria. Mycobacteria other than human or bovine tubercle bacilli causing human disease resembling TB are termed as atypical mycobacteria.

The tubercle bacilli are straight or slightly curved (rod shaped), slender organisms measuring two to four micrometre in length occurring singly, as pairs or in small groups. The bacilli are non-motile, non-sporing and non-capsulated. In liquid culture media, tubercle bacilli form characteristic long, tight, serpentine cords in which bacilli are arranged in parallel. The bacilli are Gram positive, but they do not stain readily. They resist decolourization by 25 per cent sulphuric acid and absolute alcohol for 10 minutes and hence they are termed as acid and alcohol fast. Acid fastness is due to the integrity of the cell wall. Mycobacteria are obligate aerobes. Optimum growth occurs at 37 °C and growth is stunned below 25 °C and above 40 °C. Optimum pH for growth is 6.4 to 7.0.

The various components in the mycobacterium have different pathological activities as follows :

1. Cell wall : induces resistance to infection, causes delayed hypersensitivity
2. Tuberculoprotein: elicits tuberculin reaction, induces delayed hypersensitivity, induces formation of epithelioid and giant cells.
3. Polysaccharides : induces immediate hypersensitivity, causes exudation od neutrophils from blood vessels

4. Lipids : causes accumulation of macrophages and neutrophils
5. Phosphatides : induce formation of tubercles

For the laboratory diagnosis of TB sputum is the sample of choice in case of pulmonary TB. In case of TB of any other organ, the specimen or the by-products of the function the involved organ need to be collected as cerebrospinal fluid in TB meningitis and urine in renal TB.

The best is to obtain an early morning sputum sample, before the patient has consumed food, for the simple reason that food particles can make it difficult to examine the sample[38]. While the patient is giving the sputum sample, the bacilli can be discharged into the air and spread via aerosols. Hence instructions need to be given to the patients to collect the sample out in the open air away from people or away from crowded areas. The TB bacilli are discharged into the sputum intermittently, therefore three samples are advised to be collected as follows:

1. one spot specimen when the patient first seeks medical attention
2. one early morning specimen [preferably the next day]
3. one spot specimen, concurrently along with the early morning specimen.

These are to be sent to the laboratory as separate specimens. Once the patient has sputum production, the sample collection is simple, whereby the patient is given a container on his first attendance and instructions are given as follows for the collection :

1. to inhale deeply two to three times before coughing
2. to cough out deep from the chest
3. to open the container and spit into the bottle
4. to avoid saliva or nasal secretions
5. to close the container.

A good sputum specimen should be at least 5ml, thick and purulent. The patient details are written along with the specimen and sent to the laboratory. Specimens should be transported to the laboratory as soon as possible after collection. The specimens once collected are to be sent to the laboratory immediately, failing which they are to be stored at a cool place to prevent the growth of unwanted micro-organisms. In absence of a refrigerator for storing the sample before transport, it is diluted with equal quantity of one per cent cetyl pyridinium chloride in two per cent saline and later sent to the laboratory. The current recommendation from the RNTCP requires only 2 samples for testing, the early morning sputum sample and the spot sample given along with the early morning specimen.

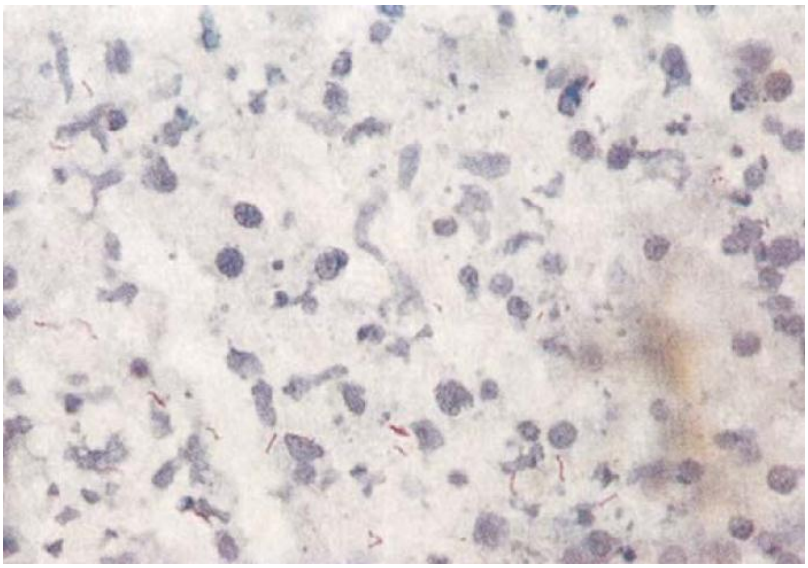
STAINING OF TB BACILLI :

The TB bacilli generally do not take up the stain readily, but due to the acid fastness, they retain the primary stain even after decolourisation with acid-alcohol. A counter-stain is used to highlight the stained organisms for contrast. The standard Ziehl-Neelson(ZN) staining was discovered in 1883 and the procedure is as follows :

1. Prepare the smear on a clean glass slide by using sterile precautions
2. Heat fix the prepared smear and alcohol fixation if the sample is not bleached
3. Add carbol fuchsin to the smear
4. Heat the slide until flames begin to rise and rest it for five minutes
5. Wash the slide with clean water so as to clear away all the stain
6. Cover the slide with three percent acid-alcohol for five minutes until the slide is decolourised
7. Wash with water
8. Add malachite green stain for two minutes and allow for the slide to dry
9. Examine under oil immersion using 100 X objective

The acid fast bacilli(AFB) will be seen as bright red to purple straight or curved rods in groups or beads. The staining can be graded based on the number of AFB detected as follows:

NUMBER OF AFB	FIELDS	REPORT
NONE	PER 100 OIL IMMERSION FIELDS	NEGATIVE
1-9	PER 100 OIL IMMERSION FIELDS	SCANTY
10-99	PER 100 OIL IMMERSION FIELDS	1+
1-10	PER OIL IMMERSION FIELD(50 FIELDS EXAMINED)	2+
>10	PER OIL IMMERSION FIELD(20 FIELDS EXAMINED)	3+



ACID FAST BACILLI SEEN ON ZEEHL-NEELSON STAINING IN TB OF LYPMH NODE.

ROENTGENOGRAPHIC MANIFESTATIONS OF PULMONARY TB :

Various imaging modalities are available for aiding the diagnosis of TB like chest X-ray, ultrasonography, computerised tomography(CT), magnetic resonance imaging(MRI). For the purpose of simplification, pulmonary TB can be divided into :

1. Primary TB

2. Post-primary TB

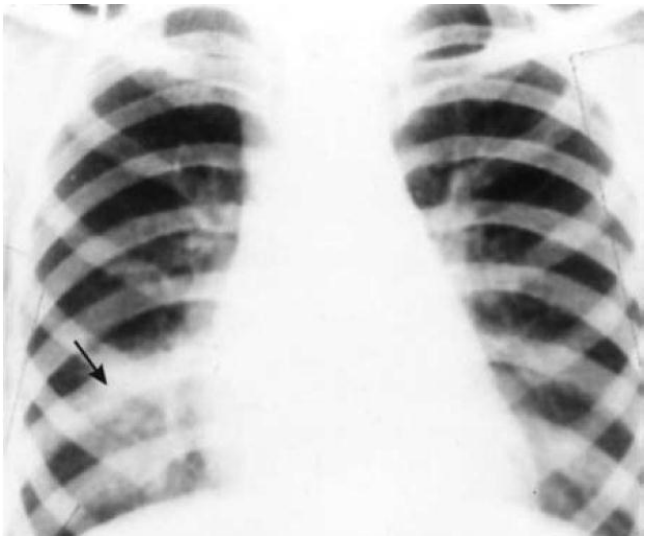
Although the lesions are characteristically different, there is a considerable amount of overlap between them in routine practice.

PRIMARY TB :

It may involve one or more of the following structures like lung parenchyma, tracheobronchial tree, pleura and lymph nodes. Up to fifteen percent of cases demonstrate a normal study. The various radiographic features visible in primary pulmonary TB are :

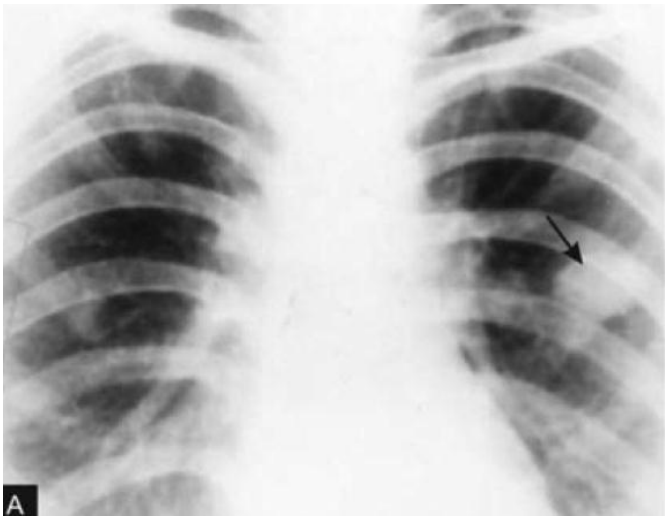
- Parenchymal consolidation
- Tuberculoma
- Miliary tuberculosis
- Airway involvement
- Pleural effusion
- Lymphadenopathy

PARENCHYMAL CONSOLIDATION: It is usually isolated, of variable size(less than two centimetres in diameter), homogeneous with poorly defined margins. Sometimes endobronchial obstruction can cause pan-lobar involvement[39]. Consolidation abutting a fissure displays sharp margins. Differentiation between bacterial pneumonia and pneumonia of tubercular origin is challenging at a radiographical level, but the lack of systemic toxicity along with associated lymphadenopathy and lack of response to conventional antibiotics favours a diagnosis of tubercular pneumonia[39]. There is no lobar or segmental preference for the development of pneumonia, although the right lung is more commonly involved than the left owing to the anatomy. Consolidation is seen as an attenuated homogeneous soft tissue lesion with air bronchogram or areas of breakdown within.



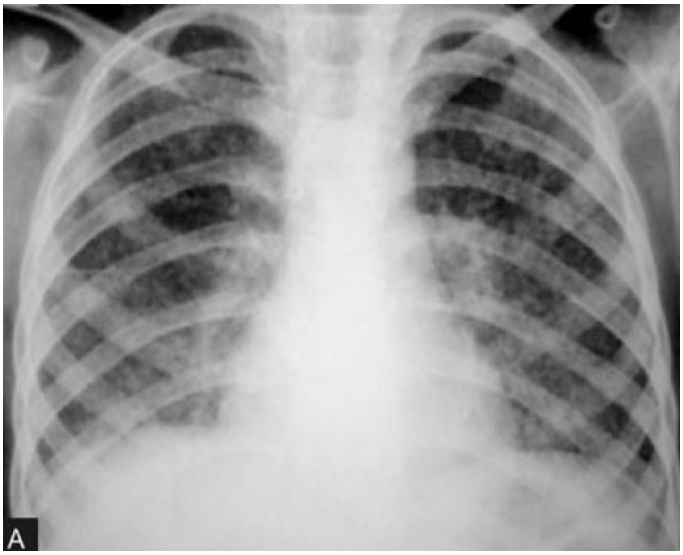
RIGHT LOWER LOBE CONSOLIDATION.

TUBERCULOMA : A tuberculoma may be a feature of either primary or post-primary TB and is seen as a persistent mass-like opacity. It is limited to seven to nine per cent of patients with TB. It is round to oval shaped, commonly seen in the right upper lobe. Satellite lesions are small, discrete lesions seen around the main lesion in as most of the cases[40]. Tuberculomas are usually less than three centimetres in diameter. Cavitation's may be seen in up to fifty percent of the cases. Calcification within the nodule or satellite nodules are common.



LEFT UPPER LOBE TUBERCULOMA

MILIARY TB: This form of TB too can be seen in both primary and post-primary forms and is detectable radiologically in up to seven percent cases[41]. During the initial phases of dissemination, the lesions are too small to be detected on radiological imaging. A period of at least six weeks from the initiation of dissemination has been documented for demonstration of lesions radiographically. When the lesions increase in size, they appear as discrete, small, pinpoint opacities, evenly distributed within the bilateral lung fields with basal predominance, attributed to the gravity dependant excess in blood flow to the basal lung fields. The lesions are as small as one millimetre in diameter to begin with and increase to around five millimetre in diameter if treatment is not initiated, which when visualised radiographically appears as “snow-storm ” appearance[40]. Associated lymphadenopathy and pneumonia are seen in a minor number of cases. Some cases which cannot be picked up on a routine chest X-ray are readily demonstrable on high resonance CT of the chest. Radiographic clearance begins to appear by two to six weeks.



MILIARY TB.

LYMPH NODAL INVOLVEMENT: Lymphadenopathy is the most common manifestation of lymph nodal involvement and is a feature more prominent in children when compared to adults[42]. This form of lymphadenopathy is essentially linked with a primary parenchymal lesion evident by the enlargement of the draining lymph nodes, most commonly unilateral hilar and para-tracheal nodes. Again, CT scan is more reliable for the diagnosis of lymph nodal involvement, which demonstrates low attenuation in the centre along with peripheral rim enhancement[43]. Such nodes can be up to five centimetres in diameter. Rim enhancement seen on the CT scan may be of the following types :

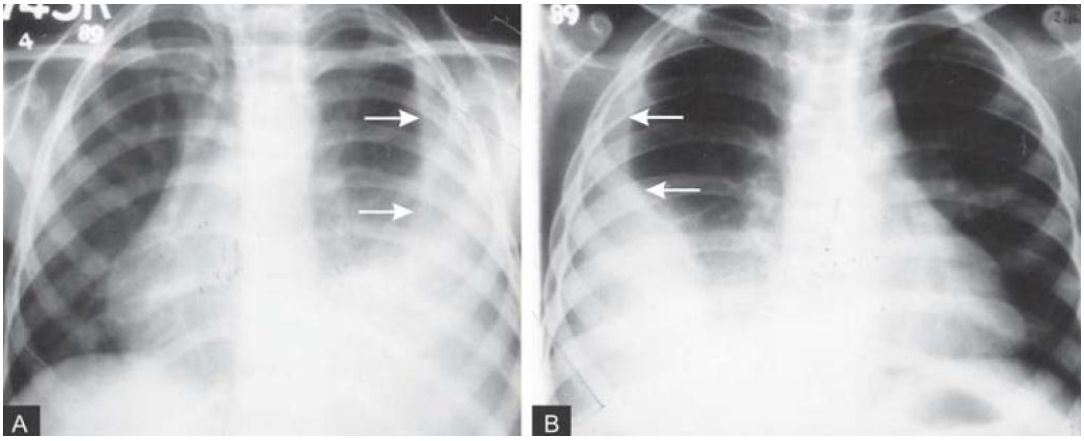
1. thin, uniform and complete enhancing rim
2. thick, irregular, complete or incomplete peripheral rim
3. group of conglomerated nodes showing central and peripheral areas of rim enhancement

The findings on the CT can be correlated with the histological findings on biopsies demonstrating areas of complete central necrosis with a highly inflammatory,

vascular and capsular perinodal reaction making the rim[43]. The enlargement of the lymph nodes can be homogenous or non-homogenous, with the latter being larger an depicting the "ghost-like" ring enhancement radiologically. Calcified lymph nodes generally are inactive in nature.

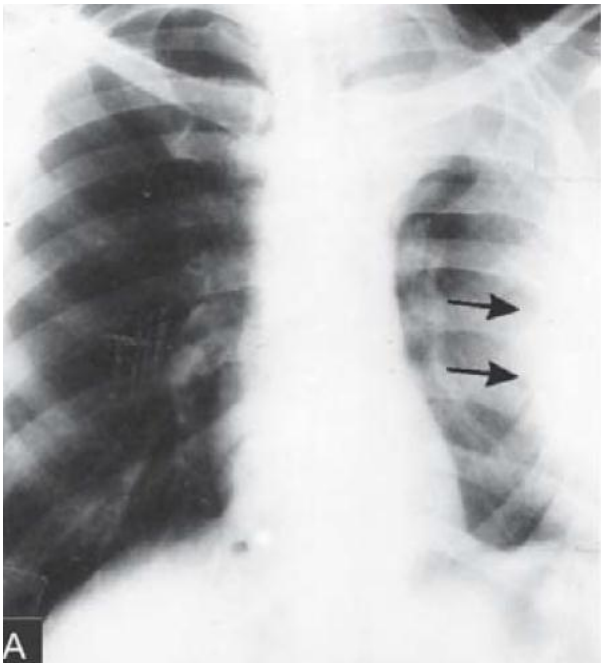
AIRWAY INVOLVEMENT: It is due to compression from the enlarged lymph nodes extrinsically or intrinsically by endobronchial TB, either of which causes lobar or segmental atelectasis in the anterior segment of upper lobe[44]. The cause of airway involvement can be well delineated on the high resolution CT scan of the chest.

PLEURAL INVOLVEMENT: Pleural effusion is the most prominent manifestation of primary TB in the pleura and occurs in a considerable amount of adults. It is mostly unilateral, non-loculated and moderate to large in quantity, in which case there is a definite mediastinal shift. Sub-pulmonic effusions produce an apparent elevation in the dome of the diaphragm. In case of doubt, a left lateral X-ray can be done to demonstrate shift of the fluid along the lateral border. Empyema, empyema necessitates, broncho-pleural fistula may be potential complications. Ultrasonography is beneficial to differentiate between pleural effusion, thickening, empyema and also helps in diagnosing small pleural effusions which cannot be seen in regular chest X-ray. CT scans are of importance in the diagnosis and prognostication of complicated pleural effusions as they explain even the extent and involvement of other surrounding structures if any.



PLEURAL EFFUSION WITH (A)MEDIASTINAL SHIFT , (B) LOCULATED.

LEFT SIDED PLEURAL THICKENING.



POST-PRIMARY TB :

Majority of cases are a result of reactivation of an infective focus acquired in earlier life. Sometimes, it results from infection by virulent organisms in individuals who have been vaccinated with Bacille Calmette-Guerin [BCG]. In rare cases, there are new exogenous infections. There various radiographic features seen in post-primary TB and there is considerable amount of overlap between the primary and post-primary TB.

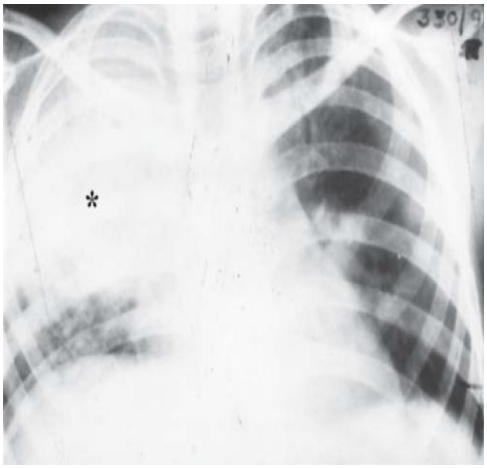
The following features suggest post-primary TB:

1. predilection for upper lobe involvement
2. favourable to develop cavitation
3. rarity of lymphadenopathy

The various radiographic features of post-primary TB are :

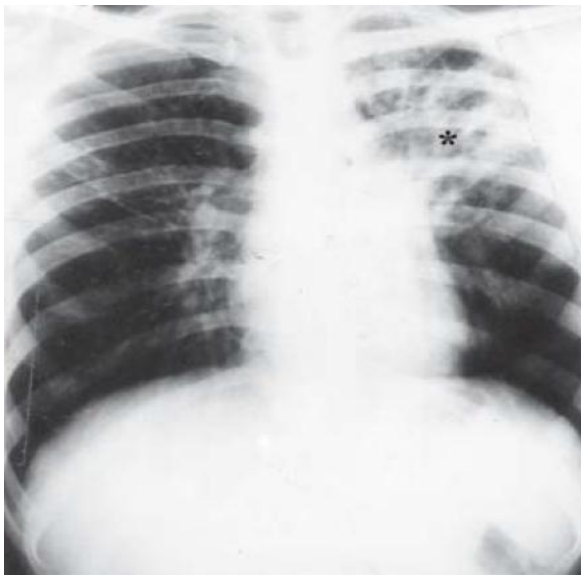
- Local exudative lesion
- Local fibroproductive lesion
- Tuberculoma
- Cavitation
- Bronchogenic spread
- Miliary tuberculosis
- Bronchostenosis
- Pleural disease

EXUDATIVE LESIONS: Patchy, ill-defined and confluent consolidation occurs in the apico-posterior segments of upper lobe or the superior segment of lower lobe usually involving more than one segment. Bilateral upper lobe involvement and calcifications respond well to treatment when started early. Exudative lesion if left untreated may cause lobar or total lung consolidation.



MASSIVE RIGHT UPPER LOBE CONSOLIDATION.

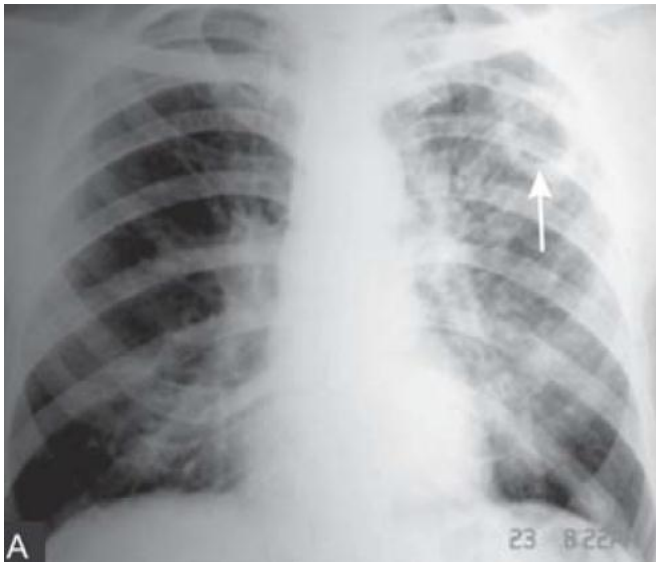
FIBROPRODUCTIVE LESIONS : Exudative lesions are replaced by reticulo-nodular opacities. The entire granulation tissue is replaced by fibrous tissue as a part of healing thereby causing significant volume loss depicted by tracheal and hilar retraction, elevation od diaphragm. Radiological evidence is usually seen when there is ongoing conversion from the exudative to fibrous lesions.



LEFT UPPER LOBE FIBRO-PROLIFERATIVE LESION.

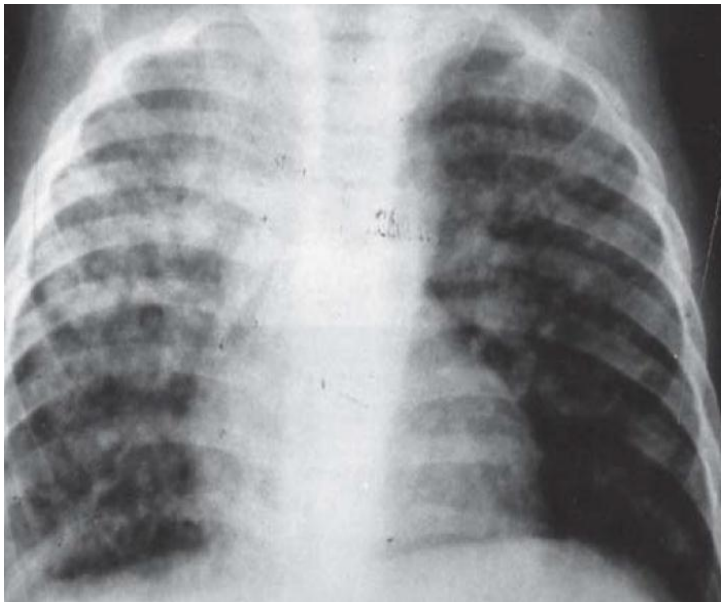
CAVITATION: Following liquefaction, the debris is pushed into the bronchial tree leaving behind a cavity. Cavitation within an existing consolidation is a striking feature of post-primary TB and indicates an active disease. These cavitation's are seen

radiologically as lesions of varying size and are usually multiple[45]. The wall of a cavity can be thick or thin, uniform or nodular and usually involves the apico-posterior segments of upper lobe in and the superior segments of lower lobe. Generally, there is wide opening bronchus that drains the cavity.



LEFT UPPER LOBE CAVITATION.

TUBERCULAR BRONCHPNEUMONIA : Endobronchial spread or intrabronchial rupture of an infected lymph node produces bronchial dissemination causing ill-defined parenchymal lesions predominantly in the dependant areas of either of the lungs. Such extension may result in an acute pneumonia, which can be differentiated from other causes of pneumonia by the presence of an open cavity and satellite nodules in the lung[40]. This pattern of spread producing nodules are best visualised with a high resolution CT scan of the chest which shows “tree-in-bud” appearance in the peribronchial and centrilobular regions. These lesions are the spreading infection producing impaction of the caseous material in the distal bronchioles, indicating active disease[46].



BRONCHOSTENOSIS : It occurs in a minor number of patients with active disease due to endobronchial spread, direct extension from an infected lymph node or lymphatic dissemination to airways. CT scan is extremely effective in depicting the stenosis[47].

PLEURAL EFFUSION : It is more a feature of primary TB rather than post-primary TB and if present is seen small loculated effusions, which are less likely to be picked up on regular chest X-ray. Frank empyema is very rare in these cases[39].

As established already that the most common form of TB in elderly is from the reactivation of old quiescent lesions, the typical sites for such activity are apical segments of the upper and lower lobes and posterior segments of upper lobe. The presentation may vary from a pneumonia to caseation and liquefactive necrosis and cavity formation. It can be generalised that TB should be kept as a diagnosis in all elderly presenting with pleuropulmonary symptoms, signs and radiographic infiltrates. Such patients usually have cough, expectoration and haemoptysis. The chest radiography reveals fibronodular or cavitary lesions in the upper zones along with hilar and perihilar enlargements. The above described features are usually seen in TB due to reactivation or reinfection. In contrast, the primary TB infection in elderly

present with more constitutional symptoms with the radiographic prominence on the mid and lower zones of the lung[58]. Hence it is a task to diagnose TB in the elderly and it has to be done promptly in order to prevent further spread and related morbidity and mortality.

SYMPTOMS OF PULMONARY TB :

Pulmonary TB has variable manifestations and mimics many diseases. Historically, the development of erythema nodosum, phlyctenular conjunctivitis and fever at the time of tuberculin conversion was considered common, although extremely rare now[48]. Usually the symptom onset is insidious and few patients are asymptomatic, but majority of the patients have constitutional or respiratory symptoms[49]. Constitutional symptoms consist of headache, weight loss, loss of appetite, tiredness, fever and night sweats. The younger population generally present with classical symptoms while the elderly present with atypical symptoms.

Fever begins initially as low-grade and progresses to high-grade with time and is usually present during the late afternoon or evening while some may remain afebrile. Weight loss may precede the symptoms. The most striking respiratory symptom of pulmonary TB is cough which generally persists for three or more weeks, may be dry or productive, making it very difficult to differentiate it from other respiratory causes like smoking. Sputum is usually scanty and may be mucoid, purulent, mucopurulent or blood-tinged. Haemoptysis is a cardinal feature and in the Indian perspective TB is the most common cause of haemoptysis. It may vary from minute blood-stained sputum to massive haemoptysis occurring most commonly due to a ruptured bronchial artery[50]. Chest pain may occur in TB due to a variety of complications. Localised wheeze may be present due to lymph node pressing on the airway.

Symptoms such as fever, weight loss, anorexia are common in the elderly patients who present with TB. The fact that these patients often do not give an accurate history owing to poor memory, dementia, speech impairment and other fallacies in communication makes the disease more burdensome. Also, co-existing diseases such as cancer and AIDS, can mask and confuse the clinical picture. Rarely, patients present without fever, but marked weight loss is present which points towards a progressive metastatic carcinoma, but eventually is a feature of miliary TB termed as cryptic miliary TB[56]. There is a marked difference in the presentation of TB between the young elderly especially with respect to the paucity of respiratory symptoms and presence of atypical symptoms in the elderly in contrast to a rather direct presentation in the younger population[57]. The non-specific symptoms that the elderly can present with include changes in the activity of daily living, chronic fatigue and unrelatable weakness, cognitive impairment, anorexia and weight loss, and chronic low grade fever which is not typical of TB. The onset of the symptoms, if present in the elderly are more delayed and also the duration of symptoms are also prolonged and usually non-resolving. These instances should hint the clinician to keep TB in the geriatric subset of population as a differential diagnosis. One of the many reasons why the diagnosis of TB in the elderly is delayed is due to the masking of the symptoms of TB by the other chronic illnesses that co-exist in the elderly like chronic bronchitis, emphysema and urinary tract infections which usually confuses the clinician in regard to the fever pattern.

The major differences between young and geriatric TB are as follows :

VARIABLES	YOUNG	ELDERLY
CONSTITUTIONAL SYSMTOMS :		
FEVER	PRESENT	ABSENT
NIGHT SWEATS	PRESENT	ABSENT
NON-SPECIFIC SYMTOMS LIKE DIZZINESS, MENTAL DULLNESS	PRESENT	ABSENT
RSPIRATORY SYMPTOMS :		
COUGH	PRESENT	ABSENT
HAEMOPTYSIS	PRESENT	ABSENT
DYSPNOEA	ABSENT	PRESENT
CO-MORBIDITIES LIKE DIABETES, STROKE	ABSENT	PRESENT
HYPOALBUMINEMIA	ABSENT	PRESENT
TST POSITIVITY	PRESENT	ABSENT
ADVERSE DRUG REACTIONS	ABSENT	PRESENT

PHYSICAL SIGNS OF PULMONARY TB:

A good general physical examination can reveal the presence of anaemia and severe cachexia owing to the marked reduction in appetite in chronic cases of TB. Anasarca, change in colour and texture of hair, leukonychia, low body mass index(BMI) may be present owing to chronic protein energy malnutrition. Fever produces tachycardia, commonly accompanied by tachypnoea. Rarely, digital clubbing may be seen along with superadded suppuration. Foci of extra-pulmonary infection may be seen in the form of cold abscess, mesenteric and cervical lymphadenopathy, spinal deformity, epididymitis, keratitis or phyctenular

conjunctivitis. When the nervous system involvement is present, signs of focal neurological deficits and meningeal irritation may be apparent.

The respiratory system involvement depends on the underlying pathology.

Consolidation:

- Trachea is central with no gross mediastinal shift as there is no increase in the volume of the lung.
- Movements of the lung fields are reduced in the areas of consolidation.
- Tactile vocal fremitus and vocal resonance are increased.
- Dull note on percussion is present,
- Tubular breath sounds may be heard along with fine or coarse crackles.

Collapse :

- Trachea and mediastinal shift to the side of the collapse is observed.
- Movements of the chest are reduced on the side of collapse of minor bronchus and absent in case of major bronchus obstruction.
- Tactile vocal fremitus and vocal resonance are usually reduced/absent in case of major bronchus obstruction and may be increased/unchanged in minor bronchus obstruction.
- Percussion note is dull in case of major bronchus obstruction and impaired in case of minor bronchus obstruction.
- Breath sounds are usually absent in major bronchus obstruction and reduced in minor bronchus obstruction with no added sounds.

Fibrosis:

- Trachea and the mediastinum is pulled towards the side of fibrosis. The undue prominence of the clavicular head of sternocleidomastoid muscle due to the pull of the trachea is termed as Trail's sign.
- Movements of the chest are reduced.
- Tactile vocal fremitus and vocal resonance are variable depending on the cause.
- Percussion note is impaired.
- Breath sounds are diminished with no added sounds.

Cavitation:

- Mediastinum and trachea may remain unchanged or pulled to same side depending upon the size and the wall of the cavity.
- Movements of the chest are usually unchanged.
- Tactile vocal fremitus and vocal resonance are increased.
- Percussion note is impaired.
- Breath sounds are of cavernous type and are accompanied by fine crepitations.

Pleural effusion or Empyema:

- Mediastinum and trachea are shifted to the side opposite side of the effusion.
- Movements over the areas of effusion and empyema are reduced.
- Tactile vocal fremitus and vocal resonance are reduced.
- Percussion note is stony dull.
- Breath sounds are either reduced or absent and a pleural rub may be heard above the level of the effusion.

Pneumothorax:

- Mediastinum and trachea are shifted to the opposite side.
- Movements over the chest wall are reduced.
- Movements over the areas of effusion and empyema are reduced.
- Percussion note is hyper-resonant
- Breath sounds are reduced or absent.

LABORATORY DIAGNOSIS OF TB:

The most common specimen tested for TB is the sputum and has been discussed earlier. The other possible specimens that can be tested for the diagnosis of TB include bronchial washings and brushings, broncho-alveolar lavage fluid, gastric lavage fluid, cerebrospinal fluid, urine, serous fluid and tissue. The other staining methods used for demonstrating AFB apart from the Zeehl-Neelson stain include Kinyoun's stain and Gabett's solution which also work by the same principles as Zeehl-Neelson stain. Fluorescent staining with Auramine-Rhodamine dyes are upcoming due to the ability to visualise bacilli with lower power of magnification, allowing a larger area to be seen at a single visual period.

The disparity in the early diagnosis of TB between the young and the elderly lies in the fact that firstly, the clinical suspicion of Tb in the elderly is less in comparison to the young owing to the varied and vague clinical presentation and secondly, the difficulty in obtaining the samples for testing. The fact that the elderly cannot express the early morning sputum sample makes it a strenuous task to diagnose TB in the elderly as sputum is one of the mainstay specimen tested for the diagnosis of TB. In such cases the use of percussion and warm steam and saline nebulisations can help in obtaining the early morning sputum sample. When the patients are comatose it is impossible to extract the sputum sample and hence

becomes imperative to depend on either gastric lavage or broncho-alveolar lavage samples.

Also the elderly are bound to think of themselves as a burden to the family and hence they do not inform the attenders regarding the symptoms that they have, which in turn delays the diagnosis and eventually the initiation of treatment in such patients. The task of extraction of history is indeed an onerous job that has to be done with precision and empathy for the elderly to feel comfortable and open up to the clinician regarding the exact mode of presentation.

The paucity of definitive symptoms along with the merging of other symptoms and also the unawareness of the elderly towards the disease process contribute majorly towards the underdiagnosis and undertreatment of geriatric TB.

MYCOBACTERIAL CULTURE:

The TB bacilli can be detected by cultures even when present in extremely small quantity and susceptibility to antibiotics, viability and species differentiation can be demonstrated by the cultures. TB bacilli have an extremely slow generation time of up to two days and require many specific growth media and environmental factors to allow for the growth in contrast to other bacteria, hence although tedious the culture is the gold standard for the diagnosis of TB[59]. The most common among the media used for culture is the Lowenstein-Jensen media containing eggs, glycerol, asparagine and mineral acids, and growth is seen in two to eight weeks at an optimal temperature of thirty seven degrees and optimum pH .

The immunodiagnostics of TB include antibody detection of the IgG and IgM immunoglobulins, antigen detection and the nucleic acid amplification tests(TB-NAAT).

TREATMENT OF TB:

The goals for treating TB are:

1. to reduce the morbidity and mortality by preventing relapse
2. reduce incidence of drug resistance
3. to break the chain of transmission thereby reducing the infective pool

The cases can be classified according to history of prior treatment for TB:

1. NEW CASES: The patient has either never taken treatment for TB or treatment taken for less than one month.
2. PREVIOUSLY TREATED PATIENTS: Has received one month or more of anti-TB drugs in the past.

- Recurrent TB case – patient who has completed the course of treatment successfully in the past and subsequently found to have TB.
- Treatment after failure – patients who have taken treatment but declared as ineffective by the end of the most recent therapy.
- Treatment after loss to follow up – patient has taken treatment for one month or more and failed to follow up after that period and subsequently found to have TB.
- Other previously treated patients – patients who have been treated for TB but the records are unknown or undocumented.

3. TRANSFERRED-IN : Cases that are diagnosed at one TB unit and received treatment in another TB unit.

The cases can also be classified on the basis of drug resistance :

1. Mono-resistance : resistant to first line anti-TB drug only.
2. Poly-drug resistance : resistance to more than one first line anti-TB drug except isoniazid and rifampicin.
3. Multi-drug resistance : resistance to both isoniazid and rifampicin with or without resistance to other first line drugs. Patients with rifampicin resistance without resistance to isoniazid are to be considered under this category itself.
4. Extensive-drug resistance : multi-drug resistance TB patient with added resistance to a fluoroquinolone(moxifloxacin/ofloxacin/levofloxacin) and a second line injectable anti-TB drug(amikacin/kanamycin/capreomycin).

The Revised National TB Control Programme(RNTCP) had adopted a thrice weekly regimen until now, which has changed to a daily regimen, the principle behind it being able to administer a fixed dose of the first line anti-TB drugs in appropriate weight bands.

TYPE OF TB CASE	REGIMEN OF INTENSIVE PHASE	REGIMEN OF CONTINUATION PHASE
NEW	(2) HRZE	(4) HRE
PREVIOUSLY TREATED	(2) HRZES + (1) HRZE	(5) HRE

The prefix to the drugs stand for the duration in months.

MULTI-DRUG RESISTANCE(MDR) TB / RIFAMPICIN RESISTANCE(RR) TB - WITHOUT ADDITIONAL RESISTANCE : These patients are subjected at resistance check to levofloxacin and kanamycin at baseline and modifications made accordingly.

TYPE OF TB CASE	REGIMEN OF INTENSIVE PHASE	REGIMEN OF CONTINUATION PHASE
RIFAMPICIN RESISTANCE + ISONIAZID SENSITIVE/UNKNOWN	(6-9) Km Lfx Eto Cs Z E H	(18) Lfx Eto Cs E H
MDR-TB (treatment is modified based on isoniazid resistance)	(6-9) Km Lfx Eto Cs Z E	(18) Lfx Eto Cs E

EXTENSIVE DRUG RESISITANCE(XDR) TB:

TYPE OF TB CASE	REGIMEN OF INTENSIVE PHASE	REGIMEN OF CONTINUATION PHASE
XDR-TB	(6-12) Cm, PAS, Mfx, high dose – H, Cfz, Lzd, Amx/Clv	(18) PAS, Mfx, high dose – H, Cfz, Lzd, Amx/Clv

MDT-TB/ RR-TB WITH ADDITIONAL RESISTANCE:

The treatment in such cases can be modified according to the drug that is resistant is addition as follows:

TYPE OF TB CASE	REGIMEN IN INTENSIVE PHASE	REGIMEN IN CONTINUATION PHASE
MDR/RR TB + ETHAMBUTOL RESISTANCE	(6-9) Km Lfx Eto Cs Z	(18) Lfx Eto Cs
MDR/RR TB + PYRAZINAMIDE RESISTANCE	(6-9) Km Lfx Eto Cs E	(18) Lfx Eto Cs E
MDR/RR TB + PYRAZINAMIDE+	(6-9) Km Lfx Eto Cs PAS	(18) Lfx Eto Cs PAS

ETHAMBUTOL RESISTANCE		
MDR/RR TB + LEVOFLOXACIN RESISTANCE	(6-9) Km Mfx Eto Cs Z E PAS Cfz	(18) Mfx Eto Cs E PAS Cfz
MDR/RR TB + MOXIFLOXACIN RESISTANCE	(6-9) Km Lfx Eto Cs Z E PAS Cfz	(18) Lfx Eto Cs E PAS Cfz
MDR/RR TB + RESISTANCE TO ALL FLUOROQUINOLONES	(6-9) Km Eto Cs Z E PAS Cfz Lzd	(18) Eto Cs E PAS Cfz Lzd
MDR/RR TB + RESISTANCE TO KANAMYCIN	(6-9) Cm Lfx Eto Cs Z E	(18) Lfx Eto Cs E
MDR/RR TB + RESISTANCE TO ALL SECOND LINE INJECTABLE DRUGS	(6-12) Lfx Eto Cs Z E PAS Cfz Lzd	(18) Lfx Eto Cs E PAS Cfz Lzd

BEDAQUILINE: It is a novel class of drug, a diarylquinoline that targets the adenosine triphosphate of the mycobacterium, thereby reducing the energy supply in the bacteria. No cross reactions have been demonstrated with the other first and second line anti-Tb drugs and is recommended for treatment of MDR-TB. The drug is metabolized through the liver and has extensive tissue binding capacity and an extended half-life.

DRUG DOSAGE FOR ADULTS: The tablets are usually administered depending on the weight band of the patient and hence are prepared as fixed dose combinations (FDC).

Weight category	Number of tablets (FDCs)		Inj. Streptomycin
	Intensive phase	Continuation phase	
	HRZE	HRE	
	75/150/400/275	75/150/275	gm
25-39 kg	2	2	0.5
40-54 kg	3	3	0.75
55-69 kg	4	4	1
>=70	5	5	1

In the elderly, the main categories of treatment the individual drugs under each of the described categories remain the same, but there are various challenges that are faced with regard to the treatment in elderly such as :

1. The elderly are somehow dependant on their children or other people for simple things like even getting to come to terms with the fact that they have been diagnosed with TB. Hence, the emotional and psychological support needs to be given To them so that they can be taken Into confidence and it will be easier for them to take the desired drugs.
2. The most common comorbidity in the elderly is diabetes mellitus and habits like smoking, alcohol consumption and lack of physical activity aggravate the disease process of TB and also pose remarkable risk of treatment failure.
3. The medications for TB will only add to the already existing medications that the elderly are taking for various ailments like MI and stroke making it very difficult for them to take so many medications and also hard to remember the frequency and schedule of so many drugs.
4. The medications for TB are large capsules although single thereby producing difficulty in swallowing for the elderly.
5. The regular follow up for obtaining TB medications from the RNTCP centres is very difficult in the elderly, thereby making them discontinue the medications and becoming potential sources of relapse.

6. Another important fact to be considered in the elderly are the innumerable drug interactions between the various medications making the effect of a few drugs sub-standard and excessively metabolised causing underactivity.
7. The adverse reactions in the elderly are also to be noted as this subset of the population cannot tolerate them as well as the younger population can.
8. The lack of awareness among the elderly is also responsible for the ignorance of the symptoms of ill health and delay in seeking medical attention.
9. The elderly also tend to first consult a local doctor/quack who can misdiagnose TB or who may not consider the possibility of TB due to the masking of symptoms by other comorbidities. The usage of several unnecessary drugs given by such practitioners can cause early the elderly to acquire resistance to drugs.
10. The elderly have higher rates of default in comparison to resistance thereby making it important to see that they do not acquire resistance easily.
11. The lack of definitive diagnostic criteria for TB in the elderly make it imperative to impose screening of all the elderly to TB by means of chest radiography and sputum analysis when suspected in a particular case.

Therefore, although it is an excellent initiative by the government to make a single capsule incorporating all the drugs in optimum dosages, further insight into reducing the frequency of the intake of tablets and other modes of drug delivery systems like amorphic powder sachets. It further is the responsibility of the social workers and the doctors to inculcate the need for seeking medical attention and early recognition of the disease activity among the elderly to ensure lesser rates of morbidity and mortality among the elderly due to TB.

MATERIAL AND METHODS

The study titled " A study of clinical and radiological profile of sputum positive pulmonary tuberculosis among elderly patients" was conducted in Shri B. M. Patil Medical College and Hospital, Vijayapura.

The **aims** of the study were:

- To study the various patterns of presentation of sputum positive pulmonary tuberculosis in geriatric population.
- To study the radiological features in these patients.

Selection of cases:

The study was conducted among the geriatric patients(above the age of 60 years), who either attended the Medicine out patient department or who were admitted to the wards in Shri B. M. Patil Medical College and Hospital, Vijayapura .

Period of study:

The study was conducted between December 2018 and June 2020.

Sample size:

70 patients were chosen to be the sample size for the study.

Inclusion criteria:

- All elderly patients aged more than 60 years who are positive for sputum acid fast bacilli.
- Sputum positive pulmonary tuberculosis, irrespective of category treatment.

Exclusion criteria:

- Age less than 60 years.
- Patients with radiological features suggestive of tuberculosis, but sputum negative.

Protocol of the study:

All the patients who satisfied the inclusion and exclusion criteria were chosen and the sputum acid fast bacilli reports were obtained to confirm the positivity for tuberculosis. These patients were now subjected to the questionnaire prepared in the proforma which includes detailed history and examination of the patient. The patient is then subjected for chest X-ray for identifying the various radiological lesions. Regular blood investigations are also done and the treatment if initiated is duly noted. The observations made were tabulated and the results were analysed with specific regard to various patterns of clinical presentations and radiological appearances.

ANALYSIS AND RESULTS:

Our Study was conducted in Shri B. M. Patil Medical College and a total of 70 patients were studied who satisfied the inclusion criteria and the following observations were made :

Table 1 : Age wise distribution of patients

Age(Years)	Number of patients	Percentage
< 65	34	48.6
65 - 69	18	25.7
70 - 74	6	8.6
75 - 79	5	7.1
80+	7	10.0
Total	70	100.0

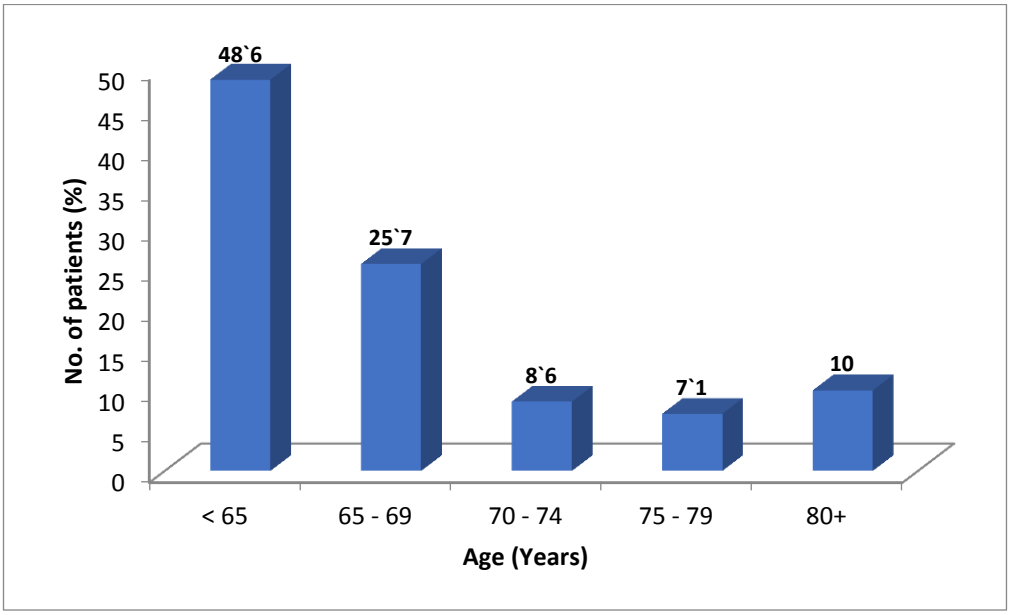


Figure 1 : Age wise distribution of patients

Table 1 and figure 1 demonstrate the age distribution of the patients studied with TB. Maximum patients belonged to the age band of 60 to 65 years constituting to 34 patients. The least number Of patients belonged to the age band of 75 to 79 years and only 7 patients who were above 80 years were present in the study.

Table 2 : gender wise distribution of patients

Gender	Number of patients	Percentage
Female	14	20.0
Male	56	80.0
Total	70	100.0

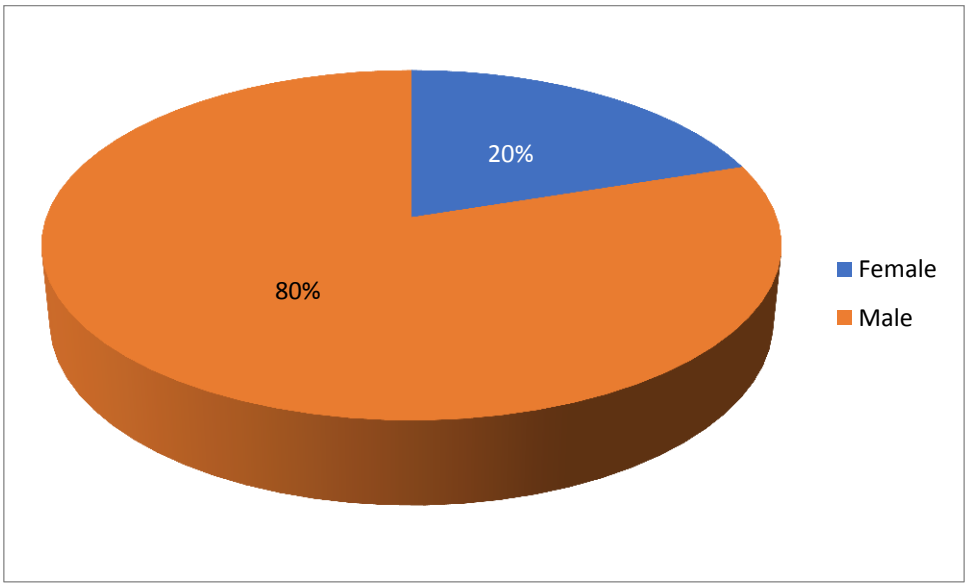


Figure 2 : gender wise distribution of patients

Table 2 and figure 2 depict the gender wise distribution of patients with about 56 patients being male and 14 patients being female.

Table 3 : Occupation wise distribution of patients

Occupation	Number of patients	Percentage
SERVICE	6	8.6
BUSINESS	3	1.4
FARMER	41	58.6
HOUSEWIFE	12	17.1
PANCHAYAT HEAD	8	4.3
Total	70	100.0

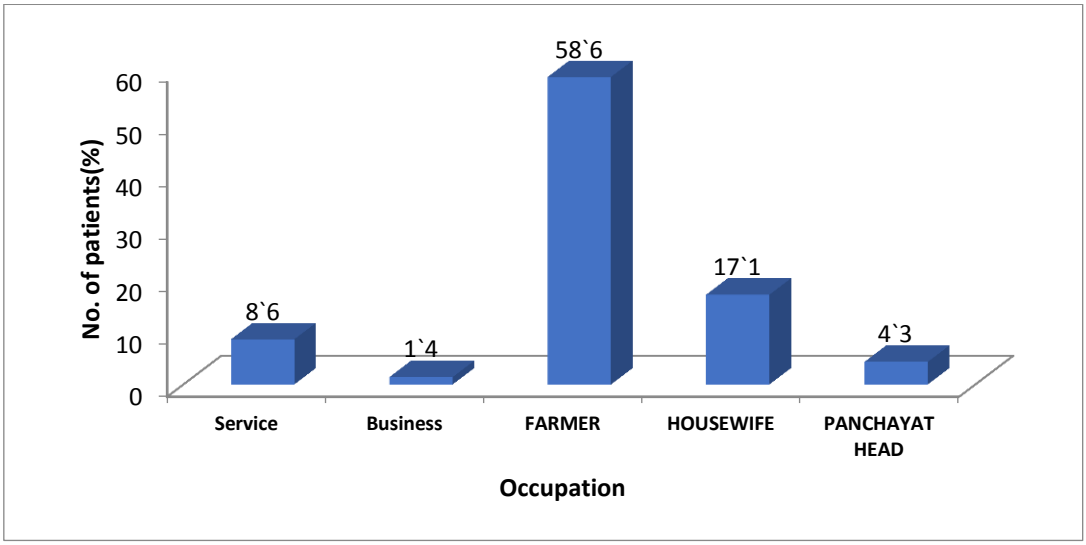


Figure 3 : Occupation wise distribution of patients

Table 3 and figure 3 depict the occupation of the patients with maximum of the patients being farmers accounting for 41 patients as the study was done in a rural set up. This fact was further supported by the fact that the least in the occupation were the business group consisting of only 3 patients.

Table 4 : Distribution of patients according to duration of symptoms

Duration	Number of patients	Percentage
NEW	48	68.6
1M	4	5.7
2M	2	2.9
3M	8	11.4
4M	6	8.6
6M	1	1.4
10M	1	1.4
Total	70	100.0

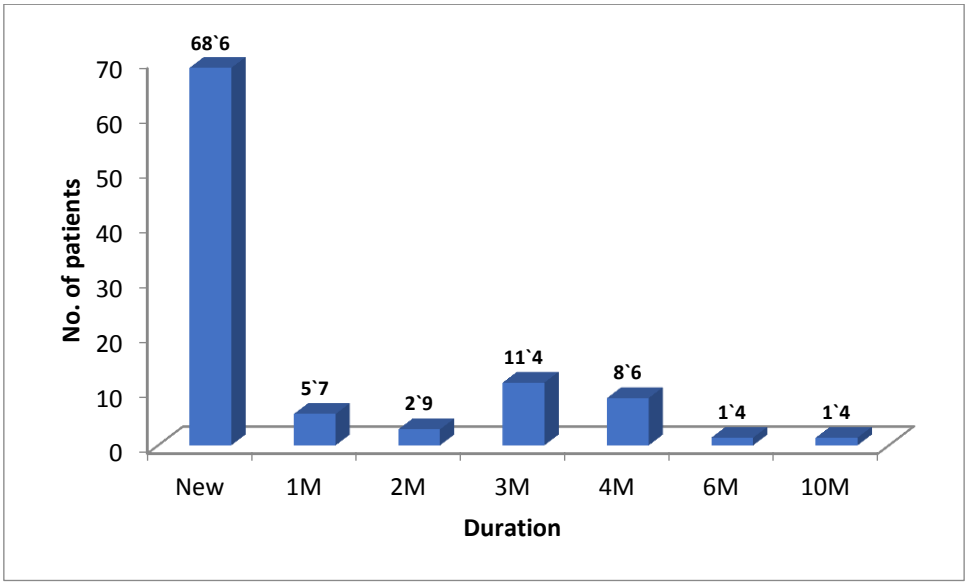


Figure 4 : Distribution of patients according to duration of symptoms

Table 4 and figure 4 depict the time duration of the patients since the onset when they were being studied. 48 patients were diagnosed with TB at the time of being studied while the least patients belonged to 6 and 10 months each contributing 1 patient.

Table 5 : Distribution of patients according to duration of treatment taken

Duration of treatment	Number of patients	Percentage
≤1 Month	42	61.43
2 Months	1	1.4
3 Months	4	5.7
4 Months	4	5.7
6 Months	1	1.4
NIL	17	24.3
Total	70	100.0

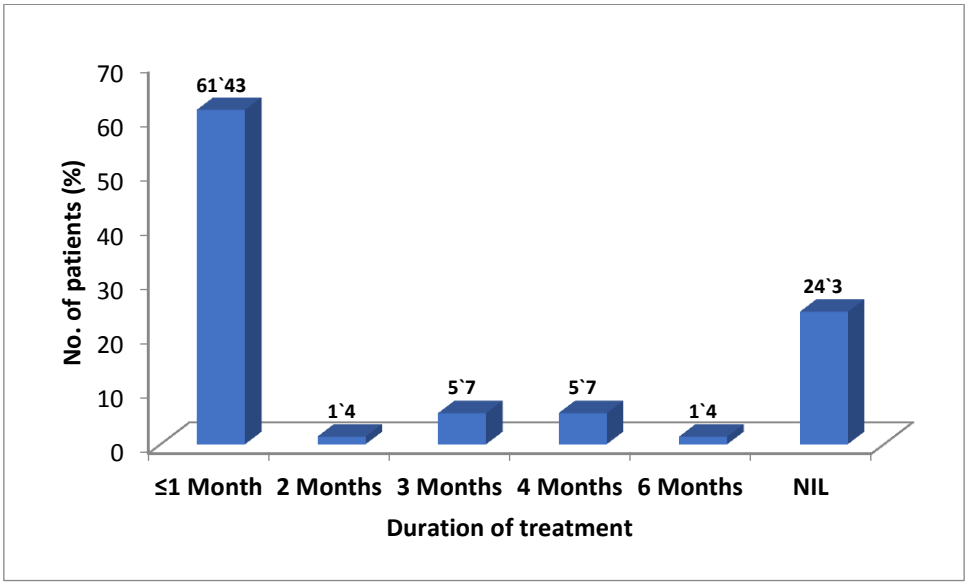


Figure 5 : Distribution of patients according to duration of treatment taken

Table 5 and figure 5 depict the distribution according to the duration of treatment that the patients have taken. Since most of the patients were diagnosed at the time of presentation, the majority of the patients had taken treatment for less than 1 month or were still to initiate therapy. 42 patients had taken treatment for less than 1 month and 17 of them had to still start therapy.

Table 6 : Distribution of patients based on default and relapse of cases

Default/ Relapse	Number of patients	Percentage
DEFAULT	7	10.0
NIL	58	82.9
RELAPSE	5	7.1
Total	70	100.0

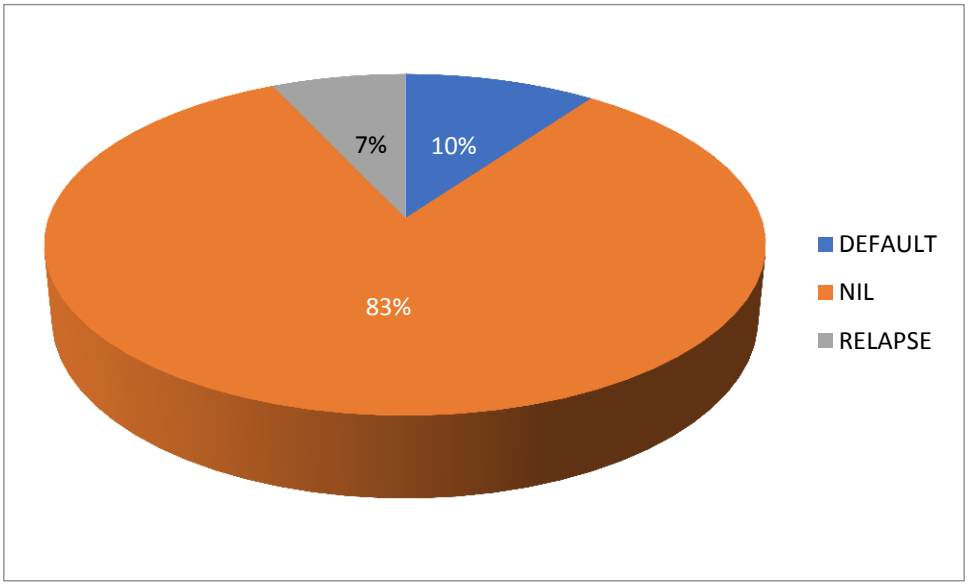


Figure 6 : Distribution of patients based on default and relapse of cases

Table 6 and figure 6 depicts the patients on the basis of default or relapse. Majority of the patients, 58 of them did not have either relapse or default. 7 patients were defaulters who did not complete the treatment and 5 of them were relapses who had a repeat infection after completion of the course of therapy.

Table 7 : distribution of patients according to past history

Past History	Number of patients	Percentage
NIL	38	54.3
# FEMUR	1	1.4
CANCER	1	1.4
CHRONIC OBSTRUCTIVE PULMONARY DISEASE	6	8.6
DIABETES MELLITUS	4	5.7
INTERSTITIAL LUNG DISEASE	1	1.4
LOWER RESPIRATORY TRACT INFECTION	1	1.4
RHEUMATIC HEART DISEASE	1	1.4
HTN	9	12.9
COPD/DM	1	1.4
HTN/DM	4	5.7
HTN/IHD	1	1.4
IHD,DM	1	1.4
IHD/HTN	1	1.4
Total	70	100.0

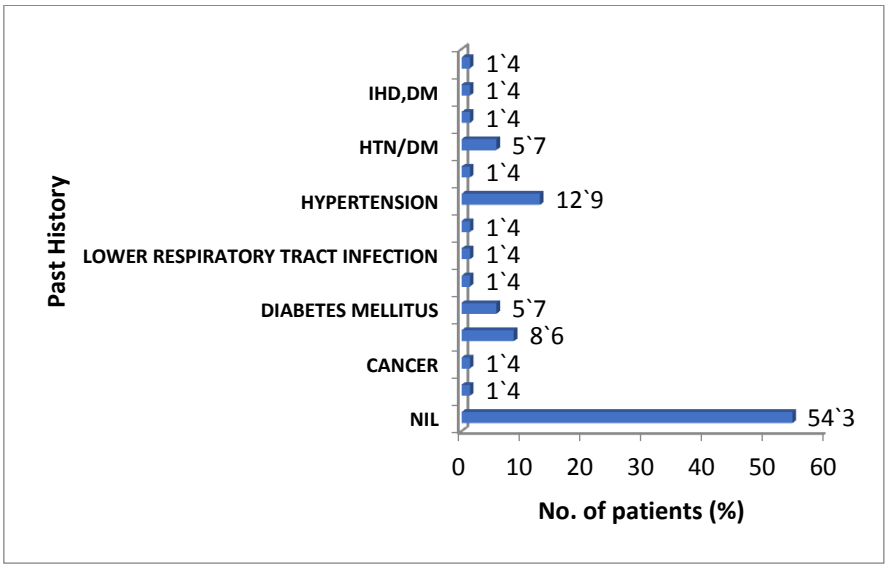


Figure 7 : distribution of patients according to past history

Table 7 and figure 7 depict the various comorbidities that the patients presented with during the study. Most of the patients, 38 of them had no definitive common comorbidities, while the others had either single or multiple illnesses most common being hypertension followed by chronic obstructive pulmonary disease and diabetes.

Table 8: distribution of patients based of family history

Family History	Number of patients	Percentage
NIL	56	80.0
TB IN FATHER	4	5.7
TB IN HUSBAND	2	2.9
TB IN SON, WIFE	1	1.4
TB IN WIFE	5	7.1
TB IN WIFE,MOTHER	1	1.4
TB IN WIFE,SON	1	1.4
Total	70	100.0

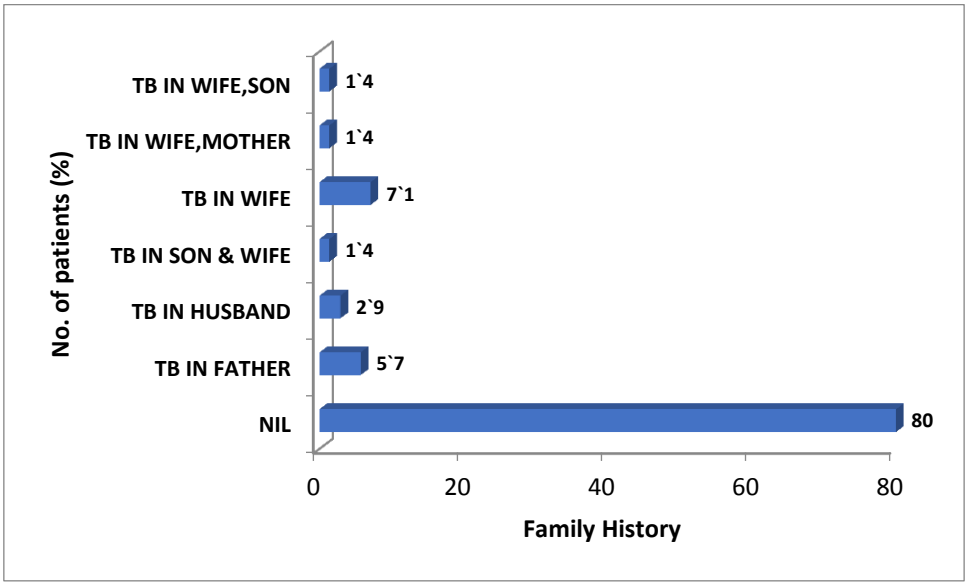


Figure 8 : distribution of patients based of family history

Table 8 and figure 8 depict the presence of TB in the first degree relatives of the patients being studied. Most of the patients, 56 of them had no significant history of TB in any of the first degree relatives. Among the ones with a positive family history, most common was in the wife, followed by husband and father.

Table 9: distribution of patients according to presenting symptoms

TYPICAL SYMPTOMS	Number of patients	Percentage
Yes	47	67.1
No	23	32.9
Total	70	100.0

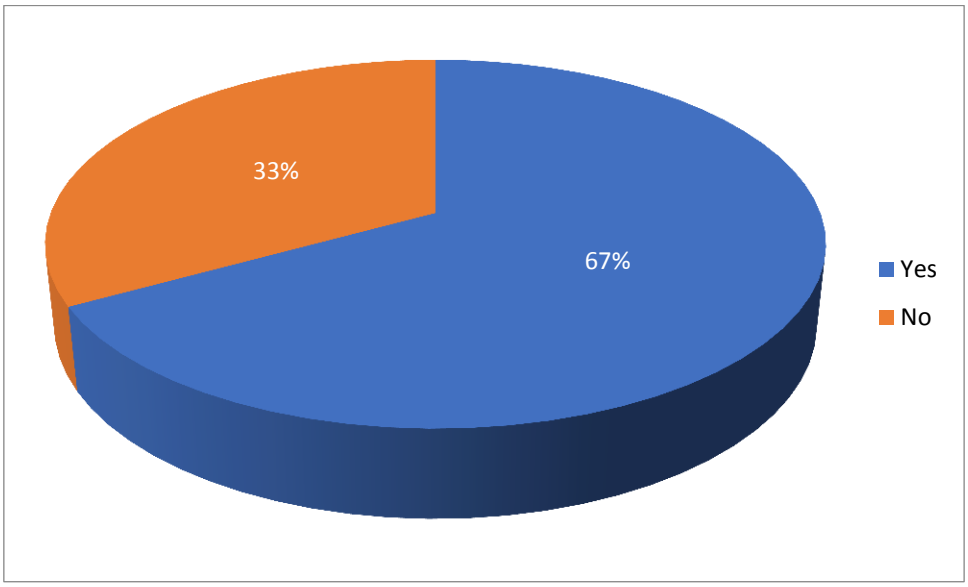


Figure 9 : distribution of patients according to presenting symptoms

Table 9 and figure 9 depict the presentation of the patients based on symptoms. Although 47 patients did present with either one of the typical symptoms of TB, a large number of 23 did not present with any of the typical symptoms and depict the need for higher degree of suspicion for TB in the elderly.

Table 10 : distribution of patients on the basis of diet

Diet	Number of patients	Percentage
MIXED	49	70.0
VEGETARIAN	21	30.0
Total	70	100.0

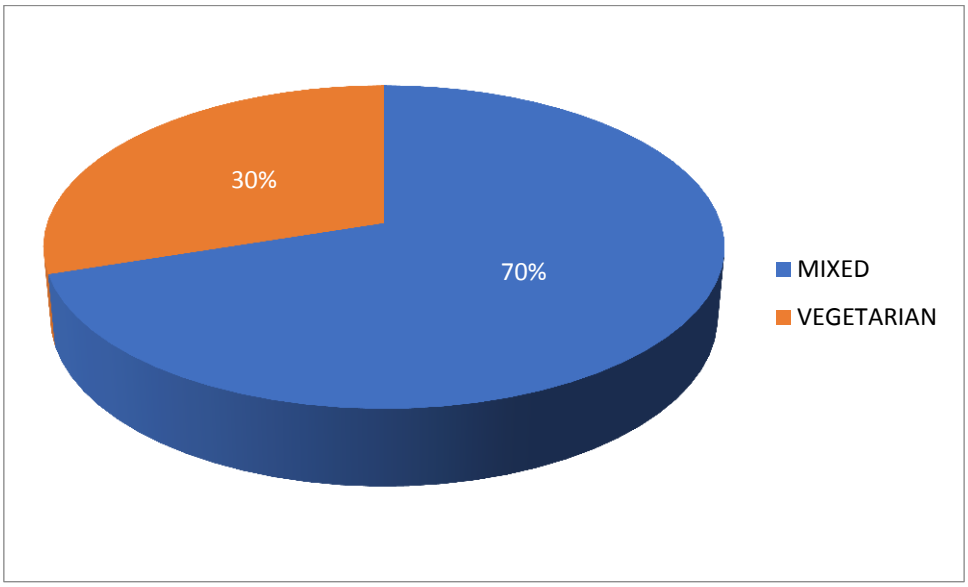


Figure 10 : distribution of patients on the basis of diet

Table 10 and figure 10 demonstrate the diet of the patients. While 49 patients had a mixed diet, 21 patients were vegetarian.

Table 11: distribution of patients on the basis of appetite

Appetite	Number of patients	Percentage
LOSS OF APPETITE	62	88.6
NORMAL	8	11.4
Total	70	100.0

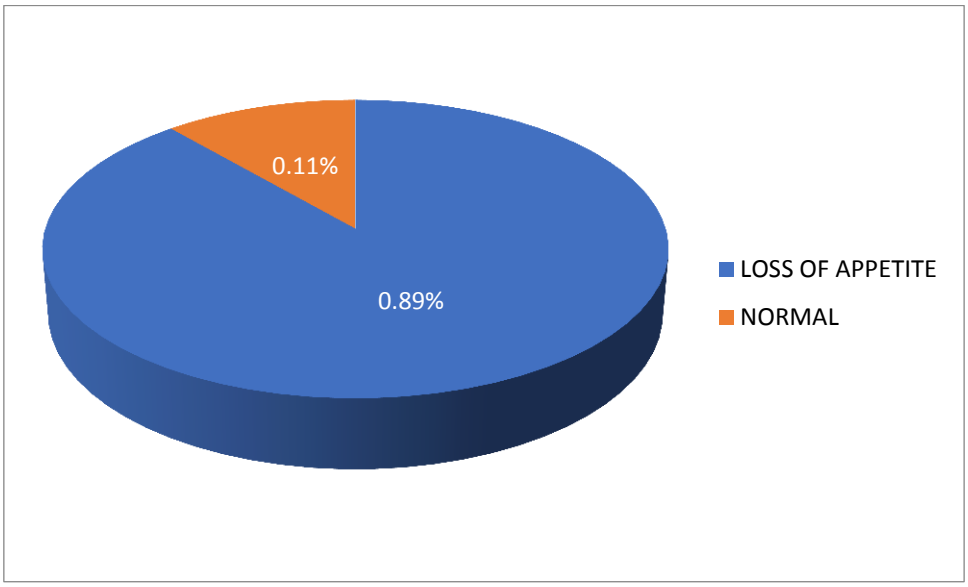


Figure 11 : distribution of patients on the basis of appetite

Table 11 and figure 11 depict that majority of 62 patients had loss of appetite, although few of them did not report it as a primary symptom but mentioned it on being questioned as a part of the study. The remaining 8 patients had a normal appetite.

Table 12: distribution of patients based on bowel and bladder habits

BOWEL AND BLADDER	Number of patients	Percentage
ABNORMAL	5	7.1
INCREASED	1	1.4
NORMAL	63	90.0
REDUCED	1	1.4
Total	70	100.0

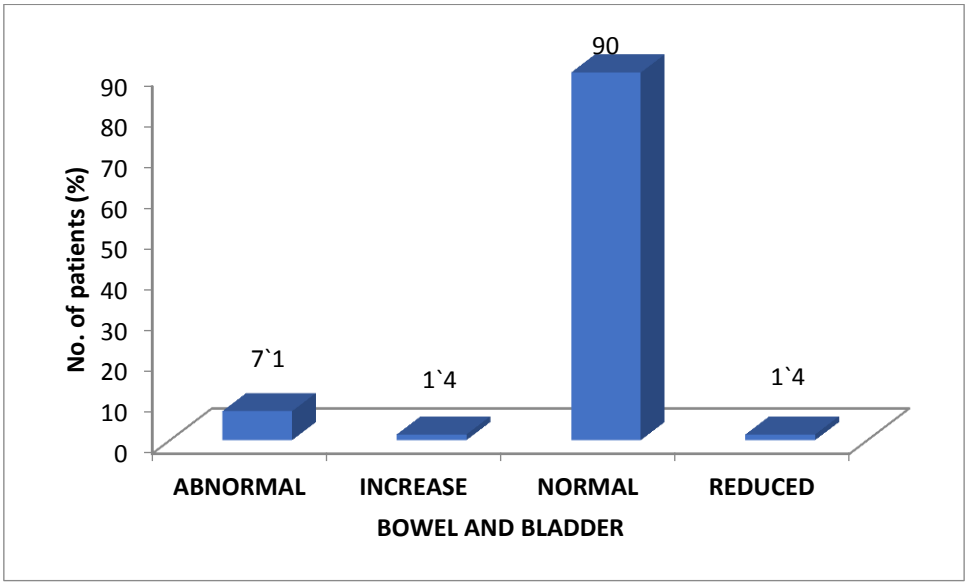


Figure 12 : distribution of patients based on bowel and bladder habits

Table 12 and figure 12 depict that while most of the patients, 63 of them had regular bowel and bladder habits, a small set of people had abnormalities like hematuria and passage of orange colour urine was noticed in those who were initiated on treatment as a side effect of treatment with rifampicin.

Table 13 : distribution of patients based on ill habits

Habits	Number of patients	Percentage
Alcohol	2	2.9
Smoking	23	32.9
Tobacco	1	1.4
Smoking & Alcohol	18	25.7
NIL	26	37.1
Total	70	100.0

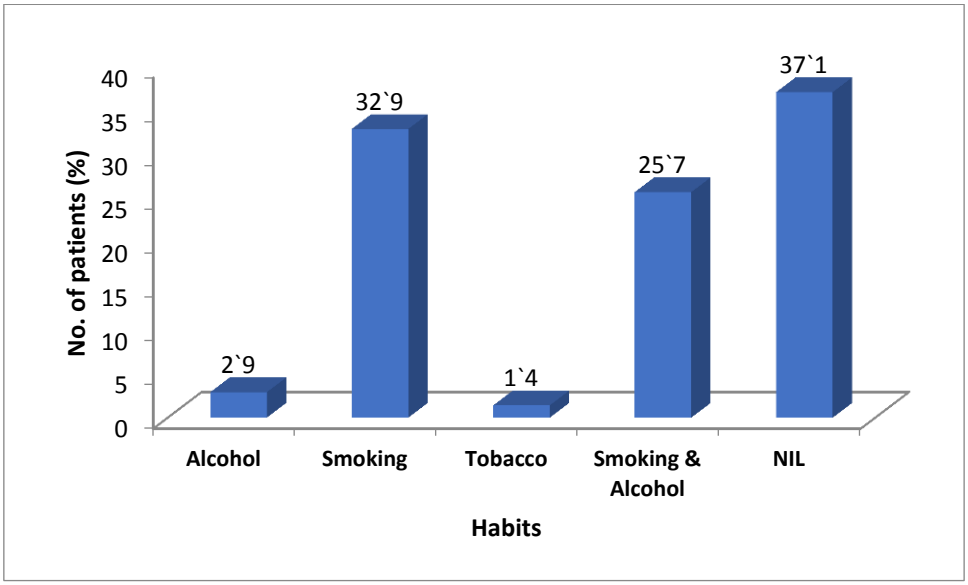


Figure 13 : distribution of patients based on ill habits

Table 13 and figure 13 depict that while 26 patients did not have any ill habits and a large number of patients had risk factors like smoking and alcohol consumption which predispose to the development of TB.

Table 14 : distribution of patients according to grams stain pattern

GRAMS	No. of patients	Percentage
FUNGI	3	4.3
NOT DONE	5	7.1
NEGATIVE CELLS	20	28.6
POSITIVE CELLS	17	24.3
PUS CELLS	10	14.3
NIL	15	21.4
Total	70	100.0

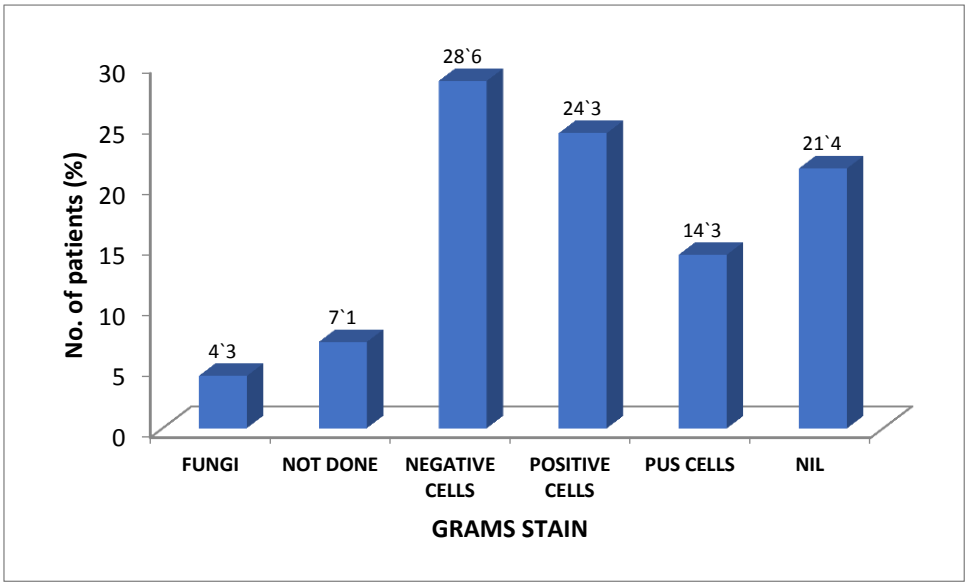


Figure 14 : distribution of patients according to grams stain pattern

Table 14 and figure 14 depict the grams stain pattern in all the subjects with gram positive and gram negative cells being almost equal, followed by pus cells and fungi. A majority of the grams stain did not reveal the presence of any organisms.

Table 15 : distribution of patients according to the Zeehl – Neelson stain

ZN STAIN	Number of patients	Percentage
1+	25	35.7
2+	27	38.6
3+	28	40.0
4+	1	1.4
SCANTY	4	5.7
Total	70	100.0

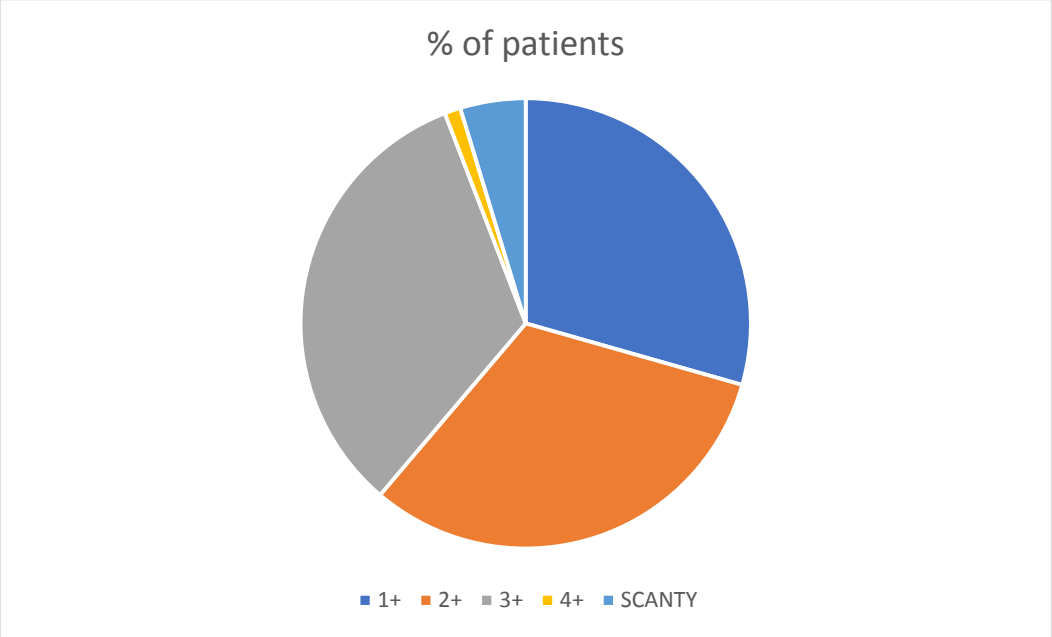


Figure 15 : distribution of patients according to the Zeehl – Neelson stain

Table 15 and figure 15 show the ZN stain pattern in subjects, associating the strength of the positivity with the bacterial load. An almost equal number was seen in 1+, 2+ and 3+ grading, with few patients having scanty bacilli and only one patient with 4+.

Table 16: distribution of patients based on culture sensitivity

CULTURE SENSITIVITY	Number of patients	Percentage
E.COLI	1	1.4
KLEB	2	2.9
NORMAL	52	74.3
NOT DONE	11	15.7
PNEUMOCOCCI	1	1.4
STREPTOCOCCI	3	4.3
Total	70	100.0

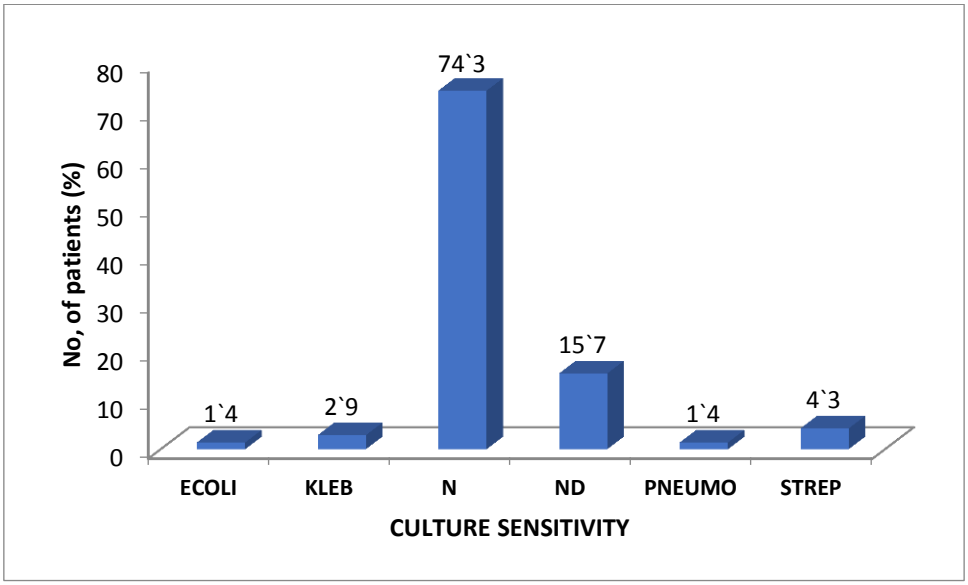


Figure 16 : distribution of patients based on culture sensitivity

Table 16 and figure 16 depict the culture sensitivity patterns of the sputum samples in the patients. While the majority of the samples did not reveal the growth of any organism, streptococci was isolated in 3 samples followed by klebsiella in 2 samples and Escherichia coli, pneumococci in 1 sample each.

Table 17: distribution of patients according to common radiographical features

COMMON RADIOGRAPHICAL FEATURES	Number of patients	Percentage
CALCIFICATION	8	11.4
CAVITATION	8	11.4
COLLAPSE	1	1.4
FIBROSIS	10	14.3
HYDROPNEUMOTHORAX	1	1.4
MILIARY MOTTLING	15	21.4
PLEURAL EFFUSION	14	20.0
PNEUMONIA	12	17.1
SYNPNEUMONIC EFFUSION	1	1.4
Total	70	100.0

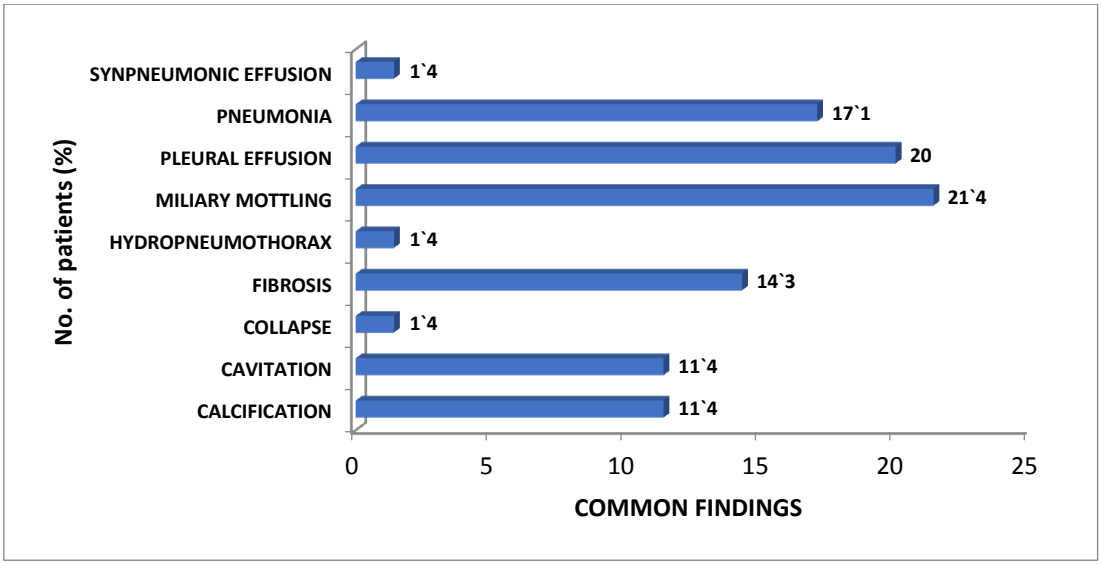


Figure 17 : distribution of patients according to common radiographical features

Table 17 and figure 17 depict the common roentgenographic features seen in all the patients. 15 patients had military mottling, 14 had pleural effusions, 12 had pneumonia, 10 had fibrosis, calcification and cavitation in 8 patients each, hydropneumothorax, collapse and synpneumonic effusion in 1 patient each.

Table 18 : distribution of patients based on CT/MRI findings

CT/MRI	No. of patients	Percentage
PULMONARY EMBOLISM	1	1.4
FIBROSIS	1	1.4
KOCHS	4	5.7
NOT DONE	64	91.4
Total	70	100.0

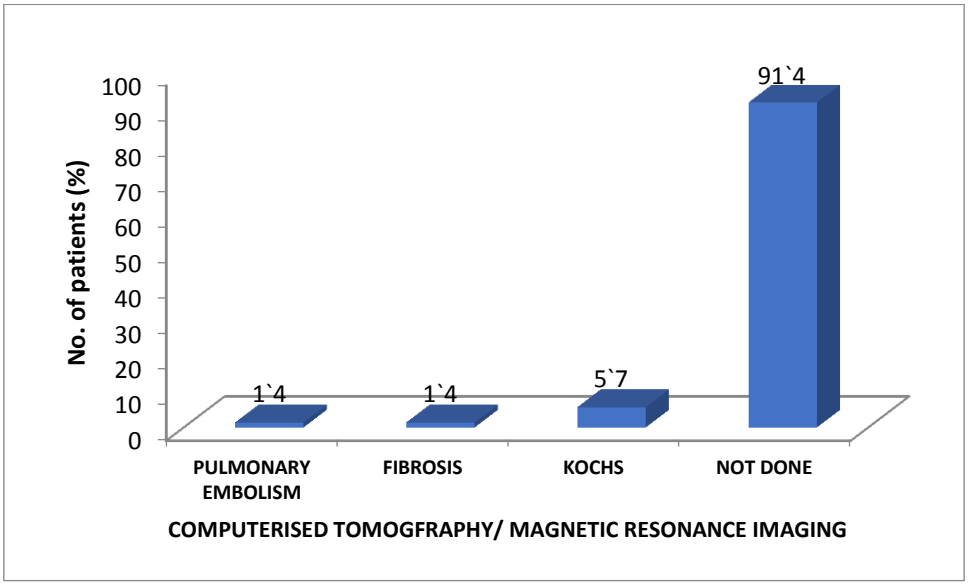


Figure 18 : distribution of patients based on CT/MRI findings

Table 18 and figure 18 show the CT/MRI findings in the patients with majority of the patients nor having it done due to financial constraints. 4 cases were reported as tubercular etiology, and one each of fibrotic changes and pulmonary embolism.

Table 19: distribution of patients according to liver function tests

LIVER FUNCTION TEST	Number of patients	Percentage
ABNORMAL	13	18.6
NORMAL	51	72.9
NOT DONE	6	8.6
Total	70	100.0

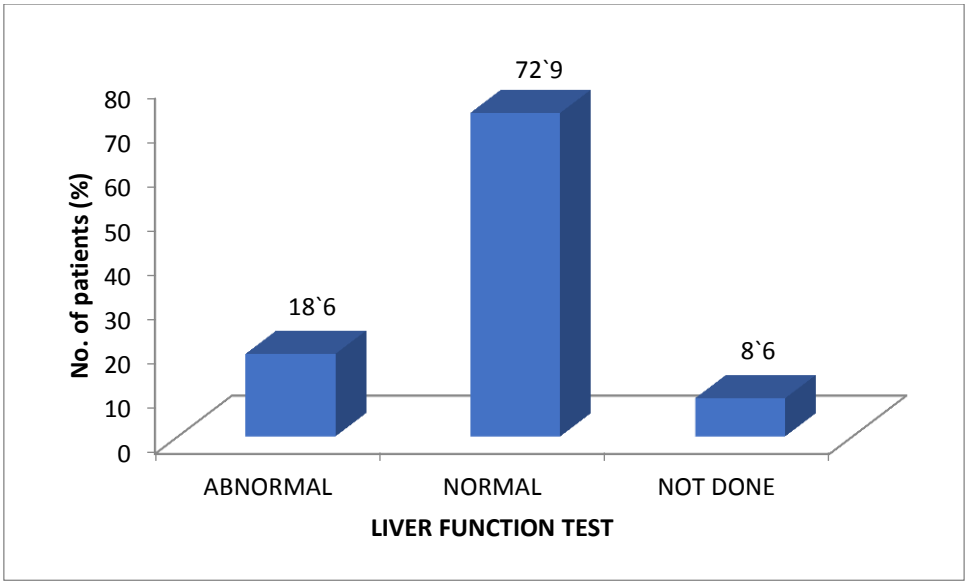


Figure 19 : distribution of patients according to liver function tests

Table 19 and figure 19 depict the baseline liver function analysis of all the patients. The tests are done to ensure proper functioning of the liver as the drugs used for TB are hepatotoxic. Most of the patients, 51 had normal results while 13 0f them had abnormal results.

Table 20 : distribution of patients based on HIV status

HIV STATUS	Number of patients	Percentage
POSITIVE	9	12.9
NEGATIVE	50	71.4
NOT DONE	11	15.7
Total	70	100.0

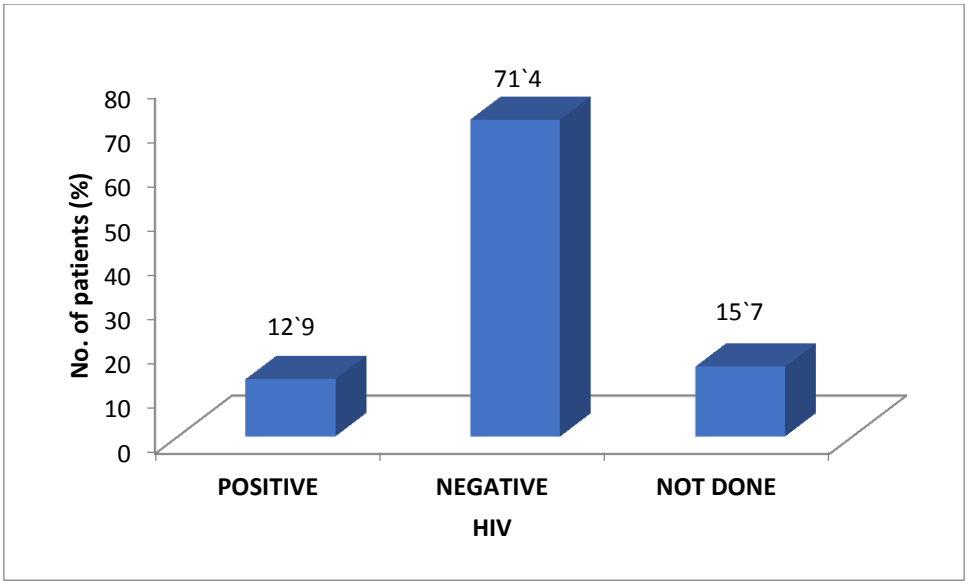


Figure 20 : distribution of patients based on HIV status

Table 20 and figure 20 depict the HIV status of the patients. While 50 patients were negative, 9 patients were positive among the 70 subjects studied. 11 patients were not tested for HIV.

Table 21: distribution of patients based on treatment being taken

TREATMENT	Number of patients	Percentage
TAB AKURIT 4 2-0-0	14	20.0
TAB AKURIT 4 3-0-0	37	52.9
TAB FDC 2-0-0	1	1.4
TAB FDC 3-0-0	6	8.6
TAB FDC 3-0-0 + INJ STM	1	1.4
TAB FORECOX 2-0-0	8	11.4
TAB RCINEX, COMBUTAL	1	1.4
NONE	2	2.9
Total	70	100.0

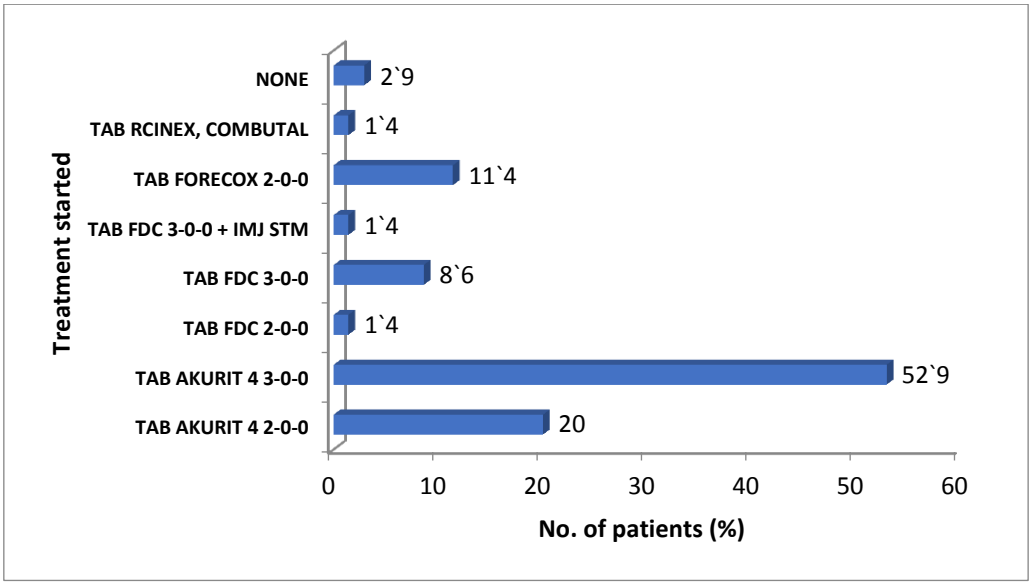


Figure 21 : distribution of patients based on treatment being taken

Table 21 and figure 21 depict the treatment the patients have been taking or have been initiated on. Majority of the patients were taking combination of all the four first line drugs based on the weight of the patient. 2 of the patients were yet to be started on therapy.

Table 22 : distribution of the patients according to the criteria for diagnosis

DIAGNOSIS BASED ON	Number of patients	Percentage
Typical symptoms, & Chest X-ray	40	57.1
Atypical symptoms & Chest X-ray	30	42.9
Total	70	100.0

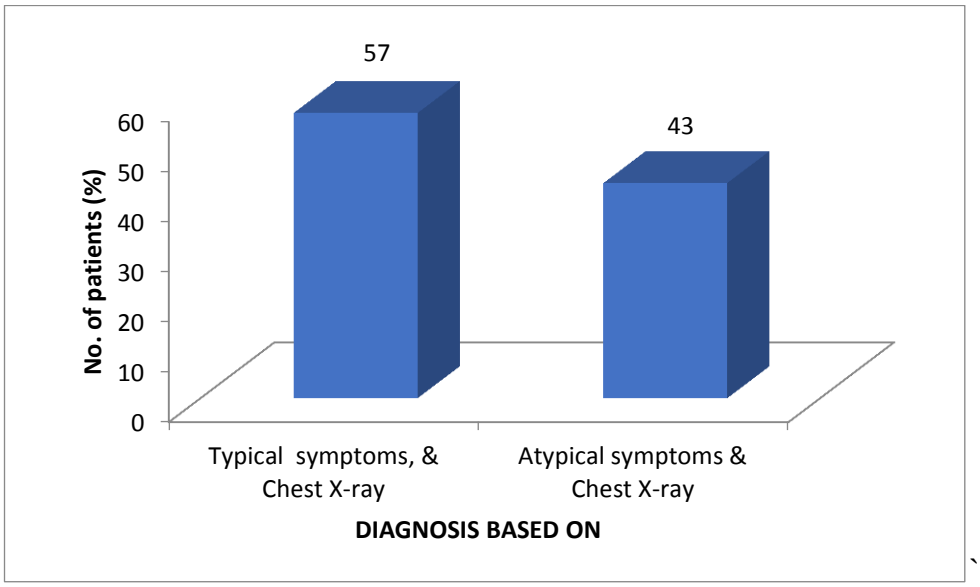


Figure 22 : distribution of the patients according to the criteria for diagnosis

Table 22 and figure 22 depict the basis for suspicion and diagnosis of the cases of TB in the subjects. While 40 patients were designated to have typical symptoms and chest X-ray features, 30 patients did not fall under the typical category and presented with atypical symptoms and chest X-ray features.

DISCUSSION

This study is a hospital based cross sectional descriptive study conducted in Shri B M Patil Medical College, Vijayapura. The study was conducted with a sample size of 70 patients who attended the outpatient department and also those who were admitted in the hospital during the study period. The study is being compared with other studies done with variable parameters and the observations are as follows :

1. Studies have been done on the geriatric population, with the mean age above 60 years in India with relation to the pattern of distribution of TB among males and females :

A study done by Sandhyarani Mohrana et al included 56 patients under the geriatric category with a male predominance of 40 and female of 16[60]. A study done by Dheeraj Gupta et al included 50 patients aged above 60 years also having a male predominance of 32 while female cases were 18 in number[61]. A study conducted by Mohammad Towhidi et al included 40 patients of the geriatric age group with 21 males and 19 females[62]. A study done by Jae Ho Lee et al studied geriatric TB in 119 individuals with 61 males and 58 females[63]. A study done by Khwaja Ubedulla Shaikh et al studied TB in 50 geriatric patients which included 36 males and 14 females. 32 patients among the geriatric group were aged between 60-65 years, 13 were between 65-70 years and 5 patients were aged above 70 years[2]. A study conducted by Jagdeesh Rawat et al studied 50 geriatric patients with 37 of them being male and 17 female[64]. Our study was conducted in 70 geriatric patients demonstrating a male predominance of 56 patients while female patients were 14 in number. Our study included majority of the patients in the age group of 60 - 65 years accounting for 48.6% followed by 65 - 69 years accounting for 18%, 8.6% in age group of 70 – 74, 7.1% in age group of 75 – 79, 10% above the age of 80 years.

2. A study done by Mohammad Towhidi et al reported that the average duration of symptoms present in the patients at the time of presentation was 103 days, varying between 3 to 4 months[62]. In our study most of the patients(68.6%) presented with complaints less than one month and were diagnosed at presentation. 11.4% patients presented with symptoms since 3 months and 8.6% had symptoms since 4 months.
3. Various studies depict the pattern of presentation of geriatric TB and the comparison with our study can be seen as follows : the symptoms of cough, dyspnoea, hemoptysis and chest pain are considered as respiratory symptoms of TB for the purpose of statistical analysis, while the symptoms of fever, night sweats and loss of weight or appetite are considered as constitutional symptoms even if they might be the only presenting symptoms sometimes.

STUDY	Sandhyarani Mohrana et al[60]	Jae Ho Lee et al[63]	Khwaja Ubedulla Shaikh et al[2]	Jagdeesh Rawat et al[64]
RESPIRATORY SYMPTOMS	Cough – 44 Dyspnoea – 33 Hemoptysis -19 Chest pain - 25	Cough – 80(67.2%) Dyspnoea – 46(38.7%) Hemoptysis – 17(14.3%) Chest pain – 5(4.2%)	Cough – 30 Dypnoea – 27 Hemoptysis – 13 Chest pain – 19	Cough – 42(84%) Hemoptysis – 3(6%) Dyspnea – 15(30%) Chest pain – 7(14%)
CONSTITUTIONAL SYMPTOMS	Fever – 35 Night sweats- 40 Weight loss -	Fever – 39(32.8%) Night sweats – 4(3.4%)	Fever – 29 Night sweats – 6 Weight loss	Fever – 38(76%) Night sweats –

	33	Weight loss – 43(36.1%)	- 27	9(18%) Weight loss : 43(86%)

STUDY	Mohammad Towhidi et al[62]	Dheeraj Gupta et al[61]	Our study
RESPIRATORY SYMPTOMS	Cough – 37(92.5%) Hemoptysis – 6(15%)	Cough – 37(74%) Hemoptysis – 7(14%) Dyspnoea – 23(46%) Chest Pain – 19(38%)	Cough – 47(67.1%) Hemoptysis – 3(4.2%) Dyspnoea – 25(35.7%) Chest pain – 16(22.8%)
CONSTITUTIONAL SYMPTOMS	Fever – 23(57.5%) Night sweats – 9(22.%%) Weight loss – 32(80%)	Fever – 37(74%) Night sweats – 25(50%) Weight loss – 40(80%)	Fever – 33(47.1%) Night sweats – 20(28.5%) Weight loss – 62(88.6)

4. Studies can be compared on the basis of the roentgenographic features seen in the geriatric TB patients. Although more than one finding can be present in one patient, they are divided for the purpose of statistical analysis as separate lesions. The observations of such comparison are :

STUDY	Dheeraj Gupta et al[61]	Mohammad Towhidi et al[62]	Sandhyarani Mohrana et al[60]	Our study
RADIOLOGICAL FEATURES	Normal – 3(6%) Infiltrates – 13(26%) Consolidation – 13(26%) Hilar lymphadenopathy – 3(6%) pleural effusion – 9(18%) miliary mottling – 3(6%) cavitation – 16(32%) fibrosis – (26%)	Upper and lower lobe infiltration – 20(50%) cavitation – 10(25%) miliary mottling – 1(2.5%)	Upper, middle and lower lobe infiltrates – 67 cavitation – 9 Miliary mottling – 7	middle and lower lobe infiltrates – 12(17.1%) Calcification – 8(11.4%) Cavitation - 8(11.4%) Collapse – 1(1.4%) Fibrosis – 10 (14.3%) hydropneumothorax – 1(1.4%) Miliary mottling – 15(21.4%) Pleural effusion – 14(20%) Synpneumonic effusion – 1(1.4%)

A study conducted by Jae Ho Lee et al demonstrated that the radiological features being definitive for TB in the geriatric population was 10.9%[63]. A study conducted by Khwaja Ubedulla Shaikh et al showed that the upper lobe infiltrates were more common than the middle and lower lobe in the geriatric study group[2]. A study done by Jagdeesh Rawat et al reported that bilateral lesions were more common than unilateral lesions and upper zone more common than the middle and lower zone lesions[64]. Our study displayed that all the lesions that were detected in terms of infiltration were in the middle and lower lobe either individually or in combination.

None of the lesions were noted in the upper lobe, which is consistent with the other studies showing more predilection for middle and lower lobes of the lung than the upper lobes in the geriatric patients. This observation is in contrast to the young patients who display most of the lesions in the upper lobe of the lung. Another feature on the roentgenogram consistent with the geriatric population is the military mottling seen in 21.4% of the geriatric subjects in our study.

5. Studies were also compared on the basis of the number of bacilli seen on the ZN stain as follows :

STUDY	Sandhyarani Mohrana et al[60]	Khwaja Ubedulla Shaikh et al[2]	Our study
ZN STAIN	Scanty – 6 3+ - 11 2+ - 8 1+ - 10	Scanty – 2 3+ - 13 2+ - 7 1+ - 7	Scanty – 4(5.7%) 3+ - 28(40%) 2+ - 27(38.6%) 1+ - 25(35.7%)

In a study conducted by Jagdeesh Rawat et al, out of 50 geriatric patients, 45 tested sputum positive and 3 were AFB positive for broncho-alveolar lavage fluid[64]. A study conducted by Jae Ho Lee et al, 70.6% of the samples tested were positive for AFB in sputum samples in geriatric age group and 7 patients were AFB positive from broncho-alveolar lavage fluid[63].

6. Studies have been compared on the basis of the various co-morbidities that the elderly have along with TB which makes them prone to develop and progress to complications of TB, and also mask the symptoms of TB thereby making it difficult to suspect and diagnose the disease at an early stage.

STUDY	Jagdeesh Rawat et al[64]	Jae Ho Lee et al[63]	Sandhyarani Mohrana et al[60]	Mohammad Towhidi et al[62]
CO-MORBIDITIES	Diabetes mellitus – 5(10%) Hypertension – 11(22%) COPD – 13(26%) Malignancy – 4(8%)	Diabetes mellitus – 30(25.2%) Hypertension – 18(15.1%) COPD – 12(10.1%) Malignancy – 1(0.8%)	Diabetes mellitus – 14% Hypertension – 24% COPD – 18%	Diabetes mellitus – 3(7.5%) Hypertension – 2(5%) COPD – 3(7.5%) Malignancy - 3(7.5%)

Dheeraj Gupta et al[61]	Khwaja Ubedulla Shaikh et al[2]	Our study
Diabetes mellitus – 8(16%) Hypertension – 2(4%) COPD – 1(2%)	Diabetes mellitus – 14% Hypertension – 24% COPD – 18%	Diabetes mellitus – 10(14.2%) Hypertension – 15(21.4%) COPD – 6(8.5%) Malignancy – 2(2.8%)

7. HIV and TB occur as co-infections to each other and multiple studies have proved the same. The clinical features in both these devastating diseases are almost the same and hence are almost indistinguishable clinically in the geriatric population. Atypical presentation with constitutional symptoms are seen commonly in this subset of the geriatric population. A study by O J Daniel et al studied 353 sputum smear positive TB patients and 58(16.4%) were positive for HIV[65]. Our study witnessed that among

the 70 geriatric subjects studied, 9(12.9%) were positive for HIV. Hence, as described by many clinicians, it is rather difficult to identify the separate entities of TB and HIV among the elderly and high level of suspicion should be kept while dealing with such patients .

8. A study conducted by Sandhyarani Mohrana et al demonstrated that among the geriatric patients studied for TB, 38(67.8%) were treated and disease free after completing the course of therapy, 18(32.1%) were not cured and remained positive even after completing the complete course of therapy, 14(25%) failed in completing the therapy and 8(14.2%) expired during the study[60]. In another study by Jae Ho Lee et al, out of the 81 geriatric subjects studied, 71(87.7%) completed the treatment course and 10(12.3%) expired during the study[63]. In our study, 7 patients were defaulters who did not complete the full course of therapy and 5 were relapses who had come positive again, after completing the course of treatment. In our study, 8 patients died during their stay in the hospital while being studied, summing up to 11.4% mortality among the study subjects. In our study, the most common cause of death was type I respiratory failure owing to the lung lesions. 2 patients among the 8 who died were positive for HIV, indicating that the prognosis of the concurrent infection of TB and HIV is not good in terms of prognosis.

SUMMARY

Our study was conducted in 70 geriatric patients who tested sputum positive for pulmonary TB and all the observations from the study can be summarised as follows:

Most of the patients were between the age group of 60-65 years and most of the study sample were male patients, thereby the most common occupation seen among the subjects was agriculture related as the study was conducted in a rural area.

Among the subjects studied, most of them were diagnosed with TB at the time of being studied and very few were already diagnosed, therefore most of the patients were under the treatment period of less than one month as they were initiated after being tested sputum positive in the study. It was also observed that among the patients being studied, a very small proportion of the sample were patients of relapse and default.

The elderly are prone to develop TB in the setting of already existing co-morbid conditions. In our study, the leading comorbidities were hypertension, type II diabetes mellitus, COPD, and malignancy.

TB has been known to occur in close contacts without adequate protection. Hence the presence of the disease among the family members of the study subjects was studied and it was seen that the presence of TB among the family members was seen in 20% of the cases.

The disease in the geriatric population does not present with typical symptoms and vague constitutional symptoms are the presenting features. This is also due to the presence of other co-morbid conditions that mask the symptoms of TB. Consistent with this, our study also depicted that 67% of the cases presented with atypical symptoms, the most striking of which was loss of appetite seen in 88% of the cases.

The AFB analysis using the ZN staining technique still remains the best method for detection of pulmonary TB. In our study, an almost equal number of samples were 1+ , 2+ and 3+.

The radiographical features in the elderly patients with TB differ from the younger population. All the cases which had infiltration involved the middle and lower lobes. Miliary mottling was a common feature in the elderly.

HIV is a very common co-infection existing in patients with TB. 12% of the geriatric patients studied were positive for both TB and HIV. Also 2 of the mortalities among the study subjects were among the subset that were HIV positive, indicating a poor prognosis in such a condition.

The mortality rate in the study was 11.4% indicating the necessity for stringent methods to detect TB earlier and start the treatment earlier in the geriatric population.

CONCLUSION

Our study showed that the various symptoms that the geriatric population presented with were cough, dyspnoea, chest pain and hemoptysis. While a significant number of the patients presented with atypical presentation, the most common feature was weight loss followed by fever and night sweats.

The radiological features present in these patients were miliary mottling followed by pleural effusion, middle and lower lobe infiltrates, calcification, cavitation, fibrosis, collapse, hydropneumothorax, synpneumonic effusion.

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ANNEXURE -I

ETHICAL CLEARENCE CERTIFICATE

B.L.D.E (Deemed to be University)
SHRI.B.M.PATIL MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE
VIJAYAPUR – 586103 IEC. no-286/18
17/11/2018

INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 13-11-2018 at 03-15 PM scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has accorded Ethical Clearance.

Title : A study of clinical & radiological profile of sputum positive pulmonary tuberculosis among elderly patients.

Name of P.G. Student : Dr Pranay Kumar R.P
Department of General Medicine.

Name of Guide/Co-investigator: Dr. Anand.P.Ambali, Professor of General Medicine.

DR RAGHAVENDRA KULKARNI
CHAIRMAN
Institutional Ethical Committee
BLDEU's Shri B.M. Patil
Medical College,BIJAPUR-586103.

Following documents were placed before E.C. for Scrutinization:

1) Copy of Synopsis/Research Project
2) Copy of informed consent form.
3) Any other relevant documents.

ANNEXURE II

INFORMED CONSENT FORM

TITLE OF RESEARCH: **A STUDY OF CLINICAL AND RADIOLOGICAL PROFILE OF SPUTUM POSITIVE TUBERCULOSIS AMONG ELDERLY PATIENTS**

GUIDE : **DR ANAND P. AMBALI**
M.D. MEDICINE

P.G. STUDENT : **DR PRANAY KUMAR R. P.**

All aspects of this consent form are explained to the patient in the language understood by him or her.

PURPOSE OF STUDY:

I have been informed that the purpose of this study is to determine the various clinical and radiological patterns of presentation in geriatric patients who have tested sputum positive for tuberculosis.

PROCEDURE:

I understand that I will undergo detailed history and clinical examination and investigations.

BENEFITS:

I understand that my participation in this study will have no direct benefit to me other than the potential benefit of treatment which is planned to prevent further morbidity and mortality in me.

CONFIDENTIALITY:

I understand that the medical information produced by the study will become a part of hospital record and will be subjected to confidentiality and privacy regulation of hospital. If the data is used for publication the identity will not be revealed.

REQUEST FOR MORE INFORMATION:

I understand that I may ask for more information about the study at any time.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or withdraw from study at any time.

(Signature of Guardian) (Signature of patient)

STUDY SUBJECT CONSENT FORM:

I confirm that **Dr. PRANAY KUMAR R. P.** has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I have been explained all above in detail in my own language and I understand the same. I agree to give my consent to participate as a subject in this research project.

DATE SIGNATURE OF PARTICIPANT

DATE SIGNATURE OF WITNESS

ANNEXURE III

PROFORMA

SCHEME OF CASE TAKING

Name : IPD/OPD No :	Age : Sex :	Past occupation :	Present occupation :
Address :	Contact no :	Marital status :	Socio economic status :

SYMPTOMS AS GIVEN BY THE PATIENT	SYMPTOMS AS GIVEN BY THE PATIENT ATTENDERS

TUBERCULOSIS DATA :

DATE OF DIAGNOSIS	
DURATION OF TUBERCULOSIS	
DURATION OF TREATMENT TAKEN	
DETAILS OF DEFAULTER/ RELAPSE	

OTHER SIGNIFICANT PAST HISTORY AND COMORBIDITIES :

FAMILY HISTORY :

PERSONAL HISTORY :

DIET	APPETITE	BOWEL AND BLADDER	SLEEP	ALCOHOL / SMOKING

MENSTRUAL HISTORY :

GPE AND SIGNIFICANT HEAD TO TOE EXAMINATION :

HEIGHT :	WEIGHT :	BMI :

VITALS :

PR	BP	RR	TEMPERATURE

HEAD TO TOE EXAMINATION:

SYSTEMIC EXAMINATION :

CVS :

RS : INSPECTION –

PALPATION –

PERCUSSION –

AUSCULTATION –

P/A :

CNS:

INVESTIGATIONS:

1) HAEMATOLOGY – Hemoglobin	gm. %
2) Total WBC counts	Cells/mm^3
3) Differential counts -	
Neutrophils	%
Lymphocytes	%
Eosinophils	%
Monocytes	%
Basophils	%
Platelet count	

ESR	At the end of 1st hour

SPUTUM EXAMINATION:

GRAMS STAIN -

ZN STAIN -

CULTURE SENSITIVITY -

Radiological investigations:

CHEST X RAY :

CT CHEST :

MRI CHEST :

3. BIOCHEMISTRY:

a. Serum electrolytes:

b. Liver function tests :

c. sugars :

4. Others:

a. ECG

b. Echo

c. HIV

FINAL DIAGNOSIS :

TREATMENT INITIATED ON(IF ANY) :

DIAGNOSIS BASED ON : 1. TYPCAL SYMPTOMS

2. ATYPICAL SYMPTOMS

3. CHEST X-RAY

MASTER CHART

NAME	AGE	SEX	TYPICAL SYMPTOMS	ATYPICAL SYMPTOMS	DOD	DURATION	DOTT	DEF/REL	PAST H/O	FAM H/O	APPETITE	HABITS	BMI	GRAMS	ZN STAIN	C/S	COMMON FINDINGS
BASAPPA	62	M	YES		1/10/2020	NEW	NIL	NIL	HTN	TB IN FATHER	LOA	S + A	23`94	NEGATIVE CELLS	2+	N	FIBROSIS
MALLANNA	65	M	YES		2/10/2020	NEW	NIL	NIL	NIL	NIL	LOA	NIL	18`66	POSITIVE CELLS	3+	N	PLEURAL EFFUSION
GURANNA B	75	M		YES	2/1/2020	NEW	NIL	NIL	HTN	TB IN WIFE	LOA	S	16`01	NEGATIVE CELLS	1+	N	CAVITATION
SADASHIVAPPA N K	84	M		YES	12/10/2019	NEW	NIL	NIL	NIL	NIL	LOA	S + A	18`56	PUS CELLS	1+	N	PNEUMONIA
KASANU CHAVAN	80	M		YES	1/1/2020	NEW	NIL	NIL	HTN	NIL	N	S + A	20`57	POSITIVE CELLS	1+	N	PNEUMONIA
KAREPPA A H	75	M	YES		1/24/2020	1M	1M	NIL	COPD	NIL	N	S + A	24`88	POSITIVE CELLS	2+	STREP	PNEUMONIA
MALLAPPA G T	82	M	YES		10/6/2019	4M	4M	NIL	NIL	TB IN WIFE	LOA	S	20`67	POSITIVE CELLS	2+	STREP	CAVITATION
SHANTAMMA M D	78	F		YES	1/12/2020	NEW	NIL	NIL	DM	NIL	LOA	NIL	20`4	NEGATIVE CELLS	1+	N	CALCIFICATION
MANGALA V S	62	F		YES	12/10/2019	1M	1M	NIL	NIL	TB IN HUSBAND	LOA	NIL	27`39	PUS CELLS	2+	N	PLEURAL EFFUSION
LAKSHMI T G	66	F	YES		6/16/2019	4M	1M	NIL	HTN	NIL	LOA	NIL	27`69	PUS CELLS	1+	N	PNEUMONIA
GOUDAPPA	60	M	YES		8/6/2019	3M	NIL	DEF	NIL	TB IN FATHER	LOA	S + A	16`01	PUS CELLS	2+	N	MILIARY MOTTLING
TEERTHAYYA	82	M		YES	2/1/2020	4M	NIL	NIL	HTN/DM	NIL	LOA	NIL	20`41	NIL	3+	N	PLEURAL EFFUSION
DHARMANNA B S	80	M	YES		1/4/2020	NEW	6D	DEF	DM	NIL	LOA	S	18`33	NIL	3+	N	MILIARY MOTTLING
KAMALAWWA	70	F		YES	1/15/2020	NEW	1W	NIL	COPD	NIL	LOA	TOBACCO	26`94	POSITIVE CELLS	1+	N	PNEUMONIA
SHANTA B S	60	F		YES	1/12/2020	NEW	5D	NIL	NIL	NIL	LOA	NIL	30`98	NIL	1+	KLEB	FIBROSIS
SHRIMANTH B S	65	M	YES		1/20/2020	3M	1M	DEF	COPD	NIL	LOA	S	17`6	POSITIVE CELLS	2+	N	MILIARY MOTTLING
KASHIBAI H S	80	F	YES		1/10/2020	4M	2M	NIL	HTN/DM	TB IN HUSBAND	LOA	NIL	26`53	PUS CELLS	2+	N	PNEUMONIA
SHARANGOUDA	60	M	YES		9/3/2020	NEW	3D	NIL	ICD	NIL	LOA	S + A	16`43	POSITIVE CELLS	3+	N	PLEURAL EFFUSION
SURYAKANTH	61	M	YES		11/10/2019	2M	NIL	NIL	# FEMUR	NIL	LOA	S	22`5	FUNGI	3+	N	PLEURAL EFFUSION
BASAPPA Y	62	M	YES		1/1/2020	NEW	NIL	REL	NIL	TB IN SON & WIFE	LOA	S + A	17`73	NEGATIVE CELLS	2+	N	FIBROSIS
PAKIRAPPA	63	M		YES	2/23/2020	NEW	NIL	NIL	COPD/DM	NIL	LOA	S	17`72	PUS CELLS	2+	N	PNEUMONIA
SHANKARAMMA B K	65	F	YES		8/30/2019	NEW	2D	NIL	NIL	NIL	LOA	NIL	19`17	POSITIVE CELLS	3+	N	SYNPNEUMONIC EFFUSION
YALLAPPA S K	60	M	YES		7/27/2019	NEW	3D	NIL	NIL	NIL	LOA	NIL	24`77	POSITIVE CELLS	2+	N	PLEURAL EFFUSION
SABU G C	62	M	YES		7/7/2019	NEW	1W	NIL	IHD/HTN	NIL	LOA	S + A	20`27	NIL	3+	N	PLEURAL EFFUSION
NIVRUTI S P	62	M	YES		7/1/2019	NEW	1W	NIL	NIL	TB IN WIFE	LOA	S	18`43	NIL	2+	N	PLEURAL EFFUSION
DHARMARAJ S B	62	M	YES		7/11/2019	NEW	1W	NIL	NIL	TB IN WIFE,MOTHER	LOA	S	23`04	NIL	3+	N	PLEURAL EFFUSION
SHRIMANTH	65	M	YES		7/17/2109	NEW	1W	NIL	NIL	NIL	LOA	S	21`66	NEGATIVE CELLS	3+	N	CAVITATION
BHIMAPPA S I	60	M	YES		8/23/2019	NEW	1W	NIL	NIL	NIL	LOA	S	18`91	POSITIVE CELLS	2+	N	MILIARY MOTTLING
SHAMARAO	80	M	YES		8/20/2019	NEW	3W	NIL	HTN	NIL	LOA	NIL	21`77	NIL	3+	N	MILIARY MOTTLING
DASTGIR	68	M	YES		5/8/2019	1M	1M	DEF	NIL	TB IN WIFE	LOA	NIL	20`67	NEGATIVE CELLS	4+	N	PLEURAL EFFUSION
SHIVARAY	60	M		YES	8/20/2019	NEW	1W	NIL	NIL	NIL	LOA	S + A	19`09	FUNGI	3+	N	PLEURAL EFFUSION
SANGAPPA P	65	M	YES		8/22/2019	6M	6M	REL	NIL	NIL	LOA	S + A	19`09	NIL	3+	N	CAVITATION
RUDRAGOUDA	67	M	YES		8/16/2019	NEW	12M	REL	HTN	NIL	LOA	S	17`72	NEGATIVE CELLS	3+	N	CALCIFICATION
BHIMAPPA R	65	M		YES	8/21/2019	NEW	2D	NIL	HTN	NIL	LOA	NIL	22`08	NEGATIVE CELLS	3+	N	CAVITATION
ADIVEPPA	60	M		YES	8/20/2019	NEW	1W	NIL	HTN	NIL	LOA	NIL	22`08	NEGATIVE CELLS	2+	N	CALCIFICATION

MAHADEVI	60	F		YES	8/6/2019	1M	1M	NIL	HTN/IHD	NIL	LOA	NIL	25`16	POSITIVE CELLS	2+	N	CALCIFICATION
BHIMAPPA	60	M	YES		8/17/2019	NEW	1W	DEF	NIL	NIL	LOA	NIL	22`07	NEGATIVE CELLS	3+	N	FIBROSIS
MAHAJANBEE	65	F	YES		8/16/2019	NEW	1W	NIL	IHD,DM	NIL	LOA	NIL	27	NEGATIVE CELLS	2+	N	COLLAPSE
NAGANNA S	62	M	YES		8/16/2019	10M	4M	DEF	HTN/DM	TB IN WIFE,SON	LOA	S	20`16	NIL	3+	KLEB	MILIARY MOTTLING
SHANTAPPA S K	68	M	YES		6/7/2019	NEW	5D	NIL	NIL	NIL	LOA	S + A	21`3	NEGATIVE CELLS	SCANTY	N	CAVITATION
CHANDRAM R H	60	M	YES		5/15/2019	NEW	15D	NIL	NIL	TB IN FATHER	LOA	S	17`77	POSITIVE CELLS	2+	N	FIBROSIS
BALAPPA B S	60	M		YES	6/7/2019	NEW	2D	NIL	NIL	NIL	N	S + A	20`7	NIL	3+	N	MILIARY MOTTLING
CHANDASA H N	65	M	YES		12/20/2019	NEW	1W	NIL	NIL	NIL	LOA	S	16`9	NEGATIVE CELLS	2+	N	PLEURAL EFFUSION
BHIMARAY S W	71	M	YES		3/20/2019	2M	15D	NIL	NIL	NIL	LOA	S	20`27	NIL	3+	ND	FIBROSIS
GOLLALAPPA B H	60	M	YES		12/4/2019	NEW	5D	NIL	COPD	NIL	LOA	S	20`19	NEGATIVE CELLS	3+	ND	MILIARY MOTTLING
SHARANAPPA H P	65	M	YES		2/24/2019	3M	1W	NIL	COPD	NIL	LOA	S + A	18`66	NIL	SCANTY	N	MILIARY MOTTLING
SHRISHAIL D A	60	M	YES		5/10/2019	NEW	15D	NIL	NIL	TB IN WIFE	LOA	A	18	PUS CELLS	3+	N	FIBROSIS
PARASAPPA S B	64	M	YES		4/14/2019	NEW	3D	NIL	LRTI	NIL	LOA	S + A	16`87	NIL	2+	N	PNEUMONIA
SHARANAPPA B N	60	M	YES		1/4/2019	3M	3M	NIL	DM	TB IN FATHER	LOA	S	19`67	POSITIVE CELLS	1+	N	MILIARY MOTTLING
MUSTAQ	60	M	YES		2/2/2019	3M	3M	NIL	HTN/DM	NIL	LOA	S	18`56	NEGATIVE CELLS	3+	N	CALCIFICATION
SHARANAPPA B N	60	M	YES		1/13/2109	3M	1M	NIL	DM	NIL	LOA	NIL	19`55	NEGATIVE CELLS	3+	PNEUMO	PNEUMONIA
BHIMASHI S G	65	M		YES	3/11/2019	NEW	3D	NIL	RHD	NIL	N	S	16`4	PUS CELLS	3+	N	CALCIFICATION
RAMAPPA K K	72	M		YES	1/23/2019	NEW	1W	REL	COPD	NIL	LOA	S + A	16`46	POSITIVE CELLS	2+	N	FIBROSIS
RUDRAPPA T K	61	M	YES		1/28/2019	NEW	1W	NIL	NIL	NIL	LOA	S	16`45	FUNGI	3+	N	MILIARY MOTTLING
SHRIMANTH B B	65	M	YES		1/23/2019	NEW	1W	NIL	NIL	NIL	LOA	S + A	17`18	NEGATIVE CELLS	3+	N	PLEURAL EFFUSION
CHANDRAM K T	69	M	YES		1/10/2019	NEW	NIL	NIL	NIL	NIL	LOA	NIL	15`62	NEGATIVE CELLS	3+	N	MILIARY MOTTLING
BHAGVANT S B	60	M		YES	1/18/2019	NEW	NIL	NIL	NIL	NIL	N	S + A	15`62	ND	2+	ND	MILIARY MOTTLING
SANGAYYA C G	75	M		YES	11/6/2018	NEW	15D	NIL	CANCER	NIL	LOA	S	17`77	NIL	1+	N	FIBROSIS
NEELAMMA T B	62	F	YES		11/1/2018	NEW	10D	NIL	NIL	NIL	LOA	NIL	14`66	NIL	SCANTY	ND	FIBROSIS
BHIMAPPA S W	63	M		YES	11/11/2018	NEW	1W	NIL	NIL	NIL	N	S	17`77	POSITIVE CELLS	2+	ECOLI	MILIARY MOTTLING
BASAVARAJ S M	65	M		YES	11/2/2018	NEW	2D	NIL	NIL	NIL	LOA	A	19`14	NEGATIVE CELLS	SCANTY	ND	PNEUMONIA
SIDDANNA	60	M	YES		2/5/2020	NEW	NIL	REL	NIL	NIL	LOA	S	23`94	ND	3+	ND	MILIARY MOTTLING
PARAPPA	72	M	YES		12/16/2019	3M	3M	NIL	NIL	NIL	LOA	S + A	23`94	PUS CELLS	2+	N	CALCIFICATION
GANESH	60	M	YES		2/3/2020	NEW	NIL	NIL	NIL	NIL	LOA	NIL	27`84	ND	2+	ND	CAVITATION
SIDDANNA S	65	M		YES	1/20/2020	4M	4M	NIL	NIL	NIL	N	NIL	27`84	POSITIVE CELLS	2+	STREP	PLEURAL EFFUSION
IRSANGAPPA	60	M	YES		2/3/2020	NEW	NIL	NIL	NIL	NIL	LOA	NIL	22`5	ND	2+	ND	HYDROPNEUMOTHORAX
SUBHADRA	78	F	YES		8/8/2019	4M	4M	NIL	NIL	NIL	LOA	NIL	28`5	PUS CELLS	2+	N	CALCIFICATION
DRAKSHAYANI	64	F	YES		2/1/2020	NEW	NIL	DEF	HTN	NIL	LOA	NIL	21`77	NEGATIVE CELLS	3+	ND	PNEUMONIA
DEVI	70	F		YES	3/1/2020	3M	3M	NIL	NIL	NIL	N	NIL	26`08	ND	1+	ND	CAVITATION
SONUBAI	70	F		YES	2/29/2020	NEW	1W	NIL	NIL	NIL	LOA	NIL	23`55	POSITIVE CELLS	2+	ND	PNEUMONIA

NAME	APPETITE	HABITS	BMI	GRAMS	ZN STAIN	C/S	COMMON FINDINGS	CT/MRI	LFT	HIV	DIAGNOSIS	TRT STARTED	D B O
BASAPPA	LOA	S + A	23`94	NEGATIVE CELLS	2+	N	FIBROSIS	ND	ND	ND	LEFT APICAL FIBROSIS	TAB AKURIT 4 3-0-0	1,3
MALLANNA	LOA	NIL	18`66	POSITIVE CELLS	3+	N	PLEURAL EFFUSION	ND	N	NEG	LEFT TUBERCULAR PLEURAL EFFUSION	TAB AKURIT 4 3-0-0	2,3
GURANNA B	LOA	S	16`01	NEGATIVE CELLS	1+	N	CAVITATION	ND	ND	ND	PULMONARY TUBERCULOSIS	TAB AKURIT 4 3-0-0	2,3
SADASHIVAPPA N K	LOA	S + A	18`56	PUS CELLS	1+	N	PNEUMONIA	ND	N	ND	LEFT LOWER LOBE PNEUMONIA SEC TO TB	TAB AKURIT 4 3-0-0	2,3
KASANU CHAVAN	N	S + A	20`57	POSITIVE CELLS	1+	N	PNEUMONIA	ND	N	NEG	LEFT PARACARDIAC PNEUMONIA SEC TO TB	TAB AKURIT 4 3-0-0	2,3
KAREPPA A H	N	S + A	24`88	POSITIVE CELLS	2+	STREP	PNEUMONIA	ND	N	NEG	RIGHT UPPER LOBE PNEUMONIA SEC TO TB	TAB AKURIT 4 3-0-0	1,3
MALLAPPA G T	LOA	S	20`67	POSITIVE CELLS	2+	STREP	CAVITATION	ND	N	ND	PULMONARY TUBERCULOSIS	TAB FORECOX 2-0-0	1,3
SHANTAMMA M D	LOA	NIL	20`4	NEGATIVE CELLS	1+	N	CALCIFICATION	ND	N	NEG	PULMONARY TUBERCULOSIS WITH DM WITH ANEMIA	TAB AKURIT 4 3-0-0	2,3
MANGALA V S	LOA	NIL	27`39	PUS CELLS	2+	N	PLEURAL EFFUSION	FCL, PE	N	NEG	RIGHT TUBERCULAR PLEURAL EFFUSION	TAB FDC 2-0-0	2,3
LAKSHMI T G	LOA	NIL	27`69	PUS CELLS	1+	N	PNEUMONIA	ND	N	NEG	LEFT TUBERCULAR PARACARDIAC PNEUMONIA	TAB FDC 3-0-0	1,3
GOUDAPPA	LOA	S + A	16`01	PUS CELLS	2+	N	MILIARY MOTTLING	ND	N	ND	MILIARY TB	NONE	1,3
TEERTHAYYA	LOA	NIL	20`41	NIL	3+	N	PLEURAL EFFUSION	ND	N	NEG	LEFT TUBERCULAR PLEURAL EFFUSION	NONE	1,3
DHARMANNA B S	LOA	S	18`33	NIL	3+	N	MILIARY MOTTLING	ND	N	POS	HIV WITH TB	TAB AKURIT 4 3-0-0	1,3
KAMALAWWA	LOA	TOBACCO	26`94	POSITIVE CELLS	1+	N	PNEUMONIA	ND	N	ND	? ATYPICAL PNEUMONIA	TAB FDC 3-0-0	2,3
SHANTA B S	LOA	NIL	30`98	NIL	1+	KLEB	FIBROSIS	FIBROSIS	N	NEG	LEFT LUNG FIBROSIS , FOCAL SEIZURES	TAB FDC 3-0-0	2,3
SHRIMANTH B S	LOA	S	17`6	POSITIVE CELLS	2+	N	MILIARY MOTTLING	KOCHS	N	NEG	PULMONARY TB	TAB AKURIT 4 3-0-0	1,3
KASHIBAI H S	LOA	NIL	26`53	PUS CELLS	2+	N	PNEUMONIA	ND	N	N	RIGHT LOWER LOBE PNEUMONIA	TAB AKURIT 4 3-0-0	1,3
SHARANGOUDA	LOA	S + A	16`43	POSITIVE CELLS	3+	N	PLEURAL EFFUSION	ND	ABN	NEG	LEFT TUBERCULAR PLEURAL EFFUSION	TAB AKURIT 4 2-0-0	2,3
SURYAKANTH	LOA	S	22`5	FUNGI	3+	N	PLEURAL EFFUSION	ND	N	POS	RIGHT TUBERCULAR PLEURAL EFFUSION	TAB AKURIT 4 3-0-0	1,3
BASAPPA Y	LOA	S + A	17`73	NEGATIVE CELLS	2+	N	FIBROSIS	ND	N	NEG	LEFT APICAL FIBROSIS	TAB FDC 3-0-0	1,3
PAKIRAPPA	LOA	S	17`72	PUS CELLS	2+	N	PNEUMONIA	ND	ND	NEG	FIBROSIS WITH PNEUMONIA	TAB AKURIT 4 3-0-0	2,3
SHANKARAMMA B K	LOA	NIL	19`17	POSITIVE CELLS	3+	N	SYNPNEUMONIC EFFUSION	ND	N	NEG	ANEMIA WITH LEFT SYNPNEUMONIC EFFUSION	TAB AKURIT 4 2-0-0	1,3
YALLAPPA S K	LOA	NIL	24`77	POSITIVE CELLS	2+	N	PLEURAL EFFUSION	ND	N	NEG	RIGHT MINIMAL PLEURAL EFFUSION	TAB AKURIT 4 3-0-0	1,3
SABU G C	LOA	S + A	20`27	NIL	3+	N	PLEURAL EFFUSION	ND	N	NEG	RIGHT PLEURAL EFFUSION WITH IHD	TAB AKURIT 4 3-0-0	1,3
NIVRUTI S P	LOA	S	18`43	NIL	2+	N	PLEURAL EFFUSION	ND	N	NEG	RIGHT TUBERCULAR PLEURAL EFFUSION	TAB AKURIT 4 2-0-0	2,3
DHARMARAJ S B	LOA	S	23`04	NIL	3+	N	PLEURAL EFFUSION	ND	N	NEG	PULMONARY TUBERCULOSIS	TAB AKURIT 4 3-0-0	1,3
SHRIMANTH	LOA	S	21`66	NEGATIVE CELLS	3+	N	CAVITATION	KOCHS	N	NEG	PULMONARY TUBERCULOSIS	TAB AKURIT 4 3-0-0	1,3
BHIMAPPA S I	LOA	S	18`91	POSITIVE CELLS	2+	N	MILIARY MOTTLING	ND	ABN	NEG	MILIARY TB	TAB AKURIT 4 3-0-0	1,3
SHAMARAO	LOA	NIL	21`77	NIL	3+	N	MILIARY MOTTLING	ND	N	NEG	MILIARY TB	TAB AKURIT 4 3-0-0	1,3
DASTGIR	LOA	NIL	20`67	NEGATIVE CELLS	4+	N	PLEURAL EFFUSION	ND	ABN	NEG	RIGHT TUBERCULAR PLEURAL EFFUSION	TAB AKURIT 4 3-0-0	1,3
SHIVARAY	LOA	S + A	19`09	FUNGI	3+	N	PLEURAL EFFUSION	ND	N	NEG	RIGHT TUBERCULAR PLEURAL EFFUSION	TAB AKURIT 4 2-0-0	1,3
SANGAPPA P	LOA	S + A	19`09	NIL	3+	N	CAVITATION	ND	ABN	NEG	IHD WITH PULMONARY TUBRCULOSIS	TAB FDC 3-0-0 + IMJ STM	1,3
RUDRAGOUDA	LOA	S	17`72	NEGATIVE CELLS	3+	N	CALCIFICATION	ND	N	POS	PULMONARY TUBERCULOSIS	TAB AKURIT 4 2-0-0	2,3
BHIMAPPA R	LOA	NIL	22`08	NEGATIVE CELLS	3+	N	CAVITATION	ND	N	NEG	PULMONARY TB WITH ACUTE GE	TAB AKURIT 4 3-0-0	2,3
ADIVEPPA	LOA	NIL	22`08	NEGATIVE CELLS	2+	N	CALCIFICATION	ND	N	POS	RVD WITH PULMONARY TB	TAB AKURIT 4 3-0-0	2,3
MAHADEVI	LOA	NIL	25`16	POSITIVE CELLS	2+	N	CALCIFICATION	ND	N	NEG	IHD, CVA WITH PULMONARY TB	TAB AKURIT 4 3-0-0	2,3

BHIMAPPA	LOA	NIL	22`07	NEGATIVE CELLS	3+	N	FIBROSIS	ND	N	NEG	POTTS SPINE WITH APICAL FIBROSIS	TAB AKURIT 4 3-0-0	2,3
MAHAJANBEE	LOA	NIL	27	NEGATIVE CELLS	2+	N	COLLAPSE	ND	N	NEG	IHD WITH PULMONARY TUBRCULOSIS	TAB AKURIT 4 3-0-0	2,3
NAGANNA S	LOA	S	20`16	NIL	3+	KLEB	MILIARY MOTTLING	ND	ABN	NEG	IHD WITH PULMONARY TUBRCULOSIS	TAB AKURIT 4 3-0-0	1,3
SHANTAPPA S K	LOA	S + A	21`3	NEGATIVE CELLS	SCANTY	N	CAVITATION	ND	N	POS	PULMONARY TUBERCULOSIS	TAB FORECOX 2-0-0	1,3
CHANDRAM R H	LOA	S	17`77	POSITIVE CELLS	2+	N	FIBROSIS	ND	ABN	NEG	RIGHT APICAL FIBROSIS	TAB FORECOX 2-0-0	1,3
BALAPPA B S	N	S + A	20`7	NIL	3+	N	MILIARY MOTTLING	ND	N	NEG	PULMONARY TUBERCULOSIS	TAB FDC 3-0-0	2,3
CHANDASA H N	LOA	S	16`9	NEGATIVE CELLS	2+	N	PLEURAL EFFUSION	ND	N	NEG	RIGHT TUBERCULAR PLEURAL EFFUSION	TAB FORECOX 2-0-0	1,3
BHIMARAY S W	LOA	S	20`27	NIL	3+	ND	FIBROSIS	ND	ABN	NEG	PULMONARY TUBERCULOSIS	TAB AKURIT 4 3-0-0	1,3
GOLLALAPPA B H	LOA	S	20`19	NEGATIVE CELLS	3+	ND	MILIARY MOTTLING	ND	ABN	ND	PULMONARY TUBERCULOSIS	TAB AKURIT 4 3-0-0	1,3
SHARANAPPA H P	LOA	S + A	18`66	NIL	SCANTY	N	MILIARY MOTTLING	ND	N	NEG	PULMONARY TUBERCULOSIS	TAB AKURIT 4 3-0-0	1,3
SHRISHAIL D A	LOA	A	18	PUS CELLS	3+	N	FIBROSIS	ND	N	NEG	RIGHT APICAL FIBROSIS	TAB AKURIT 4 2-0-0	1,3
PARASAPPA S B	LOA	S + A	16`87	NIL	2+	N	PNEUMONIA	ND	N	POS	RIGHT MIDDLE, LOWER LOBE CONSOLIDATION	TAB FORECOX 2-0-0	1,3
SHARANAPPA B N	LOA	S	19`67	POSITIVE CELLS	1+	N	MILIARY MOTTLING	ND	N	NEG	PULMONARY TUBERCULOSIS WITH BRONCHIECTASIS	TAB FORECOX 2-0-0	1,3
MUSTAQ	LOA	S	18`56	NEGATIVE CELLS	3+	N	CALCIFICATION	ND	ABN	NEG	TUBERCULOSIS WITH COR PULMONALE	TAB AKURIT 4 3-0-0	1,3
SHARANAPPA B N	LOA	NIL	19`55	NEGATIVE CELLS	3+	PNEUMO	PNEUMONIA	KOCHS	N	NEG	DM WITH BLIATERAL PNEUMONIA	TAB FORECOX 2-0-0	1,3
BHIMASHI S G	N	S	16`4	PUS CELLS	3+	N	CALCIFICATION	KOCHS	N	NEG	RHD WITH PULMONARY TUBERCULOSIS	TAB AKURIT 4 2-0-0	1,3
RAMAPPA K K	LOA	S + A	16`46	POSITIVE CELLS	2+	N	FIBROSIS	ND	ABN	NEG	PULMONARY TUBERCULOSIS	TAB RCINEX, COMBUTAL	2,3
RUDRAPPA T K	LOA	S	16`45	FUNGI	3+	N	MILIARY MOTTLING	ND	N	NEG	PULMONARY TUBERCULOSIS	TAB AKURIT 4 2-0-0	2,3
SHRIMANTH B B	LOA	S + A	17`18	NEGATIVE CELLS	3+	N	PLEURAL EFFUSION	ND	ABN	NEG	LEFT TUBERCULAR PLEURAL EFFUSION	TAB AKURIT 4 2-0-0	1,3
CHANDRAM K T	LOA	NIL	15`62	NEGATIVE CELLS	3+	N	MILIARY MOTTLING	ND	N	NEG	PULMONARY WITH LARYNGEAL TUBERCULOSIS	TAB AKURIT 4 2-0-0	2,3
BHAGVANT S B	N	S + A	15`62	ND	2+	ND	MILIARY MOTTLING	ND	ABN	NEG	PULMONARY TUBERCULOSIS	TAB AKURIT 4 2-0-0	2,3
SANGAYYA C G	LOA	S	17`77	NIL	1+	N	FIBROSIS	ND	ND	NEG	PULMONARY TUBERCULOSIS	TAB AKURIT 4 2-0-0	2,3
NEELAMMA T B	LOA	NIL	14`66	NIL	SCANTY	ND	FIBROSIS	ND	N	ND	PULMONARY TUBERCULOSIS	TAB AKURIT 4 2-0-0	1,3
BHIMAPPA S W	N	S	17`77	POSITIVE CELLS	2+	ECOLI	MILIARY MOTTLING	ND	ABN	POS	RVD WITH PULMONARY TB	TAB AKURIT 4 2-0-0	2,3
BASAVARAJ S M	LOA	A	19`14	NEGATIVE CELLS	SCANTY	ND	PNEUMONIA	ND	N	POS	RVD WITH PULMONARY TB	TAB AKURIT 4 3-0-0	2,3
SIDDANNA	LOA	S	23`94	ND	3+	ND	MILIARY MOTTLING	ND	ND	POS	RVD WITH PULMONARY TB	TAB FDC 3-0-0	1,3
PARAPPA	LOA	S + A	23`94	PUS CELLS	2+	N	CALCIFICATION	ND	N	NEG	PULMONARY TUBERCULOSIS	TAB AKURIT 4 3-0-0	1,3
GANESH	LOA	NIL	27`84	ND	2+	ND	CAVITATION	ND	ND	ND	PULMONARY TUBERCULOSIS	TAB AKURIT 4 3-0-0	1,3
SIDDANNA S	N	NIL	27`84	POSITIVE CELLS	2+	STREP	PLEURAL EFFUSION	ND	N	NEG	LEFT TUBERCULAR LOCULATED EFFUSION	TAB AKURIT 4 3-0-0	2,3
IRSANGAPPA	LOA	NIL	22`5	ND	2+	ND	HYDROPNEUMOTHORAX	ND	N	NEG	RIGHT HYDROPNEUMOTHORAX	TAB FORECOX 2-0-0	2,3
SUBHADRA	LOA	NIL	28`5	PUS CELLS	2+	N	CALCIFICATION	ND	N	NEG	STEROID INDUCED CUSHINGS + PULM TB	TAB AKURIT 4 3-0-0	1,3
DRAKSHAYANI	LOA	NIL	21`77	NEGATIVE CELLS	3+	ND	PNEUMONIA	ND	N	NEG	BILATERAL EXTENSIVE PNEUMONIA	TAB AKURIT 4 3-0-0	2,3
DEVI	N	NIL	26`08	ND	1+	ND	CAVITATION	ND	N	ND	PULMONARY TUBERCULOSIS	TAB AKURIT 4 3-0-0	2,3
SONUBAI	LOA	NIL	23`55	POSITIVE CELLS	2+	ND	PNEUMONIA	ND	N	NEG	RIGHT BASAL PNEUMONIA	TAB AKURIT 4 3-0-0	2,3