"CLINICAL AND LABORATORY PROFILE OF SPUTUM POSITIVE PULMONARY TUBERCULOSIS AMONG HIV SEROPOSITIVE AND HIV SERONEGATIVE PATIENTS – A CROSS SECTIONAL STUDY"

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Dr. V. A. KOTHIWALE MD, Ph.D
Professor and Head,
Department of General Medicine,
J. N. Medical College,
Nehru Nagar, Belgaum – 10

Date:
Place: Belgaum

Dr. V. D. Patil MD,DCH
Principal,
J. N. Medical College,
Nehru Nagar, Belgaum – 10

Date:
Place: Belgaum

LIST OF ABBREVIATIONS USED

AFB	-	Acid fast bacilli
AIDS	-	Acquired immunodeficiency syndrome
ART	-	Antiretroviral therapy
ATT	-	Antituberuculous treatment
B	-	Basophil
BAL	-	Bronchoalveolar lavage
BCG	-	Bacillus Calmette-Guérin
BMI	-	Body mass index
CDC	-	Centre for Disease Control
CNS	-	Central nervous system
CVS	-	Cardiovascular system
CXR	-	Chest X-ray
DC	-	Differential count
DF	-	Degree of freedom
dL	-	Deci litre
DOTS	-	Directly observed treatment short course
E	-	Eosinophil
EFV	-	Efavirenz
EIA	-	Enzyme immunoassay
ESR	-	Erythrocyte sedimentation rate
FBS	-	Fasting blood sugar
gm	-	Gram
HAART	-	Highly active antiretroviral therapy
Hb	-	Haemoglobin

HIV	-	Human immunodeficiency virus
I. P.	-	In patient
INFγ	-	Interferon gamma
INH	-	Isoniazid
IRIS	-	Immune reconstitution inflammatory syndrome
Kg	-	Kilogram
L	-	Litre
L	-	Lymphocyte
m	-	Meter
M	-	Monocyte
MDR	-	Multidrug resistance
meq	-	Milli equivalent
mg	-	Milligram
Min	-	Minute
mm	-	Millimeter
MTB	-	Mycobacterium tuberculosis
N	-	Neutrophil
n	-	Number
NNRTIs	-	Non nucleoside reverse transcriptase inhibitors
NRTIs	-	Nucleoside reverse transcriptase inhibitors
NVP	-	Nevirapine
OI	-	Opportunistic infections
PCR	-	Polymerase chain reaction
PGL	-	Persistent generalized lymphadenopathy
PI	-	Protease inhibitors

PPBS	-	Post prandial blood sugar
PPD	-	Purified protein derivative
PTB	-	Pulmonary tuberculosis
PVL	-	Plasma viral load
RFLP	-	Restriction fragment length polymorphism analysis
RNA	-	Ribose nucleic acid
RS	-	Respiratory system
RTV	-	Ritonavir
SQV	-	Sequinivir
TB	-	Tuberculosis
TBM	-	Tubercular meningitis
TC	-	Total count
UNICEF	-	United Nations Children's Fund
WHO	-	World Health Organization
x^2	-	Chi square

ABSTRACT

Background and objectives

Tuberculosis continues to be the most important cause of morbidity and mortality worldwide, killing approximately two million people each year. The pattern of clinical presentation of TB is reflected in the microbiological and radiological characteristics of the disease. However coinfection with human immunodeficiency virus (HIV) poses special diagnostic and therapeutic challenges. The objective of the present study was to assess the clinical and laboratory profile of sputum positive pulmonary tuberculosis among HIV seropositive and HIV seronegative patients.

Methodology

Present one year cross sectional study was conducted in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum on 104 patients with sputum positive pulmonary tuberculosis patients during the period of January 2009 to December 2009. Routine investigations, haemogram, sputum smears for AFB, chest X-ray were done.

Results

Seroprevalence of HIV among pulmonary tuberculosis patients was 23.08% with male predominance (79.17%) and highest (50%) in the age group of 31 to 40 years. The most common symptom was cough with expectoration (100%). Fever (95.83%), weight loss (83.33%), loss of appetite (70.83%) and diarrhoea (12.50%) were more common among HIV seropositives compared to HIV seronegatives. On examination, anaemia, undernourishment,

lymphadenopathy and the presence of opportunistic infections like oral candidiasis (66.67%), herpes zoster scar (50%) and genital lesions (16.67%) were more common among HIV seropositives compared to HIV seronegatives. Chest X-ray findings showed, cavitation, fibrosis and fibrocavitatory lesions predominantly among HIV seronegatives while infiltration and miliary mottling was seen in HIV seropositives. Upper zone infiltration, cavitation and fibrosis were more commonly involved among HIV seronegatives compared to HIV seropositives.

Conclusion

Clinical and laboratory presentation varied among HIV seropositives and HIV seronegatives and presence of opportunistic infections may assist in identifying PTB patients with HIV infection.

Key words

Human immunodeficiency virus; Pulmonary tuberculosis; TB-HIV coinfection; Tuberculosis;

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INTRODUCTION

About one third of world population suffers from mycobacterium tuberculosis (MTB) infection. Tuberculosis (TB) continues to be the most important cause of morbidity and mortality worldwide killing approximately two million people each year.[1]

It is estimated that there are about 14 million cases of TB in India, about two million new cases occur annually and home of one fourth the world's TB prevalence.[2]

Fuelled by high prevalence of human immunodeficiency virus (HIV) infection, the incidence rate of TB is very high as TB is the most common opportunistic infection in HIV seropositive patients, when the immune system weakens as a result of HIV infection.[1]

A retrospective study conducted at National Chest Hospital, Kingston, Jamaica among patients admitted with PTB during 1995 to 2001 showed 11.6% of the cases had HIV positive infection.[3] Another study conducted at Chest Clinic, AIIMS, New Delhi during 2000 to 2002 showed that 9.4% of PTB cases had HIV seropositive infection.[4]

The life time risk of developing tuberculosis is 60% in HIV seropositive patients compared to 10% in HIV seronegative patients.[2]

Human immunodeficiency virus related PTB can present across, a wide range of immune status, and its clinical presentation also varies accordingly. In

the early stage of HIV infection, clinical manifestations of TB are quite similar to that of HIV seronegative patients, however at a later phase, atypical features are common.[1]

Most common symptoms of PTB are cough with expectoration and weight loss. Constitutional symptoms like fever, night sweats, generalized weakness, loss of appetite and other respiratory symptoms like chest pain, haemoptysis and breathlessness.

Weight loss and fever are more common in HIV seropositive PTB patients than in those who are HIV seronegative. Conversely, cough and haemoptysis are less common in HIV seropositive PTB than in those are HIV seronegative as there is less cavitation, inflammation and endobronchial irritation.[5]

The chest X-ray (CXR) pattern also varies among HIV seropositive and HIV seronegative patients. Classical pattern is more common in HIV seronegative that is upper lobe infiltration, cavitation, pulmonary fibrosis while interstitial infiltration are common in middle and lower zones and less cavitation in HIV seropositive.[5]

A study was conducted in RIMS Hospital, Manipur on 100 HIV seropositive and 100 HIV seronegative patients. Pulmonary tuberculosis was found in 55% HIV seropositive and 25% in HIV seronegative patients. The most common clinical symptom was fever which was present in both the groups. Cough with expectoration was present among HIV seronegative patients (80%) and weight loss was present in HIV seropositive patients (38.18%). Chest X-ray

revealed pulmonary infiltrates and lower zone involvement in 69% of HIV seropositive and 68% in HIV seronegative cases.[6]

Similar study done in Mangalore, South India on 29 with HIV seropositive, 546 were HIV seronegative patients showed that, pulmonary tuberculosis was recorded in 86% of HIV seropositive and 12.2% in HIV seronegative cases. Most Clinical symptoms were fever and cough with expectoration. Chest X-ray revealed upper lobe lesions in HIV seronegative cases.[7]

Another study conducted on 257 patients in Ethiopia has showed that 52.2% were HIV seropositive and 47.8% were HIV seronegative. The most common clinical symptoms were fever, weight loss, night sweats and cough with expectoration. Chest X-ray showed classical pattern of PTB in 52.4% of which 28.6% were HIV seropositive.[1]

A study was conducted to assess single sputum specimen for acid fast bacilli (AFB) on 164 PTB patients for evaluation of HIV infection. Among them 20 patients (12%) had HIV infection.[8]

Another study conducted in Nigeria showed that, 48% of patients were smear positive for AFB and of the 85% patients tested for HIV, 55% were positive. Coinfected patients had less cavitations and lung involvement on X-rays than patients without HIV.[9]

In a prospective case series of 180 patients with PTB authors assessed characteristics of PTB in HIV seropositive and seronegative patients at

Northeastern region of Brazil. The results showed 104 patients had sputum smear positive for AFB and among them 24 patients were HIV seropositive and 80 patients were HIV seronegative.[10]

As the prevalence of PTB is increasing among HIV seropositive patients with a wide range of immune status and clinical presentations, the present study was undertaken to assess the clinical and laboratory profile of sputum positive PTB among HIV seropositive and HIV seronegative patients.

OBJECTIVES

The objective of the present study was to study the clinical and laboratory profile of sputum positive PTB among HIV seropositive and HIV seronegative patients.

REVIEW OF LITERATURE

Tuberculosis is one of the oldest diseases known to affect the human and caused by the bacteria, MTB. The tubercle lesions have been found in the vertebrae of Neolithic human in Europe and Egyptian mummies perhaps as early as 3700 years B.C. The infectious agent was discovered by Robert Koch in 1882.

Acquired immuno deficiency syndrome was first recognized in the United States in the Summer of 1981. In 1983, HIV was isolated from a patients with lymphadenopathy and by 1954 it was demonstrated clearly to be the causative agent of AIDS.

Tuberculosis and HIV have been closely linked since the emergence of AIDS. HIV infection has contributed to a significant increase in the worldwide incidence of TB.[11,12] The dual epidemic of HIV and TB (HIV-TB) is a concern for India where both these diseases are prevalent in epidemic proportions.[13] Although HIV related TB is both treatable and preventable, incidence rates continue to climb in developing nations where HIV infection and TB are endemic and resources are limited. TB and HIV are intricately linked to malnutrition, unemployment, alcoholism, drug abuse, poverty and homelessness.[14]

Thus, co-infection with HIV-TB is not only a medical malady, but a social and economic disaster and is justly described as the “cursed duet”.[15]

TUBERCULOSIS

Epidemiology

World Health Organization (WHO) has estimated that at least one third of the world's population (more than two billion people) is infected with TB.[16,17] Every year, eight million people become sick with TB and 95% of them are in the developing world,[17,18] India China and indonesia account for half of the cases.[19] TB is a leading cause of death among adults aged 15 to 59 years in developing countries. Tuberculosis kills more than three million people each year worldwide, constituting about 25% of avoidable adult deaths in the developing world; more deaths than from AIDS, malaria and diarrhea combined. Tuberculosis kills more women than from all causes of maternal mortality put together. Without urgent action, it is estimated, 70 million people may die of TB within the next two decades.

About 40% of TB cases in the world live in the South East Asian Region; each year, nearly three million cases and 750,000 deaths are estimated to occur in this region (Bangladesh, India, Indonesia, Myanmar and Thailand contribute more than 95% of the regional cases). Every day, more than 1,500 people die of TB in South East Asia, making TB a major health problem in the region.[20] The situation is further complicated by the rapidly spreading HIV epidemic: of the 6 million adults living with HIV in the region, more than 3 million are likely to be additionally infected with TB.

Mycobacterium tuberculosis

Tuberculosis is a bacterial disease caused by MTB. These organisms are also known as tubercle bacilli or as AFB.[5]

Tuberculous infection and tuberculosis

Tuberculous infection occurs when a person carries the tubercle bacilli inside the body, but the bacteria are in small numbers and are dormant. These dormant bacteria are kept under control by the body's defences and do not cause disease. Many people have tuberculous infection and are well. Tuberculosis is a state in which one or more organs of the body become diseased as shown by clinical symptoms and signs.[5]

Sources of infection

The most important source of infection is the patient with PTB, and who is coughing. Coughing produces tiny infectious droplet nuclei. A single cough can produce 3000 droplet nuclei. Droplet nuclei can also be spread into the air by talking, sneezing, spitting and singing, and can remain suspended in the air for long periods. Direct sunlight kills tubercle bacilli in five minutes, but they can survive in the dark for long periods. Transmission therefore generally occurs indoors. Droplet nuclei are so small that they avoid the defences of the bronchi and penetrate into the terminal alveoli of the lungs, where multiplication and infection begin.[5]

Two factors determine an individual's risk of infection:

- The concentration of droplet nuclei in contaminated air.
- The length of time he or she breathes that air.[5]

Risk of progression of infection to disease

Infection with MTB can occur at any age. Once infected with MTB, a person can stay infected for many years, probably for life. The vast majority (90%) of people without HIV infection who are infected with MTB do not develop TB. In these, asymptomatic individuals, the only evidence of infection may be a positive tuberculin skin test. Infected persons can develop TB at any time. The disease can affect most tissues and organs, but especially the lungs. The chance of developing disease is greatest shortly after infection and steadily lessens as time goes by. Infected infants and young children are at greater risk of developing disease than older people because they have an immature immune system. Tuberculosis is also more likely to spread from the lungs to other parts of the body in this age group. Children who develop disease usually do so within two years following exposure and infection. Most do not develop disease in childhood but may do so later in life. Various physical or emotional stresses may trigger progression of infection to disease. The most important trigger is weakening of immune resistance, especially by HIV infection.[5]

Natural history of untreated TB

Without treatment, by the end of 5 years 50% of PTB patients will be dead, 25% will be healthy (self cured by a strong immune defence) and 25% will remain ill with chronic infectious TB.[5]

Pathogenesis of Tuberculosis

Primary infection

Primary infection occurs in people who have not had any previous exposure to tubercle bacilli. Droplet nuclei, which are inhaled into the lungs, are

so small that they avoid the mucociliary defences of the bronchi and lodge in the terminal alveoli of the lungs. Infection begins with multiplication of tubercle bacilli in the lungs. The resulting lesion is the Ghon focus. Lymphatics drain the bacilli to the hilar lymph nodes. The Ghon focus and related hilar lymphadenopathy form the primary complex. Bacilli may spread in the blood from the primary complex throughout the body. The immune response (delayed hypersensitivity and cellular immunity) develops about four to six weeks after the primary infection. The size of the infecting dose of bacilli and the strength of the immune response determine what happens next. In most cases, the immune response stops the multiplication of bacilli. However, a few dormant bacilli may persist. A positive tuberculin skin test would be the only evidence of infection. In a few cases the immune response is not strong enough to prevent multiplication of bacilli, and disease occurs within a few months.[5]

Outcomes of primary infection

- No clinical disease, Positive tuberculin skin test (Usual outcome: 90% of cases).
- Hypersensitivity reactions for example erythema nodosum, phlyctenular conjunctivitis dactylitis.
- Pulmonary and pleural complications for example TB pneumonia and collapse / consolidation pleural effusion.
- Disseminated disease, lymphadenopathy (Usually cervical) meningitis, pericarditis, military disease.[5]

<u>Post primary tuberculosis</u>

Post primary TB occurs after a latent period of months or years following primary infection. It may occur either by reactivation of the dormant tubercle

bacilli acquired from a primary infection or by reinfection. Reactivation means that dormant bacilli persisting in tissues for months or years after primary infection start to multiply. This may be in response to a trigger, such as weakening of the immune system by HIV infection. Reinfection means a repeat infection in a person who has previously had a primary infection.[5]

The immune response of the patient results in a pathological lesion that is characteristically localized, often with extensive tissue destruction and cavitation. Post primary TB usually affects the lungs but can involve any part of the body. The characteristic features of post primary PTB are the following:

- Extensive lung destruction with cavitation.
- Positive sputum smear.
- Upper lobe involvement.
- Usually no intrathoracic lymphadenopathy.[5]

Patients with these lesions are the main transmitters of infection in the community.

Pulmonary Tuberculosis

- Cavities, upper lobe infiltrates, fibrosis, progressive pneumonia, endobronchial.[5]

Extrapulmonary tuberculosis (EPTB)

- Common – Pleural effusion, Lymphadenopathy (Usually cervical), Central nervous system (Meningitis, cerebral tuberculoma), Pericarditis

(Effusion / Constrictive), gastrointestinal (Ileocaecal, peritoneal), spine and other bone and joint.

- Less common – Empyema, Male genital tract (Epididymitis, orchitis), female genital tract (Tubo-ovarian, endometrium), Kidney and adrenal gland.[5]

HUMAN IMMUNODEFICIENCY VIRUS INFECTION

Epidemiology

As on December 2008, nearly 39.5 million people globally are living with HIV/AIDS.22 Estimated number of people living with HIV/AIDS, in India by 2008 are 2 to 3.1 million.[21]

Almost 25 years have now elapsed, in which HIV infection has changed from a fatal condition to a manageable chronic illness. Twenty five years, in which the development of antiretroviral therapy (ART) has been one of the dramatic advances in the history of medicine. However, for the vast majority of people living with HIV/AIDS, ART is still light years away largely inaccessible in resource poor countries where HIV continues to devastate families, communities and societies, especially the poor and the socially marginalized.[5]

HIV infection can be transmitted through:

- Unprotected sexual intercourse with an infected partner;
- Injection or transfusion of contaminated blood or blood products;

- Sharing unsterilized injection equipment that has been previously used by someone who is infected;
- Maternofetal transmission (during pregnancy, at birth, and through breastfeeding).[5]

The risk of occupational HIV transmission from contaminated needles to healthcare workers was found to be 0.3% (in case series performed prior to the availability of potent anti retroviral therapy).[5]

Immunopathogenesis of human immunodefiency virus infection

Human immunodefiency virus infects cells that have the CD4 antigen molecules on their surface. These cells are principally the helper subset of T_1-lymphocytes, which are central to cell-mediated immunity. They are called CD4+ T_1-lymphocytes. In recent years it has also been discovered that HIV needs other molecules, called chemokines, on the cell surface to gain entry into the cell. Patients who do not have some of these specific chemokines (for example, CCR5) are more resistant to HIV infection. Others, who have molecular changes in these chemokine receptors, progress more slowly to AIDS.[5]

The critical abnormality resulting from HIV infection is a progressive decline in the number of CD4+ T_1-lymphocytes. These cells are the most important cells in the cell mediated immune response. In addition the surviving CD4+ T_1-lymphocytes do not perform their functions as well as they did before infection. Progressive HIV infection therefore causes progressive decline in immunity.[5]

Natural history

Acute human immunodefiency virus infection

Acute HIV infection is also called “primary HIV infection” or “acute seroconversion syndrome”. Between 40% and 90% of new HIV infections are associated with symptomatic illness. The time from exposure to onset of symptoms is usually two to four weeks. Some people present with a glandular fever like illness (fever, rash, arthralgia and lymphadenopathy). Occasionally acute neurological syndromes may occur, which are often self limiting. These include aseptic meningitis, peripheral neuropathy, encephalitis and myelitis.[5]

Most symptomatic patients seek medical help. However, the diagnosis is infrequently made, for several possible reasons. First, the clinician may not consider HIV infection. Secondly, the nonspecific clinical features may be mistaken for another cause, e.g. malaria. Thirdly, standard serological tests at this stage are usually negative. Serological tests first become positive about 4 to 12 weeks after infection, with over 95% of patients “seroconverting” within six months of HIV transmission. The diagnosis of acute HIV infection is best established by demonstration of HIV RNA in plasma.[5]

Asymptomatic human immunodefiency virus infection

In adults, there is a long, variable, latent period from HIV infection to the onset of HIV related disease and AIDS. A person infected with HIV may be asymptomatic for 10 years or more. The vast majority of HIV infected children are infected in the perinatal period. The period of asymptomatic infection is

shorter in children than in adults. A few infants become ill in the first few weeks of life. Most children start to become ill before two years of age. A few children remain well for several years.[5]

Persistent generalized lymphadenopathy (PGL)

Persistent generalized lymphadenopathy is defined as enlarged lymph nodes involving at least two sites other than inguinal nodes. At this time, the lymph tissue serves as the major reservoir for HIV. Persistent generalized lymphadenopathy occurs in about one third of otherwise healthy HIV infected people. The enlarged lymph nodes are persistent, generalized, symmetrical and non-tender. Persistent generalized lymphadenopathy has no particular prognostic significance.[5]

Progression from HIV infection to HIV related disease and AIDS

Almost all (if not all) HIV infected people, if untreated, will ultimately develop HIV related disease and AIDS. Some HIV-infected individuals progress more quickly than others to HIV related disease and AIDS. The rate of progression depends on virus and host characteristics. Virus characteristics include type and subtype: HIV-1 and certain HIV-1 subtypes may cause faster progression. Host characteristics that may cause faster progression include: age less than five years; age more than 40 years; concurrent infections; and genetic factors.[5]

Advancing immunosuppression

As HIV infection progresses and immunity declines, patients become more susceptible to infections. These include TB, pneumonia, recurrent fungal infections of the skin and oropharynx, and herpes zoster. These infections can

occur at any stage of progression of HIV infection and immunosuppression. Some patients may develop constitutional symptoms (unexplained fever and weight loss), previously known as "AIDS-related complex" (ARC). Some patients develop chronic diarrhoea with weight loss, often known as "slim disease".[5]

Certain specific HIV related diseases occur predominantly with severe immunosuppression. These include certain opportunistic infections (for example, cryptococcal meningitis) and certain tumours (for example, Kaposi sarcoma). At this late stage, unless patients receive specific therapy for HIV infection, they usually die in less than two years. This late stage is sometimes known as "full-blown AIDS".[5]

WHO clinical staging system for HIV infection and HIV related disease

World Health Organization has developed a clinical staging system (originally for prognosis), based on clinical criteria. The definition of symptoms, signs and diseases is according to clinical judgement. Clinical condition or performance score, whichever is the higher, determines whether a patient is at clinical stage 1, 2, 3 or 4. Clinical stage is important as a criterion for starting antiretroviral (ARV) therapy.[22]

WHO clinical staging system for HIV infection

Stage 1	• Asymptomatic
	• Persistent generalized lymphadenopathy
Stage 2	• Moderate unexplained weight loss (<10% of presumed or measured body weight)
	• Recurrent respiratory tract infections sinusitis, tonsillitis, otitis media and pharyngitis)
	• Herpes zoster
	• Angular cheilitis
	• Recurrent oral ulceration
	• Papular pruritic eruptions
	• Seborrhoeic dermatitis
	• Fungal nail infections
Stage 3	• Unexplained severe weight loss (>10% of presumed or measured)
	• Unexplained chronic diarrhoea for longer than one month
	• Unexplained persistent fever (above 37.6°C intermittent or constant, for longer than one month)
	• Persistent oral candidiasis
	• Oral hairy leukoplakia
	• PulmonaryTB(current)
	• Severe bacterial infections (such as pneumonia, empyema, pyomyositis, bone or joint infection, meningitis or bacteraemia)
	• Acute necrotizing ulcerative stomatitis, gingivitis or periodontitis
	• Unexplained anaemia (<8 g/dl), neutropaenia (<0.5 × 109 per litre) or chronic thrombocytopaenia (<50 × 109 per litre)

Stage 4

- HIV wasting syndrome
- Pneumocystis pneumonia
- Recurrent severe bacterial pneumonia
- Chronic herpes simplex infection (orolabial, genital or anorectal of more than one month's duration or visceral at any site)
- Oesophageal candidiasis (or candidiasis of trachea, bronchi or lungs)
- Extrapulmonary tuberculosis
- Kaposi's sarcoma
- Cytomegalovirus infection (retinitis or infection of other organs)
- Central nervous system toxoplasmosis
- HIV encephalopathy
- Extrapulmonary cryptococcosis including meningitis
- Disseminated non-tuberculous mycobacterial infection
- Progressive multifocal leukoencephalopathy
- Chronic cryptosporidiosis (with diarrhoea)
- Disseminated mycosis (coccidiomycosis or histoplasmosis)
- Recurrent non-typhoidal Salmonella bacteraemia
- Lymphoma (cerebral or B-cell non-Hodgkin) or other solid HIV associated tumours
- Invasive cervical carcinoma
- Atypical disseminated leishmaniasis
- Symptomatic HIV associated nephropathy or symptomatic HIV associated cardiomyopathy[22]

Sero-prevalence of HIV among TB patients

In sub-Saharan Africa, HIV seroprevalence rates among patients with TB are high, ranging from 24% to 67%.[23] In Asia, the rate of HIV infection among TB patients has been lower. The seroprevalence of HIV amongTBpatients in various parts of India has been increasing steadily.[21]

Several studies in India have reported HIV seropositivity rates ranging from 0.4% to 20.1%.[24,25] In certain cities such as Chennai and Mumbai, a higher prevalence has been observed.[26,27,28]

In Pune, the HIV seroprevalence rate was observed to have steadily increased from 3.2% in 1991 to 20.1% in 1996, among patients with PTB.[26] Human immunodefiency virus seroprevalence rate at a tertiary care referral hospital at New Delhi was reported to have increased from 0.4% (1994-1999) to 9.4% (2000-2002).[4,24] The occurrence of localized epidemics and/or selection bias could be the cause of this large regional variation in reported rates of HIV seropositivity among patients with TB.[14]

It is expected that HIV will increase the number of TB cases in India by at least 10% and by a considerably higher percentage if HIV becomes much more widespread.

Tuberculosis among patients with AIDS

In countries with high prevalence of TB as well as HIV, TB is a very common presentation of AIDS and, in most cases, it is the earliest manifestation. Clinical and surveillance data show that in Asia, TB is the most important life

threatening opportunistic disease associated with HIV, For example, in Thailand, 60% of AIDS patients seen in a Bangkok hospital between 1985 and 1991 had PTB[29] and TB is the most common reported cause of hospitalization and death among AIDS patients in Thailand. Surveillance data from India and Nepal show that 83% and 56% of AIDS patients had TB.[30]

TB-HIV COINFECTION

Epidemiology

World Health Organization estimated 9.2 million new cases of TB globally in 2006 (139 per 100,000), of whom 709,000 (7.7%) were HIV positive. India, China, Indonesia, South Africa and Nigeria rank first to fifth in terms of incident TB cases.[31]

Impact of TB-HIV coinfection

Studies from many countries show that the rate of breakdown to clinical TB in TB-HIV infected people is in the range of 5 to 15% per year compared to 0.2% among those infected only with TB.[16,17] The life time risk of developingTBcould be as high as 50%.[32,33] The HIV epidemic has led to a 100 to 300% increase in TB notifications in countries like Zambia, Tanzania, Burundi and Malawi, many of which had outstanding TB control programmes.[34]

In the developing countries, drug resistance is common. In Nepal, during 1994-97, the overall prevalence of primary drug resistance was 9.8%, including 1.1% multi drug resistance (MDR).[35] Prevalence of primary MDR from India (3.4%) and Thailand (2.1%) has also been reported.[16] Prevalence of acquired drug

resistance varies according to the drug regimens used and the efficiency of the control programme. Programmes employing Rifampicin without achieving a high success rate may result in high prevalence of 40 to 50% MDR, as reported from some parts of India and Nepal.

With proper implementation of Directly Observed Treatment Short-course (DOTS), failures are fewer and MDR rates are likely to be much lower. Data from Thailand show that drug resistance is more common among patients with HIV. The higher prevalence of drug resistance in TB-HIV cases can be attributed to the very high baciliary load and probably a number of drug resistant variants. In a large study of 877 patients including 192 HIV positive cases, the prevalence of MDR was 1.5% overall, 5.2% in HIV positive cases and 0.4% in HIV negative cases ($p<0.00l$).[36]

Interaction of HIV and MTB

The influence of HIV and MTB on immunoregulation by the host is bidirectional. The incidences of post-primary TB and reactivation TB are increased in HIV infected patients in comparison to HIV seronegative individuals.[37,38] For example, the incidence of post-primary TB is increased from 5% to 30% in HIV infected subjects. Further, it is likely that TB enhances immunodeficiency in patients with chronic HIV infection.[39] Despite adequate therapy of TB the subsequent morbidity and mortality is increased in patients with HIV infection in comparison to HIV seronegative patients with TB.[40,41] While most opportunistic infections, including all other mycobacterial diseases, occur in the advanced stages of HIV infection, patients can develop TB at any

stage, regardless of the levels of circulating CD4+ T-cells.[42] More than 50% of cases with PTB occur in patients with CD4 counts more than 200 cells/mm^3 in the peripheral blood.[38] However, the incidence of disseminated TB is much higher in patients with advanced immunodeficiency.[43] Recently, it was shown that the risk of developing TB is already significantly increased in the first year following HIV antibody seroconversion.[44] The factors that lead to TB reactivation in HIV infection have not been determined in detail.

HIV infection and susceptibility to tuberculosis

HIV infected persons are at markedly increased risk for primary or reactivation TB and for second episodes of TB from exogenous reinfection. Many factors are involved in the transmission of MTB infection and in the establishment of a state of latent disease. These factors include;

a. Infectiousness of the source case,
b. Virulence of the strain of MTB involved, and
c. Defence mechanisms of the host.

Infectiousness is usually related to the duration and closeness of the contact between the source and the susceptible individual, as well as the clinical condition of the infectious case; untreated, sputum smear positive and cavitary cases are more infectious. Comparatively little is known at present regarding virulence of different strains of MTB and there is a controversy regarding the exact relationship between virulence as seen in animal models and infectiousness seen in humans. Host susceptibility, the third predisposing factor, seems to play a role in the formation of latent foci, although the exact nature of the host response

is unclear. Even prior to the AIDS epidemic, it was clear that certain populations were more susceptible to TB infection than others.[45,46]

Tuberculosis and the course of HIV infection

Studies have shown that the risk of developing TB increases with advancing immunosuppression as measured by the CD4 count.[44] An early study by Law and colleagues demonstrated that HIV infected patients with TB had fewer CD4+ T cells in their lungs compared to HIV negative patients.[47,48]

In addition, interferon gamma (IFN-γ) production too was reduced. However, they had more CD8+ T cells in their lungs which has been confirmed in a more recent study.[49] Nowakowski and colleagues had earlier reported that children with AIDS and TB had low levels of CD4+ T cells in their lungs, although the number of CD8+ T cells did not seem to be increased.[50,51] Overall, the immune derangements that act locally in the lungs of patients with HIV infection and TB have not been fully characterized, although it is likely that the most important changes are due to a decrease in the number and function of CD4+ T cells, with a resulting loss of production of the important pro-inflammatory cytokine IFN-γ.

These immune changes seem to result in an initially more benign presentation of TB in AIDS patients (mediastinal adenopathy, non-cavitary, smear-negative disease). When immune function is reconstituted in these patients, through the administration of highly active anti-retroviral therapy (HAART), an exuberant “paradoxical” reaction often occurs, characterized by fever and enlarged, necrosing lymph nodes.[52] This is likely to be the result of an

increase in the number and function of CD4+ T cells and production of the pro-inflammatory cytokine IFN γ. Exposure of alveolar macrophages and lymphocytes from HIV infected patients to MTB in vitro up-regulates retroviral replication.[53,54]

Pleural fluid from patients with TB increases HIV replication in activated lymphocytes[55] and in HIV infected patients with pulmonary TB the concentrations of retroviral ribose nucleic acid (RNA) in bronchoalveolar lavage fluid are highest in areas of tuberculous involvement.[56] Mycobacterium TB probably increases HIV replication by inducing macrophages to produce tumor necrosis factor alpha, interleukin-l and interleukin-6.[55,56]

Clinical studies have shown the detrimental effects of TB on the course of HIV infection. The risk of death in HIV infected patient with TB was reported to be twice that in HIV infected patients without TB independently of the CD4 cell count.[57] The high mortality rate among patients with TB appeared to be due to progressive HIV infection rather than TB The degree of immunosuppression is the most important predictor of survival in HIV infected patients with TB since negative tuberculin skin tests, prior opportunistic infections, and low CD4 cell counts are associated with increase mortality.[58,59]

CLINICAL PRESENTATION OF PTB AND TB-HIV COINFECTION

Symptoms

The most important symptoms in the diagnosis of PTB are the following:

- Cough for more than two or three weeks;

- Sputum production;
- Weight loss.

Over 90% of patients with sputum smear-positive PTB develop a cough soon after disease onset. However, cough is not specific to PTB. Cough is common in smokers and in patients with acute upper or lower respiratory tract infection. Most acute respiratory infections resolve within three weeks. Therefore a patient with a cough for more than two or three weeks is a PTB suspect and must submit sputum samples for diagnostic microscopy. Patients with PTB may also have other symptoms. These may be respiratory or constitutional (general or systemic).

Respiratory: Chest pain, haemoptysis, breathlessness.

Constitutional: Fever, night sweats, tiredness, loss of appetite, secondary amenorrhoea.

Patterns of HIV related TB

- Pulmonary TB
- Extrapulmonary TB

Pulmonary involvement occurs in about 75% of all HIV infected patients with TB.[5,60] Unlike other opportunistic infections which have a selective range of CD4 in which the disease occurs, TB occurs throughout the course of HIV. The interaction between HIV and TB in persons coinfected with HIV and TB is bidirectional and synergistic.

Weight loss and fever are more common in HIV positive PTB patients than in those who are HIV negative. Conversely, cough and haemoptysis are less common in HIV positive PTB patients than in those who are HIV-negative. This is probably because there is less cavitation, inflammation and endobronchial irritation in HIV positive patients.[5]

PTB in early and late HIV infection[5]

Features of PTB	**Stage of HIV infection**	
	Early	**Late**
Clinical picture	Often resembles post primary PTB	Often resembles primary PTB
Sputum smear results	Often positive	Often negative
CXR appearance	Often cavities	Often infiltrates with no cavities

Clinical features suggestive of HIV coinfection in TB patients[5]

Past history

- Sexually transmitted infection.
- Herpes zoster (shingles), which often leaves a scar.
- Recent or recurrent pneumonia.
- Severe bacterial infections (sinusitis, bacteraemia, pyomyositis).
- Recent treated TB.

Symptoms

- Weight loss (> 10 kg or > 20% of original weight).
- Diarrhoea (> 1 month).
- Retrosternal pain on swallowing (suggests oesophageal candidiasis).

- Burning sensation of feet (peripheral sensory neuropathy).

Signs

- Scar of herpes zoster.
- Pruritic (itchy) papular skin rash.
- Kaposi sarcoma.
- Symmetrical generalized lymphadenopathy.
- Oral candidiasis.
- Angular cheilitis.
- Oral hairy leukoplakia.
- Necrotizing gingivitis.
- Giant aphthous.
- Persistent painful genital ulceration.

Investigations

- Unexplained anaemia.
- Leukopenia.
- Thrombocytopenia.

Extrapulmonary TB occurs predominantly in co-infected patients with CD4+ T-cell counts of <200 cells/mm^3. The most common feature of EPTB is cervical lymphadenopathy. The involved nodes are firm and generally not painful on palpation. The formation of abscesses and draining fistulas, as well as fever and malaise are common.[5]

The clinical presentation of tuberculous meningitis (TBM) is similar in HIV infected patients and in immunocompetent patients, except that intracerebral

mass lesions are more common in HIV infected patients.104 Tuberculous meningitis often presents with nonspecific prodromal symptoms, such as headache, nausea and vomiting followed by elevated temperature and clinical signs of meningeal irritation. The basal meninges are usually involved and cranial palsies of the third and sixth nerves are common. Mono, hemi, or paraparesis as well as seizures can occur. In case of doubt, a lumbar puncture should be performed without delay.[5]

A micronodular pattern is seen on CXR in miliary TB. On radiological criteria alone, military TB cannot be distinguished from pulmonary cryptococcosis. Miliary dissemination of TB can also be detected on abdominal ultrasound of the spleen and liver and may involve the adrenals (Addison's disease) too.[5]

Distinguishing Other HIV-Related Pulmonary Diseases from Pulmonary TB

This is a common, and often difficult, diagnostic problem. Several diseases in HIV positive individuals may present in a similar way, with cough, fever, sometimes chest signs, and CXR shadowing. Pneumonia is the most frequent and important differential diagnosis. Pneumonia can also occur as a coinfection with TB. In each case, a careful clinical assessment is needed. Send sputum samples for AFBs if the patient has had cough for three weeks or more.[5]

<u>Acute bacterial pneumonia</u>

This is common in HIV positive patients. The shorter history usually differentiates pneumonia from PTB. The most common pathogen is *Streptococcus pneumoniae*. Regardless of HIV status, acute bacterial pneumonia

usually responds well to standard treatment with penicillin, cotrimoxazole or ampicillin.[5]

Kaposi sarcoma (KS)

The clinical recognition of KS is straightforward when there are typical lesions on the skin and mucous membranes. The diagnosis of pulmonary or pleural KS is more difficult. The patient usually presents with cough, fever, haemoptysis and dyspnoea, and usually has KS lesions elsewhere. Chest X-ray shows a diffuse nodular infiltrate (with infiltrates spreading out from the hilar regions) or pleural effusion. The pleural fluid is usually blood-stained. Cytology may provide the diagnosis. It can be difficult to rule out concurrent PTB.[5]

Pneumocystis carinii pneumonia (PCP)

Adult PCP is less commonly seen in patients with AIDS in sub-Saharan Africa than in developed countries. The patient usually presents with dry cough and progressive dyspnoea.

The table below shows the clinical and CXR features that help to distinguish PCP from PTB.[5]

	Typical of PCP	**Typical of TB**
Symptoms	Dry cough, Sputum mucoid (if any), Dyspnoea	Productive cough, purulent sputum, pleuritic chest pain, haemoptysis
Signs	May be normal fine inspiratory crackles	Signs of consolidation, signs of pleural effusion
CXR	Bilateral diffuse interstitial shadowing may be normal	Lobar consolidation, cavitation, pleural effusion, intrathoracic lymphadenopathy

DIAGNOSIS OF PTB AND TB-HIV COINFECTION

Sputum Smear Microscopy

Persons with cough for two weeks, or more, with or without other symptoms suggestive of TB, should be promptly identified as PTB suspects and steps taken to subject them to sputum smear microscopy for AFB, for diagnosis of TB. Two sputum specimens are collected over one, or two consecutive days. Of the two sputum specimens, one is collected on the spot and the other is an early morning specimen collected at home by the patient. Sputum smear examination is done by Ziehl-Nielson or auramine, rohdamine (fluorescent) staining which is the simplest test to perform.[5]

Slide reporting

The number of bacilli seen in a smear reflects disease severity and patient infertility. Therefore it is important to record the number of bacilli seen on each smear.[5]

Number of bacilli	Microscopy	Result reported
No	AFB per 100 oil immersion fields	0
1 – 9	AFB per 100 oil immersion fields	Scanty
10 – 99	AFB per 100 oil immersion fields	+ (1+)
1 – 10	AFB per oil immersion field	++ (2+)
> 10	AFB per oil immersion field	+++ (3+)

Categorizing the patients as smear positive or negative require results from more than one smear

Smear positive	Intermediate	Smear negative
Aleast 2 smears examined and both positive i.e. reported 1 – 9 per 100 fields, scanty or greater	Several possibilities a. Only one smear examined (whatever the grading) b. Three smear examined but only one reported In either of these situations either further sputum smear or chest X-ray are required before a patient can be classified	Atleast two smears reported negative

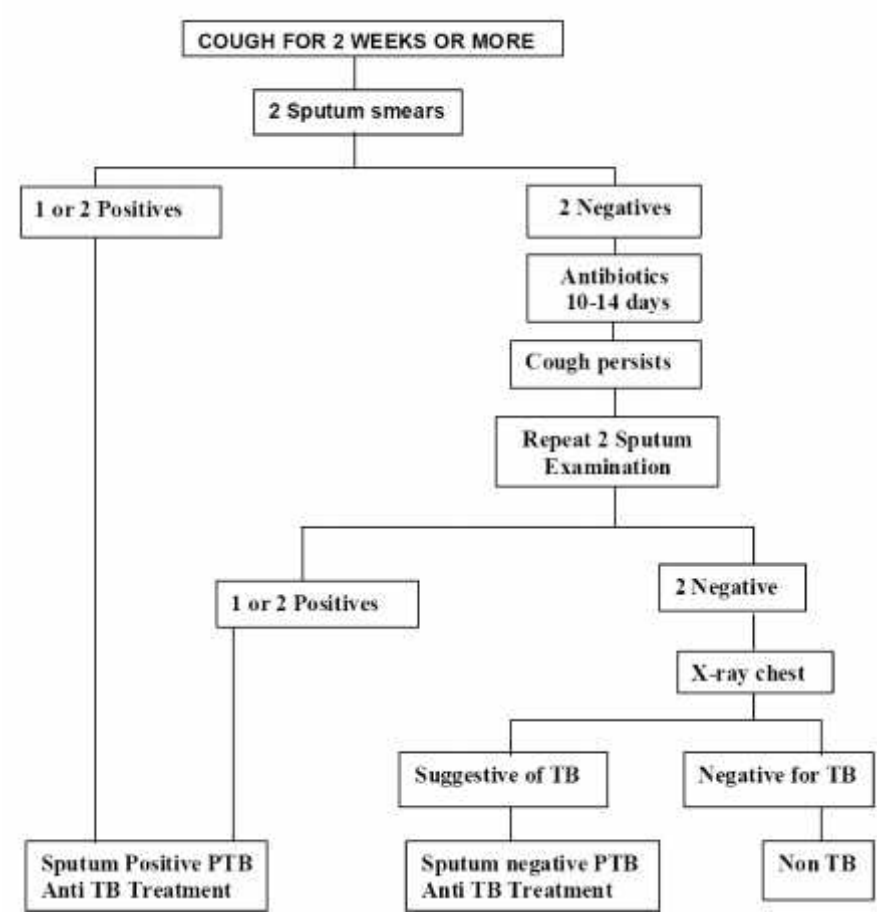


Fig. 1 Diagnostic algorithm for pulmonary TB

Culture

Culture (on Lowenstein-Jensen medium) is much more sensitive but takes six to eight weeks and requires specialized laboratory facilities and trained staff. Automated liquid culture systems that detect bacterial carbon dioxide production or oxygen consumption with radiometric (BACTEC), fluorescent (MGIT), colorimetric (MB/BacT system) or pressure sensors (ESP culture system II) roughly halve the time to detection and are being evaluated for use in resource-poor settings.[61]

Molecular assay

Molecular assays have been developed for rapid detection of growth as well as species identification and drug susceptibility testing. The three most widely used assays, PCR (Roche Diagnostics), transcription mediated amplification (Genprobe) and strand displacement amplification (Becton Dickinson) have shown excellent sensitivity, specificity and speed.

Chest radiography

While radiography is a commonly performed investigation, it must be remembered that patients with HIV co-infection may not have typical radiographic features of PTB. While patients with higher CD4 cells (>350 cells/mm^3) have radiographic abnormalities similar to their HIV negative counterparts, patients with immunosuppression often have minimal or atypical findings.[62]

Diffuse pulmonary infiltrates/opacities are the dominant radiological presentation and cavitation is uncommon in HIV patients. Miliary pattern, mediastinal adenopathy and pleural effusion are more common and x-rays may be normal in 5 to 10% of HIV positive individuals.[63] Differential diagnoses include PCP, non-tuberculous mycobacteria, nocardiosis, fungal infections and non-infective complications like lymphocytic interstitial pneumonitis.

The table below shows so-called "classical" and "atypical" CXR patterns. The classical pattern is more common in HIV-negative patients, and the atypical pattern in HIV-positive patients.

Classical pattern	Atypical pattern
Upper lobe infiltrates	Interstitial infiltrates (especially lower zones)
Bilateral infiltrates	Intrathoracic lymphadenopathy
Cavitation	No cavitation
Pulmonary fibrosis and shrinkage calcification	No abnormalities

Tuberculin skin test (Mantoux test)

A positive tuberculin skin test can detect an immunological memory to previous or ongoing contact with MTB antigens. However, in HIV-1 infected patients with CD4+ T-cell counts of less than 200 cells/mm^3, the tuberculin skin test is usually non-reactive.[64] False positive results may be found in patients who were BCG-vaccinated or who had contact with non-tuberculous mycobacteria. The test should only be performed intradermally according to the method described by Mendel and Mantoux. The standardized dose that is recommended

by the WHO and the IUATLD is 2 TU in 0.1 ml of PPD RT 23/Tween 80. In the United States and some other countries, 5 TU PPD-S is used, which is thought to be similar in strength, is in use. Following the intradermal inoculation of the injection, the diameter of the induration along the short axis of the lower arm is measured by the ball-point technique.[65] In HIV-infected patients, an induration of > 5 mm is positive.108 In TB high prevalence countries like India, tuberculin skin testing has no value in TB diagnosis, as a positive test indicates prior infection only and false negativity (due to anergy or other unknown factors) is common. In the late stages of HIV, the tuberculin test may be negative because of anergy but anergy testing with a panel of antigens (tetanus, mumps, Candida) is not recommended as the results are not reproducible.

Induced sputum/gastric lavage

Inhalation of nebulised hypertonic saline or salbutamol 2 mg given three times daily for a week are used in order to induce sputum production. Gastric lavage can be performed in patients unable to expectorate. The acidic gastric aspirate should be buffered in phosphate solution prior to transportation to the laboratory. These specimens are tested for AFB by smear and culture.[5]

HIV Testing

HIV infection is usually diagnosed through detection of antibodies to the virus. Production of these antibodies usually begins three to eight weeks after infection. The period following infection but before antibodies become detectable is known as the “window period”. Diagnosis of HIV infection is also possible through detection of the virus (p24 antigen, nucleic-acid based tests or culture).[5]

HIV antibody tests

The most widely available way of identifying HIV-infected individuals is the detection of HIV antibodies in serum or plasma samples. The table below shows the two main methods of testing for HIV antibodies.[5]

The enzyme immunoassay (EIA)

Enzyme immunoassays are probably the most efficient tests for testing large numbers of samples per day, as in large blood banks or for surveillance studies.[5]

Simple/rapid tests

Several antibody tests can equal the performance of EIA and do not need special equipment or highly trained staff. These tests are considered rapid if they take less than 10 minutes and simple if they take longer.[5]

There are four types of assay: agglutination, comb/dipstick, flow through membrane and lateral flow membrane. In most formats, the appearance of a clearly visible dot or line indicates a positive result. Many of the tests have an internal control sample, which validates each test run.[5]

Strategy for HIV antibody testing in TB patients

In general, WHO recommends different HIV testing strategies, depending on the objective of testing. The aim is to maximize accuracy and minimize cost. The table below shows the strategy appropriate for each objective of testing.[5]

Objectives, strategies and interpretation of HIV tests[5]

Objective	Testing strategy	Interpretation of result
Individual patients management	Test sample with EIA or simple / rapid assay	1st Assay negative = patient HIV negative or test to be repeated 1st assay positive + 2nd assay positive = patient HIV positive assay positive + 2nd assay negative = repeat both assays Result remain discordant – repeat sample and testing
Surveillance (in population with HIV prevalence > 10%)	Test sample with EIA or simple / rapid assay	Assay negative = patient HIV negative Assay positive = patient HIV positive

Flexible fibreoptic bronchoscopy with lavage (BAL) and transbronchial Biopsy

Bronchoscopy is usually indicated if the suspicion of TB remains high, but no AFBs are found on microscopic examination of sputum. Bronchial secretions or bronchoalveolar lavage is not superior to sputum in the diagnosis of TB in patients with HIV infection[66] but bronchoscopy may be very helpful in differentiating between TB and other diseases in the differential diagnosis,[67] particularly since the coincidence of more than one pulmonary process has been seen in patients with HIV infection.

Total lymphocyte and CD4 counts

The percentage and absolute number of CD4 cells in blood provide a good index of the stage of HIV disease and have a prognostic value, especially when considered with the extent of viral load. Tuberculosis also produces a decline in CD4+ T cells, thereby worsening the immunologic status. Further, at very low CD4+ cell counts, the manifestations of TB may be atypical and a high index of suspicion is required for making a diagnosis. Hence, the CD4 count, if available, is useful in assessing the prognosis as well as for recommending anti-retroviral treatment (ART).

The absolute lymphocyte count has been found to correlate broadly with the CD4 count and has been recommended as a cheaper alternative to CD4 testing. A total lymphocyte count of <1500 cells/mm^3 may indicate a CD4 count of < 200 cells/mm^3.

The following newer diagnostic techniques are also available:

PCR (Polymerase chain reaction): This is a rapid and sensitive technique to detect the nucleic acid of tubercle bacilli in clinical specimens. This is especially helpful in differentiating the species, when AFB are found on microscopic analysis. In this setting, a positive MTB-PCR is more than 95% sensitive for the diagnosis of TB. Unfortunately, the sensitivity of the MTB-PCR is decreased to around 40-77% in smear-negative sputum samples.[68]

In EPTB where acid fast stains often remain negative, or when rapid diagnosis is needed, for example in TBM, MTB-PCR should be performed in the initial routine evaluation. For PCR analysis, biopsies should not be fixed in formalin but rather be preserved in "HOPE" (Hepes-glutamic acid buffer-mediated organic solvent protection effect) media.[69] Because the use of

mycobacterial PCRs in the setting of acid-fast negative stains can lead to false results, these results should always be questioned. However, this technique is still under evaluation and its role in the clinical management of HIV-TB has not yet been finally determined. Further, the commonly used DNA probe 1S6 110 has a lower sensitivity for south Indian strains because of the lower copy number of insertion sequence. However, a combination of primers may improve the sensitivity of this assay.

Restriction-Fragment-Length Polymorphism Analysis: The restriction fragment-length polymorphism (RFLP) analysis of MTB isolates allows identification of specific TB strains and can document the transmission of disease between patients. Although it was previously thought that 90% of cases of TB in the United States resulted from reactivation of infections acquired in the remote past, RFLP analyses show that recent infection accounts for up to half the cases of TB among both HIV-infected and HIV negative patients in urban areas. RFLP analysis is used most frequently to confirm that clusters of cases of TB are linked by recent transmission.

However RFLP analysis can also be helpful in making clinical decisions about individual HIV -infected patients with TB. When a single specimen with negative results on acid-fast staining yields MTB but the clinical findings in the patient are not consistent with the presence of TB the possibility of a false positive result from cross-contamination with another patients sample can be evaluated by comparing the RFLP patterns of the two isolates. Identical RFLP patterns strongly suggest cross-contamination, allowing the discontinuation of potentially toxic anti TB medications. RFLP analysis is also useful when two MTB isolates from a patient differ in drug susceptibility.

In the past years, new diagnostic tools for the diagnosis of infection with MTB have been developed. The ELISPOT (T-SPOT-TB Test) and ELISA (Quantiferon- Gold-in tube Test) detect the secretion of gamma-interferon by mononuclear cells in venous blood, specific for MTB peptides, ESAT-6 and CFP-I0. These tests are more sensitive and specific for the diagnosis of MTB infection and are superior to the T-SPOT-TB Test in patients with Immunosuppression.[70,71,72] However, in patients with advanced immunosuppression, a substantial proportion of ELISA results are indeterminate and the performance of these assays in patients with HIV infection and low CD4+ T-cell counts still needs to be evaluated in clinical practice.

The diagnosis of TB in patients with HIV is more difficult for the following reasons:

(a) Sensitivity and reliability of tuberculin test gets reduced since HIV infection causes depression of cell-mediated immunity. Only 30% to 50% of co-infected patients have a positive result. Therefore, full diagnostic evaluation should be undertaken in all patients who have clinical features compatible with TB. There are recent reports of restoration of cell-mediated immunity and delayed hypersensitivity including tuberculin test in HIV infected patients on highly active anti-retroviral therapy (HAART) within the first month of therapy.

(b) In HIV infected patients with PTB sputum culture is positive for AFB in about 90% of cases and by smear in about 50% to 70%, similar to results seen in immunocompetent adults with reactivation TB. Polymerase chain reaction and gene probes for rapid identification of MTB in sputum smears are more sensitive than traditional staining methods but are not as

sensitive as culture. However, the sensitivity of culture approaches 90%, depending on the number of samples tested.

(c) Chest X-ray abnormalities are even more nonspecific in HIV infected patients than in HIV negative patients, which may result in underdiagnosis. Radiological patterns depend on the level of immunity in the host. 'Typical pulmonary lesions are seen only in about one third of the HIV infected patients with clinical TB.

TREATMENT OF PTB AND TB-HIV COINFECTION

Tuberculosis is one of the commonest opportunistic infection (OI) and is a leading cause of death amongst HIV infected patient in developing countries. Management of HIV and TB co-infection is complicated because of drug-drug interactions, overlapping toxicities, additional pill burden and development of immune reconstitution inflammatory syndrome (IRIS).[73]

Tuberculosis

All HIV/TB patients should be treated with standard four drug anti-TB combinations as per TB treatment guidelines. Current guidelines recommend that irrespective of HIV status, TB requires a minimum of six months of treatment with four drugs (including rifampin) in the intensive phase and two drugs in the continuation phase. Treatment consists of isoniazid (INH), Rifampin (R), Ethambutol (E) and Pyrazinamide (Z) for two months followed by INH and R for four months, given either daily or intermittently.

In India, under the RNTCP, patients with newly diagnosed TB receive a 6-month thrice-weekly regimen (Cat I – 2EHRZ3/4RH3) while those with

relapse, default or failure receive an 8-month regimen (Cat II – 2SEHRZ3/1EHRZ3/5EHR3).

Patients with smear negative pulmonary TB and localized extrapulmonary forms are treated with Cat III - 2HRZ3/ 4RH3, however this regimen should be avoided in persons with HIV infection.

Antiretroviral therapy (ART)

Using ART in patients with TB and advanced HIV disease has been shown to reduce mortality and the risk of development of other OI's.[74] Currently available antiretroviral agents fall in the following groups of drugs:

- Nucleoside reverse transcriptase inhibitors (NRTIs).
- Non-nucleoside reverse transcriptase inhibitor (NNRTI's).
- Protease inhibitors (PI).
- Integrase inhibitors,
- Chemokine receptor antagonists and Entry (fusion) inhibitors.

Antiretrovirals approved for use

NRTI	NNRTI	PI	Entry inhibitor	Intergrase inhibitor
Zidovudine	Nevirapine	Saquinavir	Enfuvirtide	Raltegravir
Stavudine	Efavirenz	Indinavir	Maraviroc	
Lamivudine	Delavairidine	Ritonavir		
Didanosine	Etravirine	Lopinavir		
Zalcitabine		Atazanavir		
Abacavir		Amprenavir		
Emtricitabine				
Tenofovir				

HAART refers to the use of a combination of at least three ART drugs to form a maximally suppressive regimen. Regimens recommended for use in India for patients with concomitant TB are a combination of two NRTI's with EFV or less commonly NFV.

The NRTI combinations used commonly are Zidovudine with Lamivudine, Stavudine with Lamivudine, Tenofovir with Lamivudine, and rarely Abacavir with Lamivudine or Didanosine with Lamivudine (only the first two combinations are available in the national program). Among the PI's, nelfinavir, saquinavir, lopinavir and ritonavir can be used but additive hepatoxicity may occur with anti-TB drugs requiring close monitoring of liver function.

Fixed dose combinations available in India

Two drugs	Three drugs
Stavudine + Lamivudine	Stavudine + Lamivudine + Nevirapine
Zidovudine + Lamivudine	Zidovudine + Lamivudine + Nevirapine
Tinofovir + Emtricitabine	Tinofovir + Emtricitabine + Efavirenz

CD4 cell count is important in deciding when to start ART in HIV/TB co-infected patients.

1. HIV/TB with CD4 cells > 350/mm^3: ART is delayed till the completion of TB treatment.

2. Patients with CD4 cells ≤ 350/mm^3: Delaying ART can result in HIV-related morbidity and even mortality due to risk of occurrence of other OI's. In such situations ART should be initiated as soon as anti-TB medicines are tolerated and patient has shown clinical improvement (usually two to four weeks of initiation of ATT).

One should closely monitor for the development of IRIS in patients initiating ART with baseline CD4<50/mm^3.

Combination antiretroviral regimen

There are limited options available for ART in HIV/TB co-infected patients. Rifampicin, a critical component of antituberculous therapy interacts with PI's and NNRTIs and reduces exposure of PI's by 75 to 95% and NNRTIs nevirapine by 20 to 55% and efavirenz up to 20%.[75,76] Sub-optimal exposure to these ARVs may lead to development of drug resistance. Unfortunately, of all available PI's and NNRTIs, rifampicin can be concomitantly used only with full dose ritonavir or with efavirenz.[77]

Though some experts recommend increasing the dose of EFV to 800 mg when using with rifampicin, studies in developing countries have documented comparable antiretroviral effectiveness with a dose of 600 mg.[78,79]

Ritonavir boosted saquinavir should not be used with rifampicin due to significant elevation (up to 20 x upper limit of normal) of serum transaminases in a Phase I study evaluating the pharmacokinetic interaction of this drug combination in healthy volunteers.[80]

Therapeutic strategies for concomitant use of ART and ATT include:

1. Using Efavirenz + 2NRTIs: Efavirenz based ART when used at standard dosages in HIV/TB patient-receiving rifampicin has demonstrated good clinical, immunological and virological outcomes.[78,81] Although EFV is more expensive than NVP, it should be used at least until the duration of TB therapy. After the completion of ATT, EFV may be substituted back to NVP in order to make the regimen less expensive. However, before the

substitution it is necessary to document good virologic control of the EFV based regimen (PVL<400 copies/ml). The hepatic induction effect continues for up to 2 weeks after discontinuation of rifampicin. The substitution of EFV with NVP should be made after 2 weeks of rifampicin discontinuation. A lead in dose is not necessary when NVP is substituted for EFV in this situation. An additional concern is the risk of severe hepatitis in patients who are switched from EFV to NVP at higher CD4 counts, close monitoring is warranted.

2. Rifabutin has less pronounced interaction with PI's, and it is currently available in India. If rifabutin is used, the dose should be reduced with PI's to avoid ocular and other toxicities. With PI/r recommended dose of rifabutin is 150 mg every alternate day or thrice weekly.[82]

3. Though nevirapine concentrations are affected by rifampicin, several cohort studies have demonstrated high rates of virologic suppression when both these drugs were concomitantly used.[83,84] Nevirapine can be used as an alternative if there is absolute contraindication for use of efavirenz or the patient cannot afford rifabutin. Careful monitoring for hepato-toxicity is recommended in these situations.

4. When TB develops in patients already receiving ART, the regimen should be modified to EFV based to make it compatible with TB treatment. Following the completion of antituberculous therapy the EFV based regimen can be continued or changed in accordance with the clinical and immunological status of the patient. Anti TB treatment without rifampicin

in HIV/TB co-infected patients is discouraged due to a significantly lower cure rate and higher incidence of TB relapses.[85]

Tuberculosis in patients on antiretroviral treatment

In patients that develop active TB within six months of initiating first line ART, one has to consider modification of treatment and the possibility of ART failure. Although ART decreases the incidence of TB by at least 80%, the risk of developing TB is still higher than in the HIV negative population. Previously undiagnosed TB may present within the first six months as part of IRIS. If TB occurs during the first six months following the initiation of ART, this should not be considered as a treatment failure and the ART regimen has to be adjusted for co-administration of a rifampicin containing TB regimen. If TB develops more than six months after the initiation of ART, the decision as to whether the TB diagnosis represents ART failure depends on the CD4 count and viral load if available or whether the TB is pulmonary or extra pulmonary, or whether there are other non-TB clinical stage 3 or stage 4 events. The development of TB after six months of ART initiation without other clinical and immunological evidence of disease progression should not be regarded as representing ART failure. However, extra pulmonary TB should be considered as indicating treatment failure.

Second line antiretroviral therapy for patients with tuberculosis

There are significant drug interactions with PIs and rifampicin. Unboosted PIs cannot be used with rifampicin containing regimens because PI levels are sub-therapeutic therefore boosted PIs (Lopinavir 400 mg / ritonavir 400 mg twice

daily or SQV 400 mg / RTV 400 mg can be considered but with close laboratory monitoring for hepatotoxicity (RTV 400 mg is quite hepatotoxic). Rifabutin if available, maybe used in place of rifampicin but is contraindicated in patients with WBC counts below 1000/mm^3. We should avoid use of any of the current PIs with rifampicin.

Immune Reconstitution Inflammatory Syndrome

Antiretroviral therapy partially restores immune defects caused by chronic HIV infection. This typically includes restoration of protective pathogen-specific immune responses. This has resulted in a sharp decline in the incidence of opportunistic infections in HIV patients.[86]

However, suppression of HIV viraemia by ART is accompanied by atypical OI manifestations or other inflammatory diseases in some patients. In these situations restoration of an immune response following ART is immunopathological rather than protective. These conditions are therefore labeled as IRIS. Other names for IRIS include Immune Restoration Disease and paradoxical reactions.

The IRIS is defined as a new occurrence or worsening of existing clinical conditions and/or laboratory parameters despite a favorable outcome in HIV surrogate markers (CD4 counts and PVL).[87]

Patients may present with painful and swollen lymph nodes, chest Symptoms (Progressive pneumonitis or the development of organizing

pneumonia after treatment for PTB or PCP), Progression of organ dysfunction or enlargement of pre-existing lesions and unexplained fevers among others.

The IRIS occurs when the immune response against a particular antigen increases after the start of ART, leading to an inflammatory reaction. Initiation of ART can also unmask previously undiagnosed infections by improving the inflammatory response due to the repair of the immune system.

The IRIS events may occur in up to 40% of patients treated for TB who start ART and up to 5% in those with cryptococcal disease. The risk is higher in those with advanced HIV disease with low CD4 counts. IRIS events often occur between two to eight weeks of ART initiation and less commonly after many months of ART.

The diagnosis of IRIS should be considered by ART providers when a patient who has recently started ART (last three months) develops new symptoms when they should be getting better. This is particularly the case in patients with a known co-infection such as TB or cryptococcal meningitis who seemed to be responding well and adhering to treatment but then deteriorate within weeks after starting ART.

The TB IRIS presents with worsening clinical symptoms after initial improvement and may occur in up to 40% of persons with TB who initiate ART. Patients with PTB may develop worse chest symptoms, new infiltrates on chest film, and enlarged lymph nodes that may become tender or form abscesses. Tuberculosis meningitis and/or tuberculomas may present with confusion, fits

and/or new focal neurological features. Abdominal TB may present with intestinal obstruction or even bowel perforation.

The TB IRIS is more common if ART is started early in the course of TB treatment and in patients with low CD4 counts. Most cases resolve without any intervention and ART can be safely continued. However, serious reactions like tracheal compression from massive lymphadenopathy or respiratory difficulty may require use of corticosteroids.

Treatment of IRIS

There are no standard guidelines for treatment of IRIS. There is very limited information on the effectiveness of various interventions to manage IRIS, with lack of evidence from randomized clinical trials. Most cases will resolve without any additional treatment. Milder forms of IRIS resolve with continuing anti-infective therapy and ART.

Non-steroidal anti-inflammatory drugs (NSAIDS) may be helpful in controlling inflammation and fever associated with IRIS.[87] However, in severe IRIS a course of oral prednisolone is required to alleviate symptoms. The dose and duration required is very variable and should be judged clinically. Severe disease will require at least 1 to 2 mg/kg of prednisolone. Thalidomide has also been tried effectively in some patients.

PREVENTIVE THERAPY FOR TB IN HIV INFECTED INDIVIDUALS

As people with HIV are at a great risk of developing TB, preventing the onset of TB in them is clearly a priority because in co-infected people it may play

a critical role in limiting a possible increase in the number of cases of symptomatic TB.[81,88] TB in HIV infected people is predominantly caused by endogenous reactivation of dormant foci with a higher frequency than in the general population, which can be prevented by chemotherapy.

Several randomized clinical trials in HIV-infected persons have shown that the incidence of TB can be reduced by 40-60% by preventive therapy.[89] Prior to the initiation of preventive therapy for TB it is essential to rule out active TB. The optimal duration of preventive therapy with single drug isoniazid, daily or twice weekly, should be greater than six months to provide the maximum degree of protection against TB. The effectiveness of preventive therapy should be evaluated at regular intervals by monitoring patients. Current recommendations for preventive therapy against TB in HIV infected persons

1. WHO recommendation: Isoniazid is the recommended drug (5 mg/kg - maximum 300 mg) as daily, self-administered therapy for six months. Individuals should be seen monthly and given only one month's supply of medication at each visit.

2. CDC recommendation: Isoniazid is chosen for prevention of TB in persons with HIV infection, nine months is recommended rather than 6 months41. Rifampicin and pyrazinamide may be offered daily for two months to contacts of patients with INH resistant, rifampicin susceptible TB.

3. American Thoracic Society: Isoniazid is recommended for 12 months as prophylaxis in person infected with HIV infection.[14,90,91]

It is advisable that people coming into close contact with HIV positive TB patients should be screened for TB and given DOTS, if found to be suffering from TB. If contacts are healthy children aged 0-5 years, Chemoprophylaxis with INH should be given for six months, followed by BCG vaccination if they are Mantoux negative or the PPD test cannot be done because BCG vaccine is effective in preventing the disseminated forms of TB in children, namely, TBM and miliary TB. The efficacy of BCG against adult forms of TB or exogenous re-infection disease is debatable. However, BCG vaccine is not safe in individuals with AIDS because of the risk of disseminated BCG disease. WHO and UNICEF, therefore, recommend that asymptomatic HIV infected children living in areas where the risk of TB is high should be BCG vaccinated at birth or as soon as possible; thereafter, BCG vaccine may be given in accordance with standard childhood immunization policies but withheld in infants with symptomatic HIV infection.

The TB preventive therapy should form part of the care and support package for HIV positive persons that includes co-trimoxazole prophylaxis, nutritional counseling and supplementation and ART.

OUTCOME

The mortality of HIV-infected patients with TB is comparatively higher than that of HIV-negative TB patients.[92-95] The mortality depends upon the type of disease and the degree of underlying immunosuppression. In HIV-infected patients with TB meningitis, mortality is about 60 to 70%, despite adequate treatment.[92,93] However, with adequate antituberculosis therapy, occurrence of

TB has been found to have no independent effect on mortality in hospitalised HIV infected patients.[96] Other OIs which often go undiagnosed are a common cause of death in patients with HIV-TB, especially those dying later during antituberculosis treatment.[97] In a study from south India, the median survival in HIV infected patients with PTB and EPTB was found to be 45 and 40 months, respectively.[98] Earlier studies from Uganda and Europe have documented a median survival of 22 to 24 months in HIV infected patients with TB.[99,100]

METHODOLOGY

The present study was conducted in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum on patients with sputum positive PTB during the period of January 2009 to December 2009.

Study design

The study design was one year cross sectional study.

Study period and duration

The present one year study was conducted during the period of January 2009 to December 2009.

Method of collection of data

Source of Data

Patients admitted with sputum positive PTB to KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum over a period of one year.

Sample size

All newly diagnosed patients with sputum positive PTB admitted at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during study period were selected for the study.

Selection criteria

Inclusion Criteria

- Patients diagnosed to have sputum positive PTB proved by laboratory reports that is;
    - Two or more initial sputum smear examinations positive for AFB or
    - One sputum smear positive for AFB, and CXR abnormalities consistent with active PTB as determined by a clinician or
    - One sputum smear positive for AFB, which is also culture positive for MTB.

Exclusion Criteria

- Chronic obstructive pulmonary disease (COPD).
- Bronchial asthma with secondary infection.
- Non tubercular Pneumonia and Bronchiectasis.
- Patients of PTB presently on anti koch's treatment (AKT) of more than two weeks duration.

Procedure

The study was approved by the Ethical and Research Committee of Ethics Committee, Jawaharlal Nehru Medical College, Belgaum. Patients admitted in the wards of Medicine and Respiratory Department at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum were evaluated based on selection criteria. The patients were selected by detailed medical history, physical examination and diagnosis of pulmonary tuberculosis according to RNTCP

guidelines. The selected patients were briefed about the nature of the study, the interventions used and a written informed consent was obtained (Annexure–I).

Demographic data like gender and age were collected along with relevant history and recorded on predesigned and pretested proforma (Annexure-II). A thorough clinical examination was conducted and the findings were also recorded. Routine investigations such as blood group, haemogram that is, haemoglobin, total count, differential count, erythrocyte sedimentation rate were done.

Sputum smears for AFB was done and were graded according RNTCP guidelines as grade 0 with NO AFB per 100 oil immersion fields, scanty with 1 to 9 AFB per 100 oil immersion fields, grade + with 10 to 99 AFB per 100 oil immersion fields, grade ++ with 1 to 10 AFB per oil immersion field and grade +++ with more than 10 AFB per oil immersion field.

Chest X-ray was done and evaluated for lesions involved like infiltration, cavitation, fibrosis, fibrocavitatory lesions and miliary mottling along with different zones involved. Others tests like serum urea, serum creatinine, serum sodium, serum potassium were carried out. HIV status was confirmed by voluntary counselling and testing centre along with CD4 count.

Statistical analysis

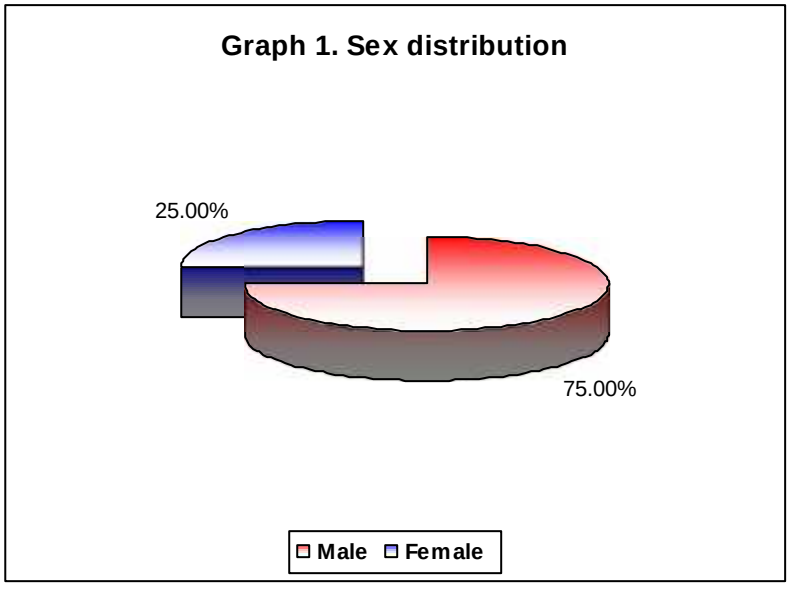
The results were tabulated and the data was analysed using rates, ratios and percentages for different clinical manifestations. The data was compared using chi-square (x^2) test, 'Z' test and student 't' test.

RESULTS

The present study was conducted in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum on 104 patients with sputum positive PTB during the period of January 2009 to December 2009. The HIV ELISA was done in all 104 patients to confirm HIV seropositivity. The data obtained was tabulated and analysed as below.

Table 1. Sex distribution

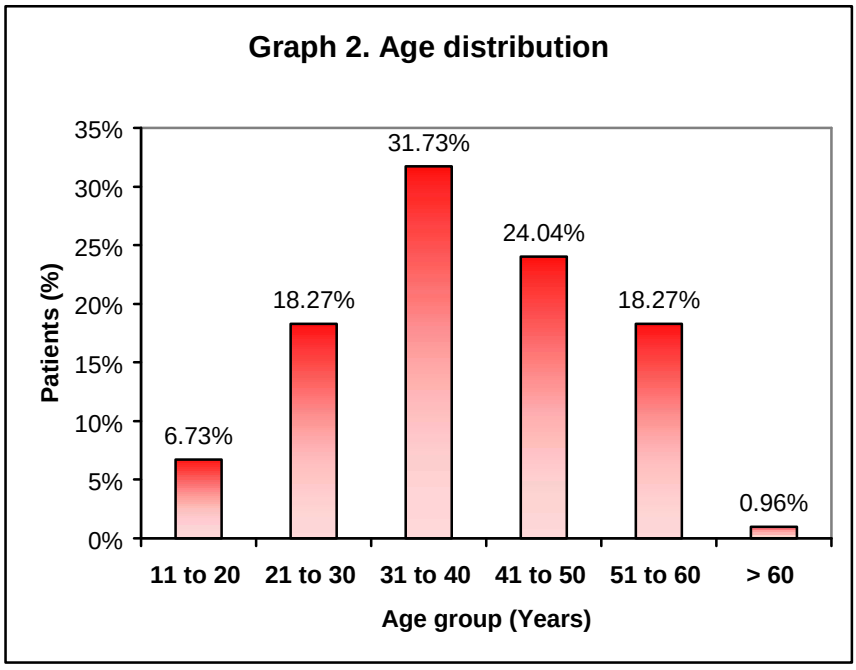
Sex	Patients	
	Number	**Percentage**
Male	78	75.00
Female	26	25.00
Total	**104**	**100**



In the present study, among 104 patients, 78 (75%) were males and 26 (25%) were females with male to female ratio of 3:1.

Table 2. Age distribution

Age (Years)	Patients	
	Number	**Percentage**
11 to 20	7	6.73
21 to 30	19	18.27
31 to 40	33	31.73
41 to 50	25	24.04
51 to 60	19	18.27
> 60	1	0.96
Total	**104**	**100**



In the present study, the age of the patients varied from 11 years to 60 years. Majority (31.33%) of the patients were in the age group 31 to 40 years, followed by 24.04% in 41 to 50 years age group, 18.27% in 21 to 30 years and 51 to 60 years age group, 6.73% in 11 to 20 years age group and 0.96% in patients >60 years age group.

Table 3. Marital status

Marital Status	Patients	
	Number	**Percentage**
Married	90	86.53
Single	14	13.46
Total	**104**	**100**

In the present study, 90 patients (86.53%) were married and 14 (13.46%) were single.

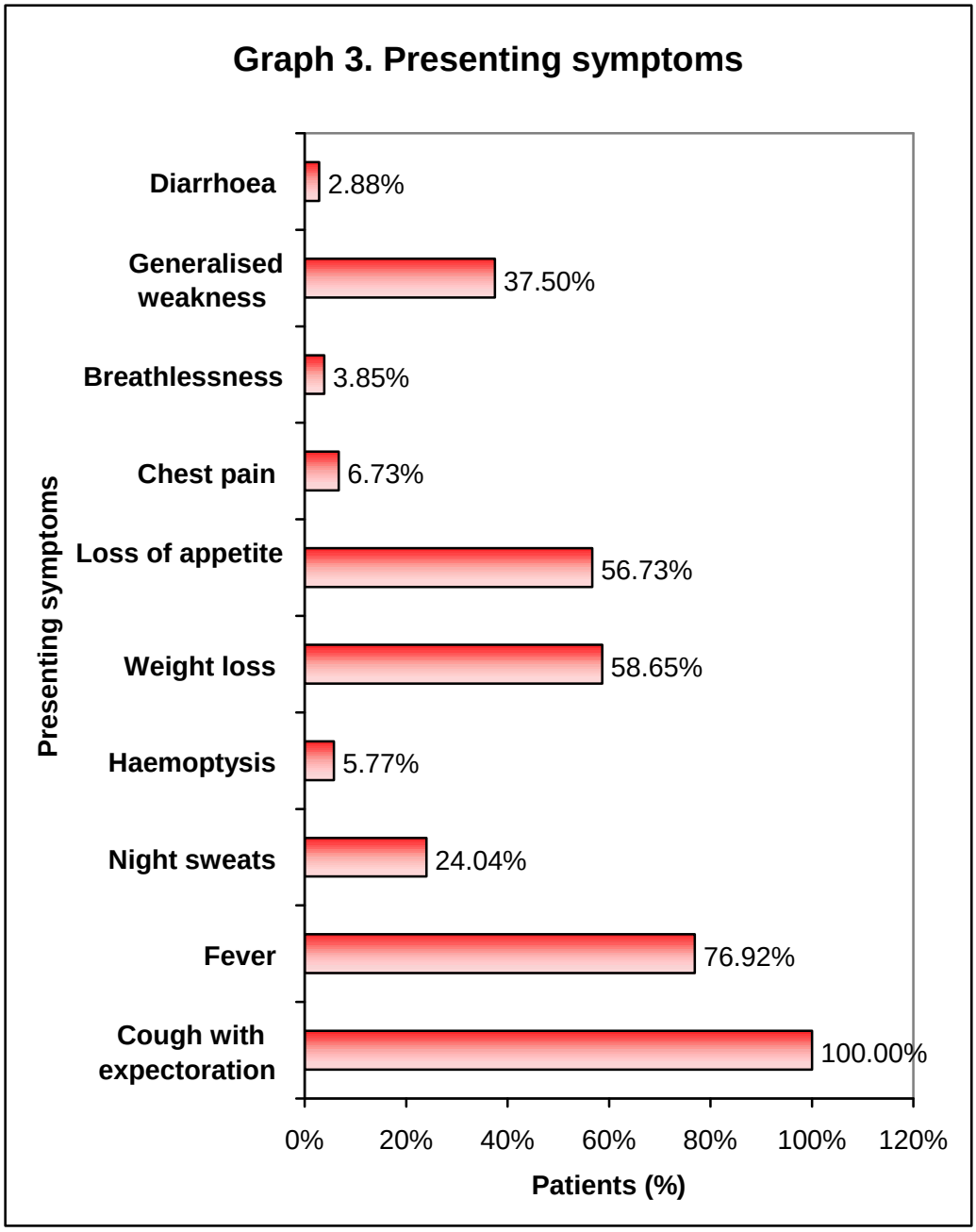
Table 4. Occupation

Occupation	Patients	
	Number	**Percentage**
Agricultural worker	27	25.96
Public servant	10	9.62
Driver	8	7.69
House wife	15	14.42
Labourer	20	19.23
Merchant / Business Owner	7	6.73
Student	17	16.35
Total	**104**	**100**

In the present study, 25.96% of patients were agriculture workers, 19.23% were daily labourers, 16.35% were students 14.42% were house wifes, 9.62% were in service, 7.69% were drivers and 6.73% were merchant / business owners.

Table 5. Presenting symptoms

Symptoms	Patients	
	Number	**Percentage**
Cough with expectoration	104	100.00
Fever	80	76.92
Night sweats	25	24.04
Haemoptysis	6	5.77
Weight loss	61	58.65
Loss of appetite	59	56.73
Chest pain	7	6.73
Breathlessness	4	3.85
Generalised weakness	39	37.50
Diarrhoea	3	2.88



In the present study, among 104 patients, all patients (100%) presented with cough with expectoration, 76.92% of patients had fever, 58.65% had weight loss, 56.73% had history of appetite. 37.50% had generalized weakness and 24.04% of patients had history of night sweats. History of chest pain, breathlessness, haemoptysis and diarrhoea was seen in 7.73%, 3.85%, 5.77% and 2.88% of patients respectively.

Table 6. Past history

Past History	Patients	
	Number	**Percentage**
Known HIV status	8	7.69
Contact with TB	17	16.35
Herpes zoster	13	12.50
Diabetes mellitus	11	10.58

In the present study, 7.69% of patients were known HIV positive, earlier contact with sputum smear positive TB was present in 16.35% of patients. 12.50% of patients had Herpez Zoster and 10.58% of patients were diabetic.

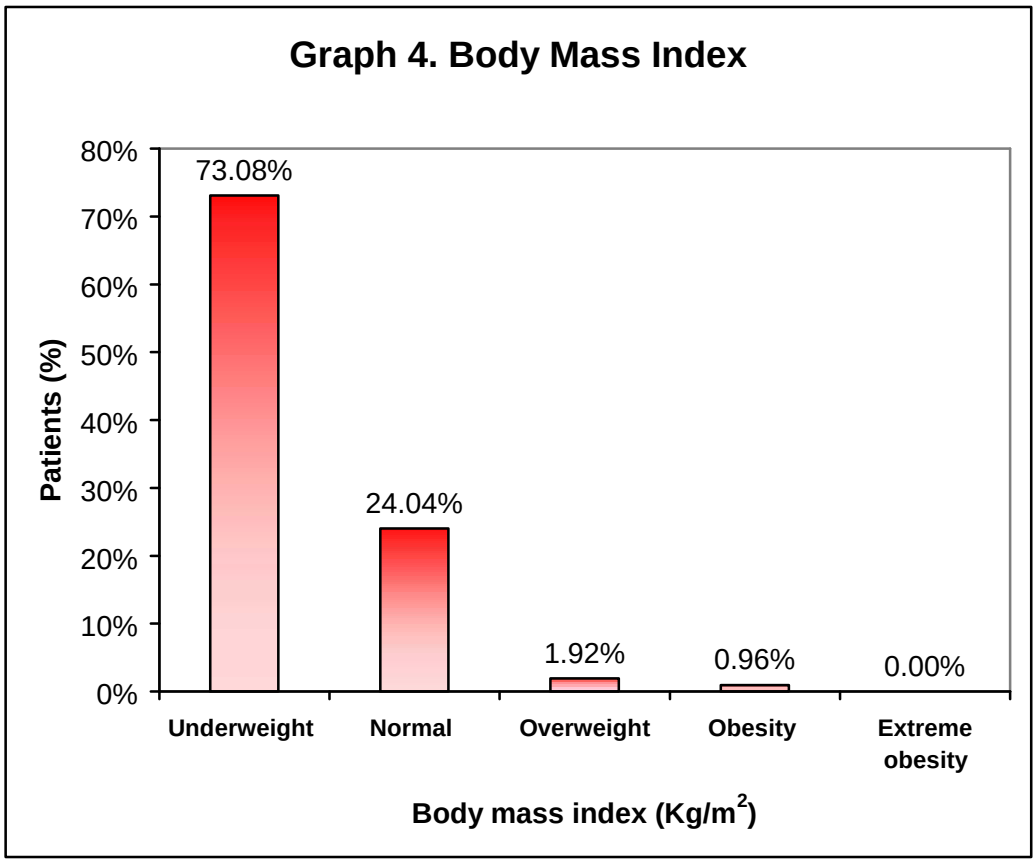
Table 7. Risk factors for HIV

Risk factors	Patients	
	Number	**Percentage**
Multiple sexual partner	11	10.58
History of blood transfusion	6	2.80
Sexually transmitted disease	3	1.40
Intravenous drug abuse	0	0.00

In the present study, among HIV seropositive patients, 10.58% of patients had history of multiple sexual partners, 2.80% of patients had history of blood transfusion and 1.40% of patients (n =3) had sexually transmitted disease.

Table 8. Body Mass Index

BMI (Kg/m^2)	Patients	
	Number	Percentage
Underweight (<18.5)	76	73.08
Normal (18.5 to 24.9)	25	24.04
Overweight (25.0 to 29.9)	2	1.92
Obesity (30 - 39.9)	1	0.96
Extreme obesity (>40)	0	0.00



In the present study, majority (73.08%) of the patients were underweight. 24.04% of patients had normal BMI, 1.92% of patients were over weight and 0.96% of patients were obese.

Table 9. Physical findings

Physical findings	Patients	
	Number	**Percentage**
Pallor	48	46.15
Lymphadenopathy	30	28.85
Oral candidiasis	18	17.31
Herpetic scar	13	12.50
Genital lesions	4	3.85

In the present study on physical examination 46.15% of patients had pallor, 28.85% had lymphadenopahty, 17.31% had oral candidiasis, 12.50% of patients had herpetic scar and genital lesions were present in 3.85% of patients.

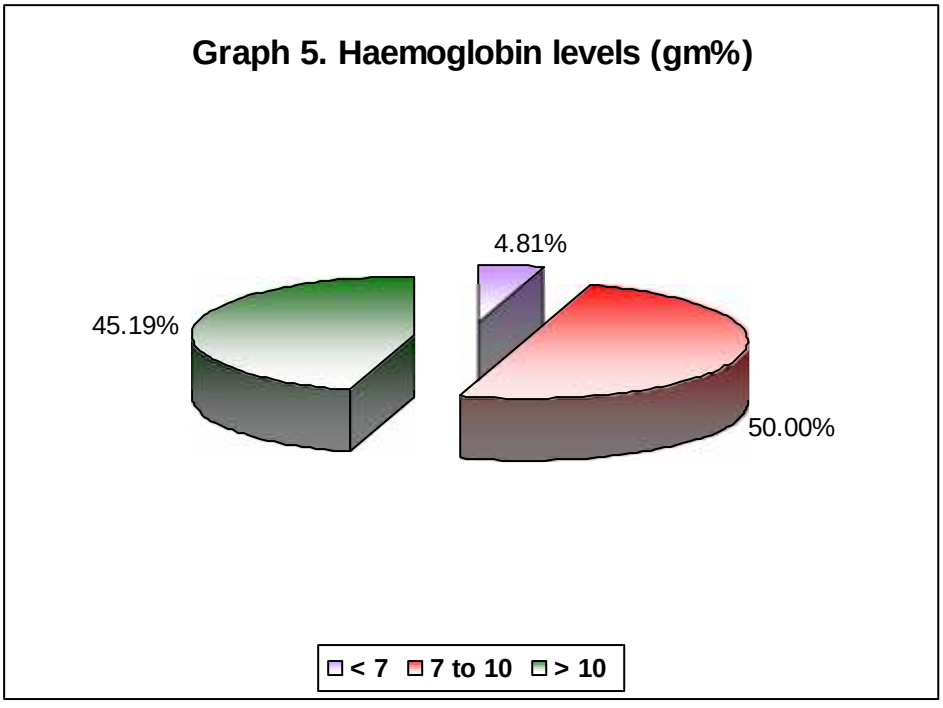
Table 10. Respiratory system examination

Signs	Patients	
	Number	**Percentage**
Cavitation	40	38.46
Fibrosis	90	86.54
Infiltration	11	10.58

In the present study, on systemic examination, 86.54% of patients had signs of fibrosis, 38.46% of patients had signs of cavitations and 10.58% of patients had signs of infiltration.

Table 11. Haematological parameters

Haematological parameters	Range	Number	Percentage
Hb (gm%)	Less than 7	5	4.81
	7 to 10	52	50.00
	More than 10	47	45.19
Total count	Less than 4000	3	2.88
(Cells/mm^3)	4000 to 11000	46	44.23
	More than 11000	55	52.88
ESR (mm)	0 to 20	0	0.00
	More than 20	104	100.00



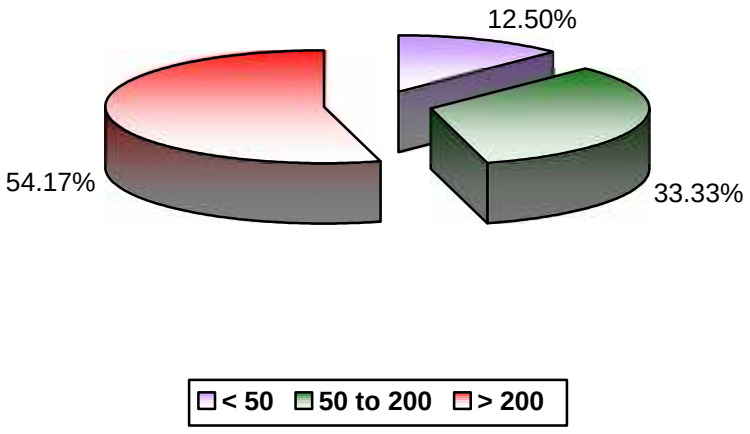
In this study, majority of the patients (50%) had haemoglobin levels between 7 to 10 gm% followed by more than 10 gm% in 45.19%. Haemoglobin levels were less than 7 gm% in 4.81% of patients. In the present study 52.88% of

patients total leucocyte count was more than 11,000, 44.23% of patients had in the range of 4000-11,000 and 2.88% of patients had less than 4000 cells/mm^3. All patients had their ESR raised more than normal (100%).

Table 12. CD4 count among HIV seropositive patients

CD4 Count (Cells/mm^3)	Number	Percentage
Less than 50	3	12.50
50 to 200	8	33.33
More than 200	13	54.17

Graph 6. CD4 count among HIV seropositive patients (cells/mm^3)



In the present study, majority (54.17%) of HIV seropositive patients had CD4 count more than 200 cells/mm^3 followed by 50 to 200 cells/mm^3 (33.33%) and less than 50 cells/mm^3 (12.50%).

Table 13. Biochemical parameters

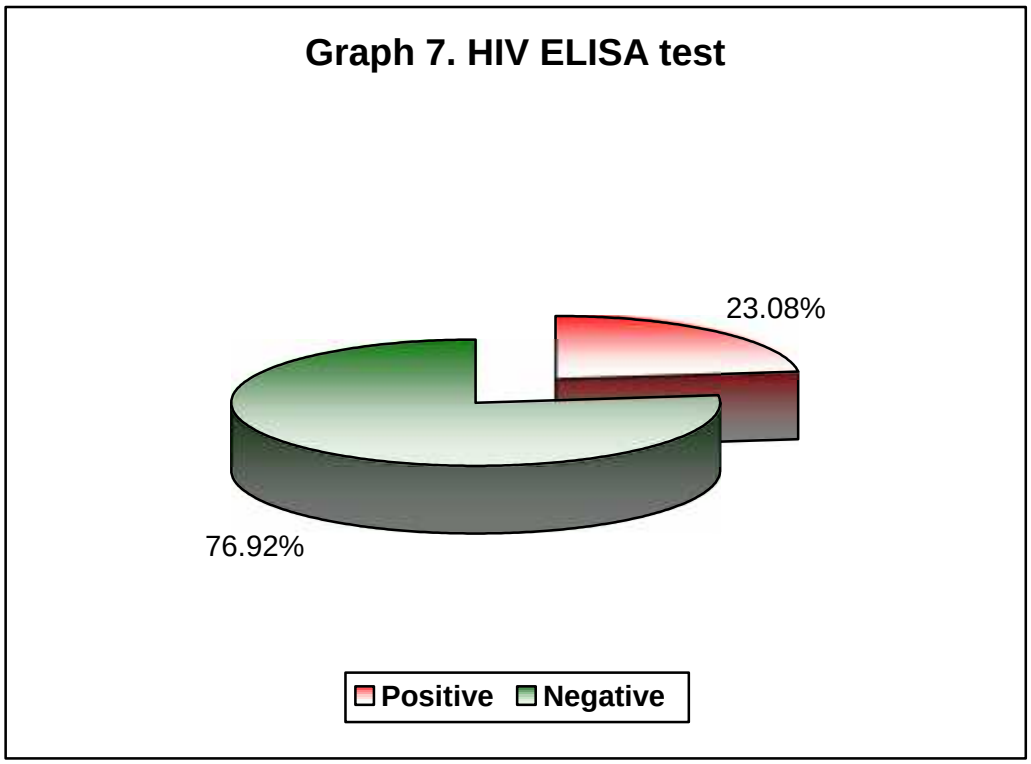
Biochemical parameters	Range	Number	Percentage
Urea (mg/dL)	Less than 10	0	0.00
	10 to 39	78	75.00
	More than 39	26	25.00
Serum creatinine (mg/dL)	Less than 0.5	2	1.92
	0.5 to 1.3	84	80.77
	More than 1.3	18	17.31
Serum sodium (meq/L)	Less than 130	5	4.81
	130 to 145	89	85.58
	More than 145	10	9.62
Serum potassium (meq/L)	Less than 3	5	4.81
	3 to 5	94	90.38
	More than 5	5	4.81

In the present study, 75% of patients blood urea levels were within the normal range and 25% of patients had elevated blood urea levels. 80.77% of patient's sr. creatinine levels were within normal range, 17.31% of patients had elevated sr. creatinine levels and 1.52% of patients serum creatinine levels were less than 0.50 normal range. 85.58% of patients sr. sodium levels were within normal range, 9.62% of patients had elevated sodium levels and 4.81% of patients had low Sr. sodium levels. 90.38% of patients Sr. potassium levels were

within normal range, 4.81% of patients had elevated Sr. potassium levels and 4.81% of patients potassium levels were low.

Table 14. HIV ELISA test

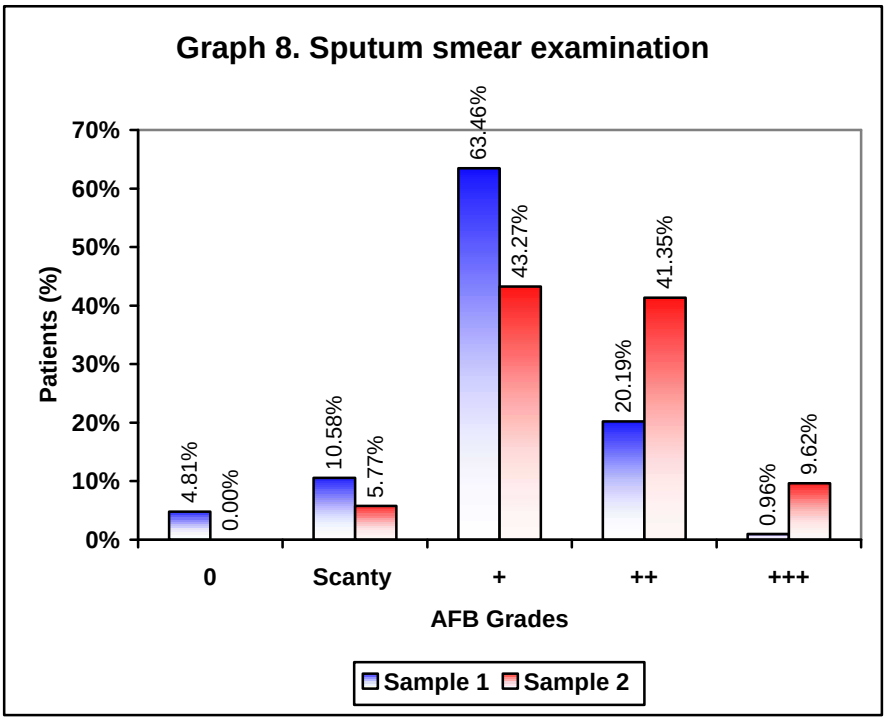
HIV ELISA Test	Number	Percentage
Positive	24	23.08
Negative	80	76.92



In the present study, 76.92% of patients were HIV seronegative and 23.08% of patients were HIV seropositive.

Table 15. Sputum smear examination

AFB Grades	Sample 1		Sample 2	
	Number	Percentage	Number	Percentage
0	5	4.81	0	0.00
Scanty	11	10.58	6	5.77
+	66	63.46	45	43.27
++	21	20.19	43	41.35
+++	1	0.96	10	9.62



In the present study, 63.46% of patients were recorded as grade + (1+), 20.19% as grade ++ (2+), 10.58% as scanty, 4.81% as grade 0 and 0.6% as +++ (3+) on sputum smear examination for AFB. In sample 2, 43.27% of patients were recorded as grade ++ (2+), 9.62% as +++ (3+), 5.77% as scanty on sputum smear examination for AFB.

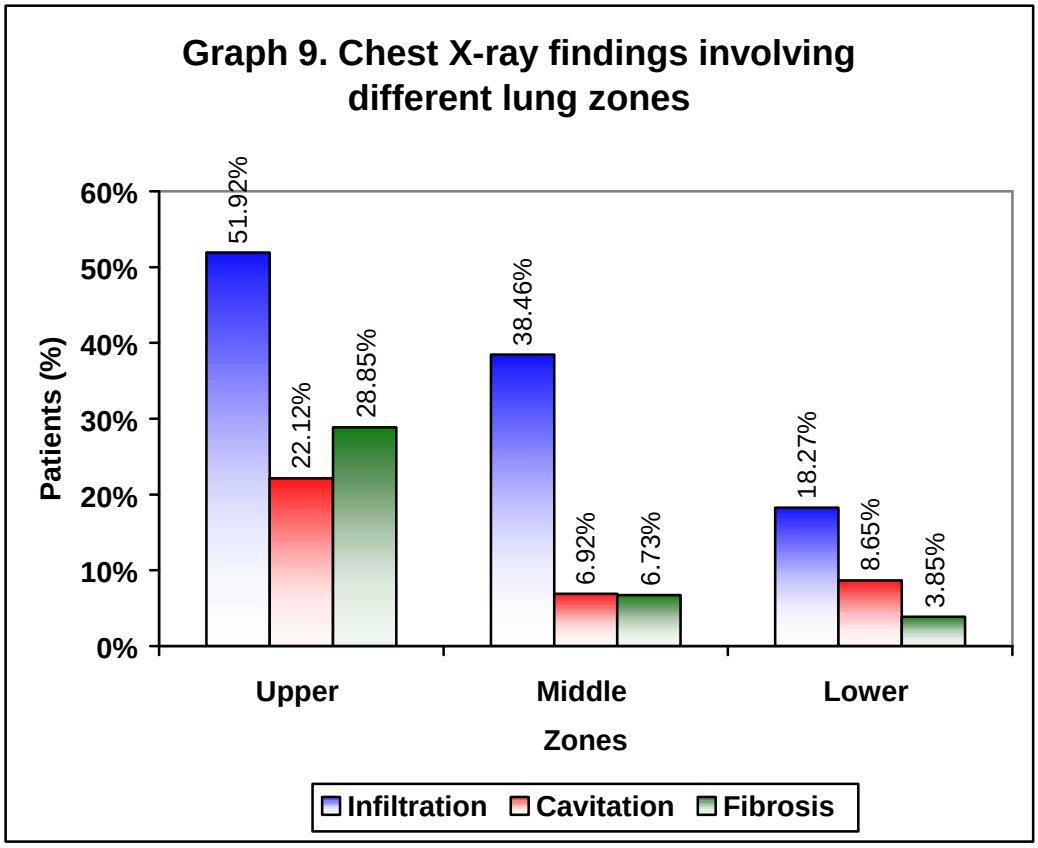
Table 16. Chest X-ray findings

Chest X-ray findings	Patients	
	Number	**Percentage**
Infiltration	70	67.31
Cavitation	39	37.50
Fibrosis	40	38.46
Fibrocavitatory lesion	34	32.69
Miliary mottling	4	3.85

In the present study among 104 patients, 67.31% of patients on chest X-ray had infiltration, 37.50% had cavitation and 38.46% had fibrosis. The fibrocavitatory lesions were seen in 32.69% and miliary pattern was present in 3.85% patients.

Table 17. Chest X-ray findings involving different lung zones

Chest X-ray findings	Zones					
	Upper		**Middle**		**Lower**	
	No.	**%**	**No.**	**%**	**No.**	**%**
Infiltration	54	51.92	40	38.46	19	18.27
Cavitation	23	22.12	10	9.62	9	8.65
Fibrosis	30	28.85	7	6.73	4	3.85



In the present study, 51.9% of patients had upper zone infiltrations. 38.46% of patients had middle zone infiltration and 18.27% of patients had lower zone infiltration.

22.12% of patients had upper zone cavitation, 9.62% of patients had middle zone cavitation and 8.65% of patients had lower zone cavitations.

28.85% of patients had upper zone fibrosis, 6.73% of patients had middle zone fibrosis and 3.85% patients had lower zone fibrosis.

COMPARISON OF CLINICAL AND LABORATORY PROFILE AMONG HIV SEROPOSITIVE AND HIV SERONEGATIVE PATIENTS

Table 18. Sex

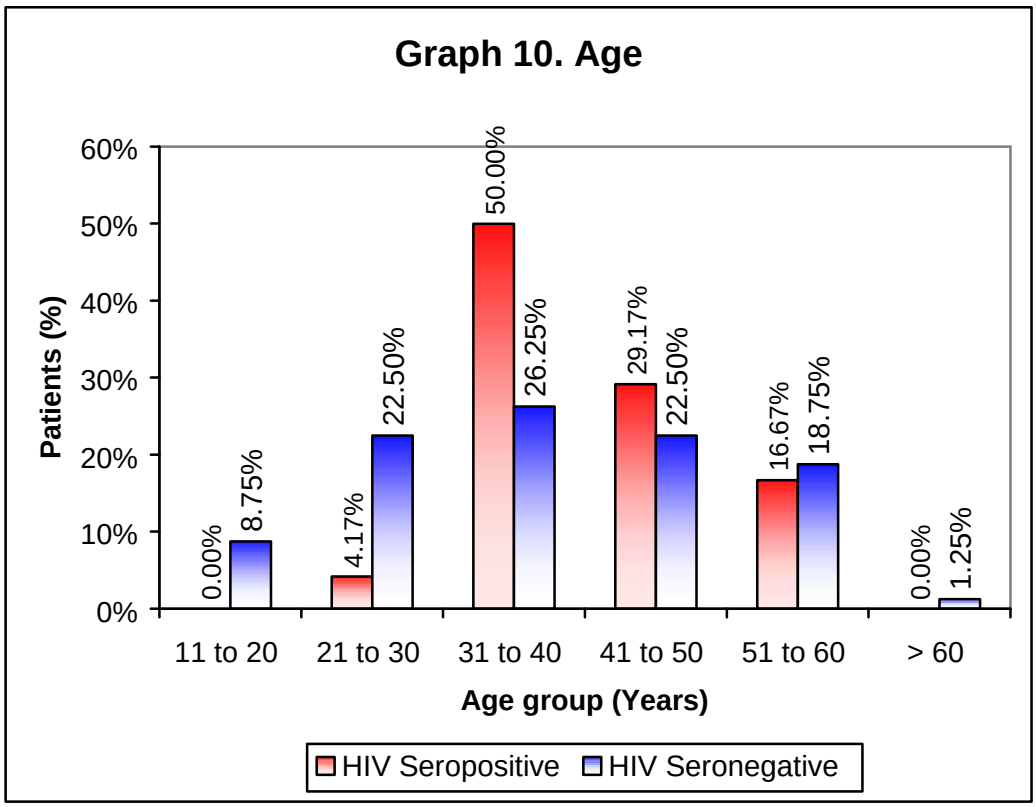
Sex	HIV Seronegative (n=80)		HIV Seropositive (n=24)	
	Number	**Percentage**	**Number**	**Percentage**
Male	59	73.75	19	79.17
Female	21	26.25	5	20.83
Total	80	100	24	100
$x^2 = 0.289$		DF = 1		P = 0.591

In the present study, male patients comprised of 73.75% in the HIV seronegatives and 79.17% in HIV seropositives, female patients comprised of 26.25% in HIV seronegatives and 20.83% in HIV seropositives suggesting equal distribution of gender in both the groups.

Table 19. Age

Age group (Years)	HIV Seronegative (n=80)		HIV Seropositive (n=24)	
	Number	Percentage	Number	Percentage
11 to 20	7	8.75	0	0.00
21 to 30	18	22.50	1	4.17
31 to 40	21	26.25	12	50.00
41 to 50	18	22.50	7	29.17
51 to 60	15	18.75	4	16.67
> 60	1	1.25	0	0.00
Total	80	100	24	100

x^2=9.146 DF=3 p=0.027



In the present study, 26.25% of patients were aged between 31 to 40 years in the HIV seronegatives compared to 50% in HIV seropositive group. This difference was statistically significant (p=0.027).

Table 20. Occupation

Occupation	HIV Seronegative (n=80)		HIV Seropositive (n=24)	
	Number	**Percentage**	**Number**	**Percentage**
Agricultural worker	22	27.50	5	20.83
Public servant	8	10.00	2	8.33
Driver	4	5.00	4	16.67
House wife	12	15.00	3	12.50
Labourer	14	17.50	6	25.00
Merchant / business owner	3	3.75	4	16.67
Student	17	21.25	0	0.00
Total	80	100	24	100

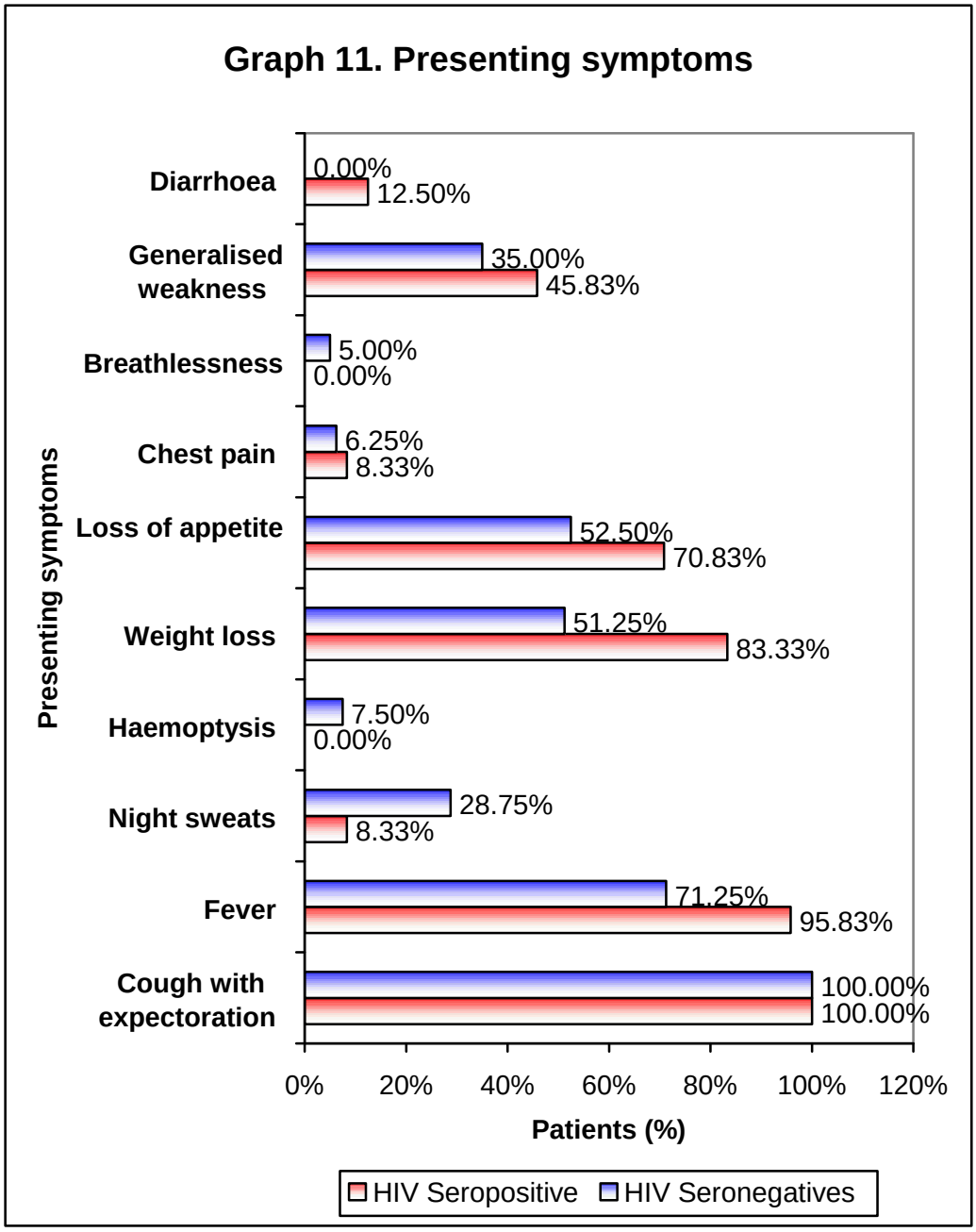
x^2 = 13.932 DF = 6 P = 0.030

In the present study, 27.50% of patients were agriculture workers in the HIV seronegatives compared to 20.83% in HIV seropositives. This difference was statistically significant. 17.50% of patients with HIV seronegative status were daily labourers compared to 25% in the HIV seropositive and 3.75% of patients with HIV seronegative status were businessmen/merchants compared to

16.67%. 21.25% of patients were students in the HIV seronegatives. 15% of patients were housewives in HIV seronegatives compared to 12.50% in HIV seropositives. 10.00% of patients and 5% were public servants and driver in HIV seronegatives compared to 8.33% and 16.67% in the HIV seropositives.

Table 21. Presenting symptoms

Symptoms	HIV seronegative (n = 80)		HIV Seropositive (n=24)		Z	P
	No.	%	No.	%		
Cough with expect	80	100.00	24	100.00	0	1
Fever	57	71.25	23	95.83	2.51	<0.02
Night sweats	23	28.75	2	8.33	2.05	<0.05
Hemoptysis	6	7.50	0	0.00	1.37	>0.05
Weight loss	41	51.25	20	83.33	2.79	<0.01
Loss of appetite	42	52.50	17	70.83	1.60	>0.05
Chest pain	5	6.25	2	8.33	2.36	>0.05
Breathlessness	4	5.00	0	0.00	1.12	>0.05
Generalised weakness	28	35.00	11	45.83	0.96	>0.05
Diarrhoea	0	0.00	3	12.50	3.2	<0.05



In the present study, majority of the patients presented with cough with expectoration, fever, night sweats, loss of weight and loss of appetite. Fever, loss of weight, loss of appetite, generalized weakness and diarrhoea symptoms were more predominant in HIV seropositive patients compared to HIV seronegative. This difference between the two groups was statistically significant. However,

symptoms such as night sweats, haemoptysis, chest pain and breathlessness were more predominant in HIV seronegative patients compared to HIV seropositives.

Table 22. Past history

Past History	HIV seronegative (n = 80)		HIV Seropositive (n=24)		Z	p
	No.	%	No.	%		
Known HIV status	0	0.00	8	33.33	5.37	0.000
Contact with TB	16	20.00	1	4.17	1.84	>0.05
Herpes zoster	1	1.25	12	50.00	6.33	0.000
Diabetes mellitus	10	12.50	1	4.17	1.16	>0.05

In the present study, 33.33% of patients were known HIV positive. 50% of patients had history of Herpes zoster in the past in HIV seropositives compared 1.25% in the seronegatives and this difference was statistically significant.

20% of patients among HIV seronegative had history of contact with sputum smear positive TB compared to HIV seropositive and diabetes was seen in 12.5% of patients whereas 4.17% had diabetes among HIV seronegatives with no statistical significance.

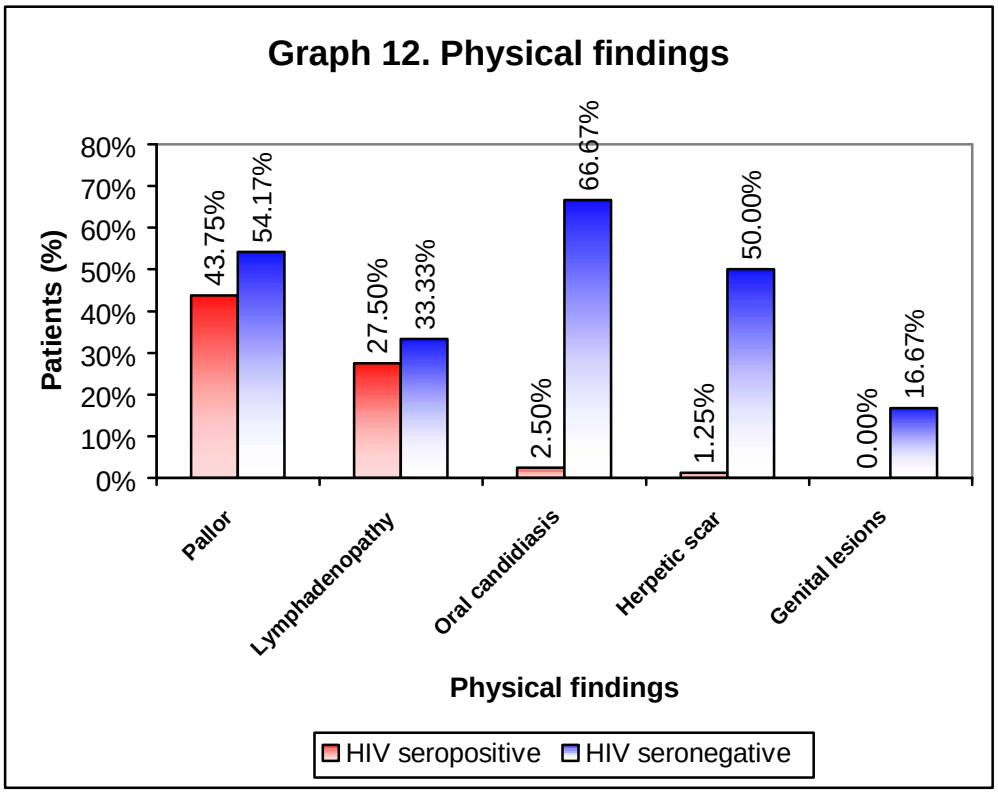
Table 23. Body mass index

Parameter	HIV seronegative (n=80)		HIV seropositive (n=24)		t	DF	P
	Mean	SD	Mean	SD			
BMI (Kg/m^2)	17.4	4.03	15.8	2.23	2.857	102	0.05

In the present study, the mean BMI was significantly low (15.8±2.23 kg/m^2) in HIV seropositive compared to HIV seronegative patients (17.4±4.03 kg/m^2).

Table 24. Physical findings

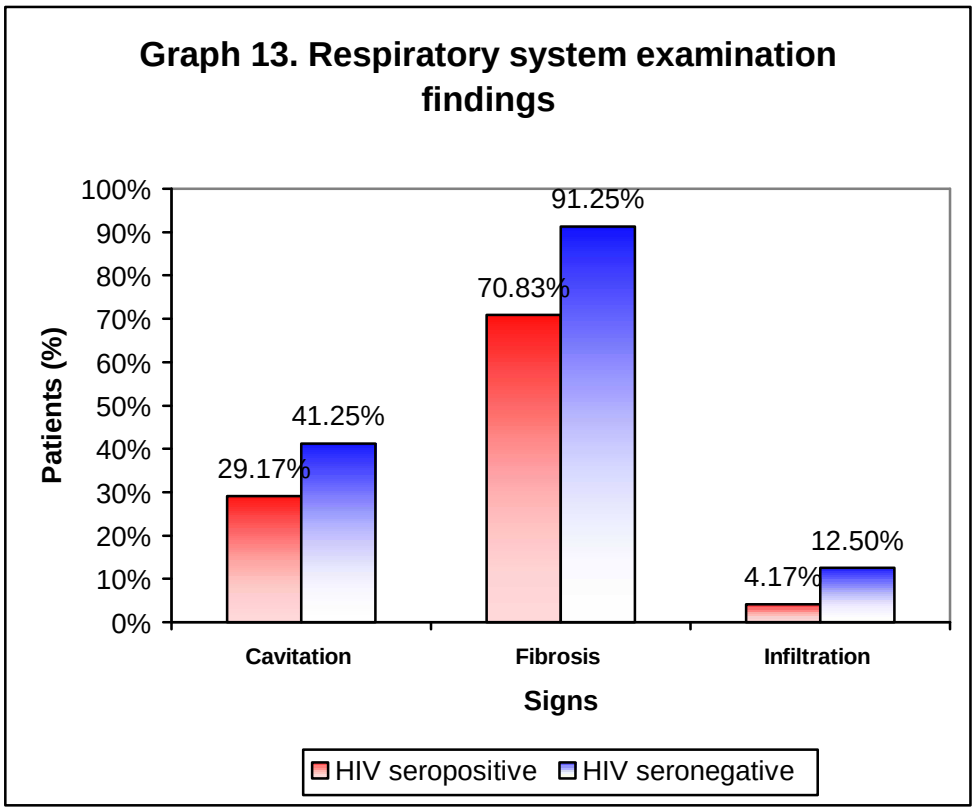
Physical findings	HIV seronegative (n = 80)		HIV seropositive (n = 24)		Z	P
	No.	%	No.	%		
Pallor	35	43.75	13	54.17	0.89	>0.05
Lymphadenopathy	22	27.50	8	33.33	0.55	>0.05
Oral candidiasis	2	2.50	16	66.67	7.28	< 0.001
Herpetic scar	1	1.25	12	50.00	6.33	< 0.001
Genital lesions	0	0.00	4	16.67	3.74	< 0.001



In the present study, on physical examination, 66.67% of patients in the HIV seropositives had oral candidiasis, 50% had herpetic scar and 16.67% had genital lesions compared to HIV seronegatives. This difference was statistically significant. 43.75% of patients had pallor, 27.50% had lymphadenopathy in the HIV seronegatives compared to HIV seropostiives. This difference was not significant.

Table 25. Respiratory system examination findings

Signs	HIV Seronegative (n=80)		HIV Seropositive (n=24)		z	p
	No	%	No	%		
Cavitation	33	41.25	7	29.17	1.07	> 0.05
Fibrosis	73	91.25	17	70.83	2.57	< 0.01
Infiltration	10	12.50	1	4.17	1.16	> 0.05



In the present study, 91.25% of patients among HIV seronegatives had signs of fibrosis compared to 70.83% HIV in seronegatives and this difference was statistically significant ($p<0.01$). In the seronegatives, 41.25% and 12.5% of patients had signs of cavitation and infiltration compared to 29.17% and 4.17% in the HIV seronegatives.

Table 26. Haematological parameters

Haematological Parameters	HIV seronegative (n=80)		HIV seropositive (n=24)		t	DF	p
	Mean	SD	Mean	SD			
Hb (gm%)	10.1	1.92	8.2	0.88	4.690	102	<0.005
TLC (Cells/mm^3)	12465.8	3950.62	7039.2	2049.57	6.458	102	<0.001
ESR (mm)	62.4	26.60	59.70	25.36	0.441	102	>0.05

In the present study, the mean haemoglobin percentage and TLC in the HIV seropositive patients were significantly low compared to HIV seronegatives. However, no statistically significant association was found with regard to ESR.

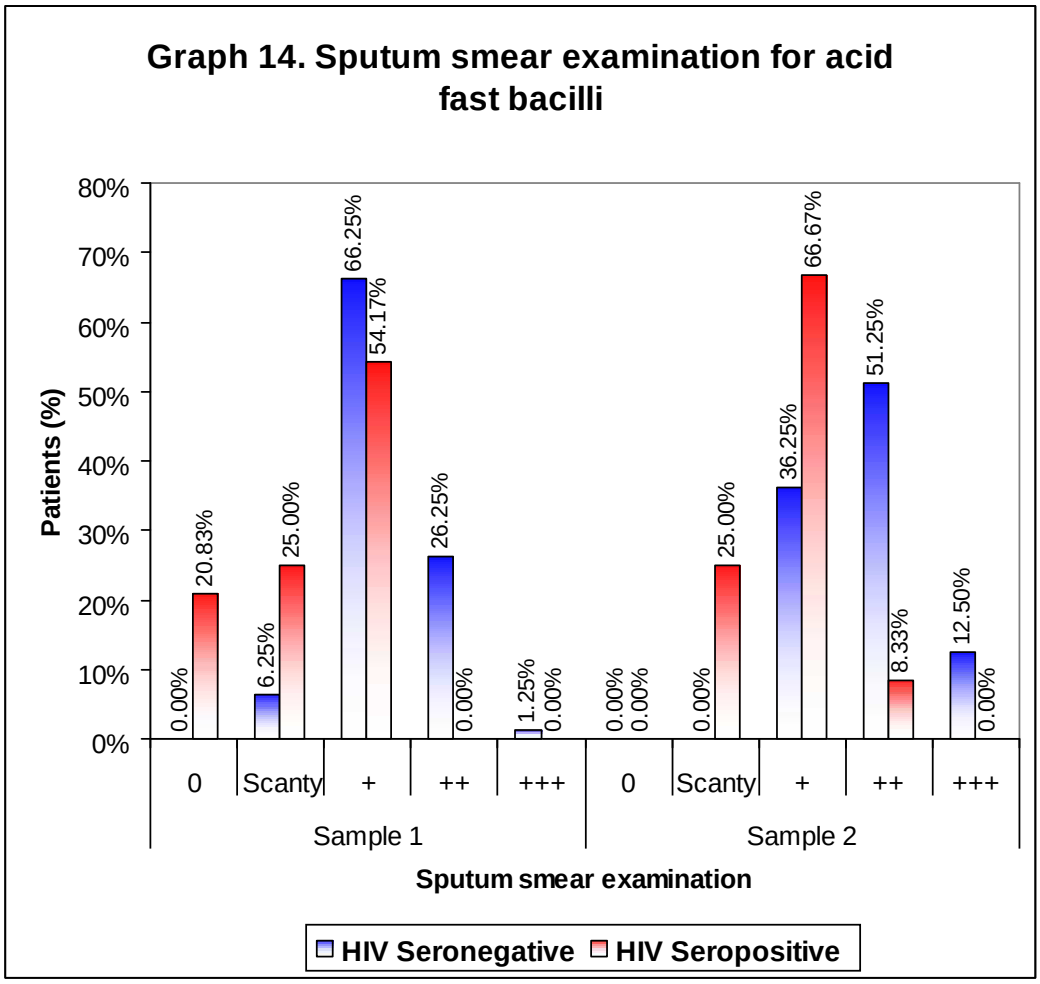
Table 27. Biochemical parameter

Biochemical Parameters	HIV seronegative (n=80)		HIV seropositive (n=24)		t	DF	P
	Mean	SD	Mean	SD			
Urea (mg/dL)	30.4	18.52	32.30	12.95	0.923	102	>0.05
Serum Creatinine (mg/dL)	1	0.35	1.00	0.29	0	102	1
Serum sodium (meq/L)	138.7	4.61	137.00	5.69	0.419	102	>0.05
Serum potassium (meq/L)	3.9	0.62	3.70	0.58	1.406	102	>0.05

In the present study no association was found in mean serum urea, serum creatinine, serum sodium and serum potassium levels among HIV seropositive and HIV seronegative patients.

Table 28. Sputum smear examination for acid fast bacilli

AFB Grades	Sample 1				Sample 2			
	HIV Seronegative (n=80)		HIV Seropositive (n=24)		HIV Seronegative (n=80)		HIV Seropositive (n=24)	
	No.	%	No.	%	No	%	No	%
0	0	0.00	5	20.83	0	0.00	0	0.00
Scanty	5	6.25	6	25.00	0	0.00	6	25.00
+	53	66.25	13	54.17	29	36.25	16	66.67
++	21	26.25	0	0.00	41	51.25	2	8.33
+++	1	1.25	0	0.00	10	12.50	0	0.00
	x^2=29.829	DF=3	p=0.000		x^2=35.171	DF=3	p=0.000	



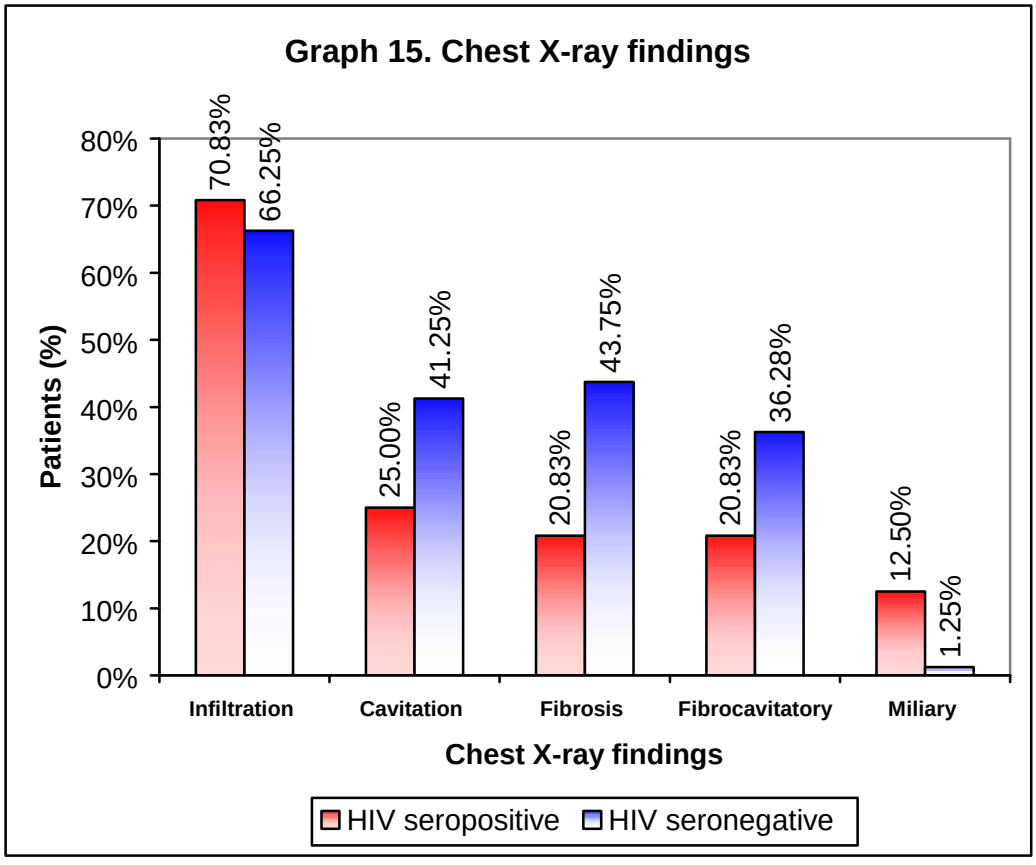
In the present study, findings of first sputum smear sample revealed 66.25% of patients had grade +, 26.75% with ++ and 1.25% with +++ in the HIV seronegatives compared to HIV seropositives. Whereas, 20.83% and 25% of patients had grade 0 and scanty AFB grading in the seropositives compared to seronegatives. This difference was statistically significant.

The results of sample 2 revelaed that, 66.67% of patients had grade + for AFB on sputum smear examination in the seropositives compared to 36.25% in the HIV seronegatives. Whereas, 51.25% of patients and 12.50% of patients had ++ and +++ grading for AFB in the HIV seronegative group compared to HIV seropositive group. However 25.00% of patients had scanty grading for AFB

among HIV seropositive patients compared to HIV seronegative. These differences were statistically significant.

Table 29. Chest X-ray findings

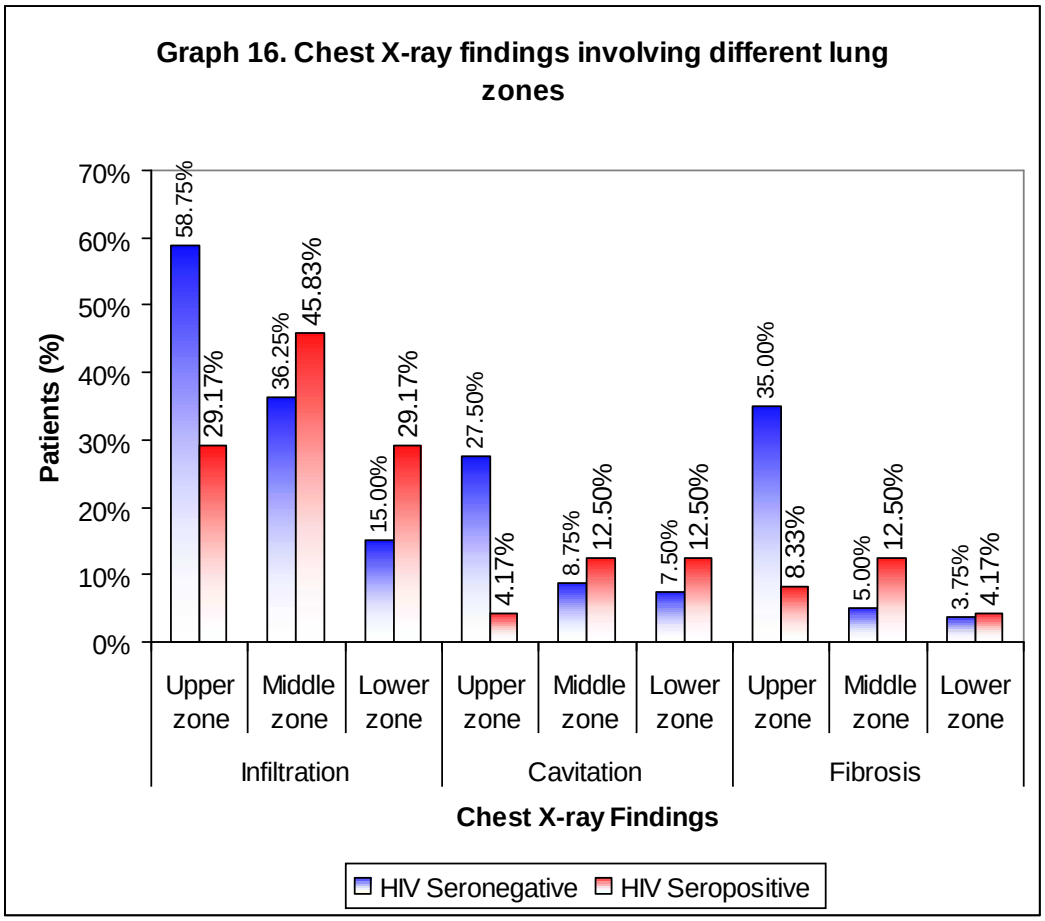
Chest X-ray findings	HIV seronegative (n=80)		HIV seropositive (n=24)		Z	P
	No	%	No	%		
Infiltration	53	66.25	17	70.83	0.47	>0.05
Cavitation	33	41.25	6	25.00	1.44	>0.05
Fibrosis	35	43.75	5	20.83	2.02	<0.05
Fibrocavitatory Lesion	29	36.25	5	20.83	1.34	>0.05
Miliary mottling	1	1.25	3	12.50	2.52	<0.02



In the present study, 43.75% of patients had fibrosis and 1.25% had military pattern on chest X ray in HIV seronegatives compared to 20.83% and 12.5% in HIV seropositives. This difference was statistically significant. However no statistically significant difference was found with respect to infiltration, cavitation and fibrocavitatory lesions.

Table 30. Chest X-ray findings involving different lung zones

Chest X-ray findings	HIV Seronegative (n = 80)		HIV Seropositive (n = 24)		Z	p
	No	%	No	%		
Infiltration						
Upper zone	47	58.75	7	29.17	2.54	<0.02
Middle zone	29	36.25	11	45.83	0.84	>0.05
Lower zone	12	15.00	7	29.17	1.57	>0.05
Cavitation						
Upper zone	22	27.50	1	4.17	2.41	<0.03
Middle zone	7	8.75	3	12.50	0.73	>0.05
Lower zone	6	7.50	3	12.50	0.77	>0.05
Fibrosis						
Upper zone	28	35.00	2	8.33	2.53	<0.02
Middle zone	4	5.00	3	12.50	1.29	>0.05
Lower zone	3	3.75	1	4.17	0.09	>0.05



In the present study, 58.75% of patients among HIV seronegative had upper zone infiltrations compared to 29.17% in HIV seropositives. This difference was statistically significant. 45.83% of patients and 29.17% among HIV seropositive had middle and lower lobe infiltration compared to 36.25% and 15% in the HIV seronegatives.

In the present study, 27.50% of patients among HIV seronegative had upper lobe cavitations compared to 4.17% in the HIV seropositives. This difference was statistically significant. 8.75% of patients and 7.50% in the seronegatives had middle and lower lobe cavitations compared to 12.50% and 12.50% in the HIV seropositives.

In the present study, 35% of patients among HIV seronegatives had upper zone fibrosis compared to 8.33% among HIV seropositive. This difference was statistically significant. 5% and 3.75% of patients among seronegatives had middle and lower zone fibrosis.

DISCUSSION

The present study, comprised of 104 patients out of which, 75% were males and 25% were females with male to female ratio of 3:1 (Table 1).

Most of the patients (31.73%) in the present study were between 31 to 40 years of age with mean age of presentation being 38.68 ± 12.01 (Table 2).

A study[7] conducted in Chennai showed 64.80% of TB patients aged between 21 to 40 years whereas another study[1] done in Ethiopia showed 54% of TB patients were in the age group of 31 to 40 years.

In the present study, most of the patients were agricultural workers (25.96%) and labourers (19.23%) which reflects low socio economic status (Table 4).

A study[1] done in Northwest Ethiopia among PTB patients reported that 19.1% were farmers and 18.1% were daily labourers. Another study[6] done in Mangalore showed 28% were agricultural workers, 21% were daily laboruers and 6% were drivers.

In the present study, majority (100%) of the patients had cough with expectoration, fever (76.92%) weight loss (58.65%), loss of appetite (56.73%) and generalized weakness (37.50%) (Table 5).

A study[1] conducted at a Teaching Hospital of Northwest Ethiopia showed that among PTB patients, 93% had low grade fever, 90.3% had weight loss, 86% had night sweats and 82.5% had cough with expectoration.

In the present study, 7.69% of patients were known HIV seropostive, 16.35% of patients had history of contact with sputum positive pulmonary tuberculosis and 12.50% patients had history of herpes zoster in the past (Table 6).

In this study among PTB patients coinfected with HIV, 10.58% of patients had history of multiple sexual partners, 2.80% of patients got infected through blood transmission and 1.4% had STD's. The possible source of HIV infection could not be ascertained in four patients (Table 7).

A study[101] done in Lucknow, India showed 86.6% HIV seropositive patients with a history of heterosexual promiscuity and 9.7% got infected through blood and blood products. Another study[102] done in Vadodara, Gujarat found that, 65% of patients had history of multiple sexual partners and 13.8% had blood transfusion among HIV seropostivie patients.

In the present study, majority (73.08%) of the patients were underweight (BMI less than 18.5 kg/m^2) and 24.04% of patients had normal BMI (Table 8).

A study[1] conducted in Ethiopia among TB patients found 67.3% of the patients with BMI of less than 18.5 kg/m^2.

In this study patients had a wide spectrum of physical signs on examination. Majority of the patients had pallor (46.15%) and lymphadenopahty (28.85%). Other signs like oral candidiasis, herpetic scar and genital lesions were present in 17.31%, 12.50% and 3.85% of patients respectively (Table 9).

On respiratory system examination, 86.54% of patients had signs of fibrosis, 38.46% had signs of cavitation and 10.58% had signs of infiltration (Table 10).

A study[1] done in Ethiopia among PTB patients found 47.1% of patients had sign of fibrosis, 77% of patients had signs of cavitation and 50% of patients had signs of infiltration.

In the present study, majority (50%) of the patients had moderate anaemia (Hb 7 to 10 gm%) and severe anemia was present in 4.85% of patients (Hb less than 7 gm%) (Table 11).

A study[103] done in Tanzania showed 75.6% of patients had moderate anemia and 24.4% had severe anemia.

In this study, 44.23% of patients had TLC in the range of 4000 to 11000 and 52.88% of patients had leucocytosis and 2.88% had leucopenia. All patients had elevated ESR levels (Table 11). Among the patients with PTB co-infected with HIV, 54.17% of patients CD4 count levels were more than 200 cells/mm^3 and 33.33% had in between 50 to 200 cells/mm^3 whereas 12.5% had CD4 count less than 50 cells/cm^3 (Table 12).

In this study, blood urea was normal in 75% and serum creatinine in 80.77% of patients. Serum sodium levels were normal among 85.5% of patients and in 90.38% of patients serum potassium levels were within normal range. 4.81% of patients had hyponatremia (Table 13).

In the present study, 76.92% of patients were HIV seronegative and 23.08% of patients were HIV seropositive (Table 14).

A study[1] done in Ethiopia among PTB patients showed 47.9% were seronegative and 52.1% were seropositive. Another study[101] done at Lucknow showed 2.8% as of patients were HIV seropositive.

On sputum examination, majority of patients sputum smear for AFB showed 63.46% of patients in the sample 1 and 43.27% of patients in sample 2 were recorded as grade + (1+) followed by grade ++ (2+) in 20.19% in sample 1 and 41.35% in sample 2 (Table 15).

In this study, chest X-ray findings among 104 patients showed 67.35% had infiltrations, 37.50% had cavitation and 38.46% had fibrosis. The fibrocavitatory lesions were seen in 32.69% and miliary mottling was present in 3.85% of patients (Table 16). The upper zone involvement was more predominant with regard to infiltration, cavitations and fibrosis (Table 17).

COMPARISON OF CLINICAL AND LABORATORY PROFILE AMONG HIV SEROPOSITIVES AND HIV SERONEGATIVES

In the present study among 104 patients, 80 (76.92%) patients were HIV seronegative and 24 (23.08%) patients were HIV seropositive (Table 18). The seroprevlance of HIV among PTB patients in this study was 23.08%. Studies[3,4] done in Jamaica and AIIMS, New Delhi have reported a prevalence of 11.6 to 20% among PTB patients.

In this study, HIV seroprevalence rate among men was 79.17% and 20.83% in females. This is a comparable to a study[1] in Northwest Ethiopia where HIV seroprevalence among men was 74% and 26% in females. Another study[7] done in Chennai showed HIV seroprevalence in 79.25% among men and 20.75% in women.

In the present study, seroprevalence of HIV was highest (50%) in the age group of 31 to 40 years (Table 19). This age group had significant association with seroprevalence (p=0.027). This is comparable to a study[7] done in Chennai which showed 74.94% of patients within the age of 21 to 40 years. National statistics reported by NACO shows 89% of cases in the age group of 15 to 44 years.[104] This age reflects the sexually active age group which is commonly affected by the disease and screening for HIV should be done among male patients aged between 31 to 40 years, as 50% of all seropositive cases were found in this age group.

In the present study, among the occupations HIV seroprevalecne was highest (25%) in labourers followed by agriculture workers (20.83%) indicating

co-infection affects people of low socio-economic status (Table 20). Study[4] done in New Delhi found similar occupational profile.

In the present study, majority of the patients presented with symptoms of cough with expectoration, fever, night sweats, loss of weight and loss of appetite. Fever, loss of weight, loss of appetite and generalized weakness were more predominant among HIV seropositive patients compared to HIV seronegative (Table 21).

These findings were comparable to a study[5] conducted in Manipur where cough with expectoration was present among HIV seronegative and weight loss, loss of appetite and generalized weakness among HIV seropositives. Another study[1] done in Ethiopia showed similar presentation where cough with expectoration, haemoptysis, chest pain, breathlessness were more in HIV seronegative and weight loss, loss of appetite and diarrhoea in HIV seropositives. A Study[7] done in Chennai reported similar clinical profile of symptoms. All patients presented with cough with expectoration which reflects mild immuno suppression with higher CD4 count.

In the present study, among HIV seropositive patients, 33.33% of patients were known HIV earlier and history of contact with sputum smear positive PTB was present in 20% of HIV seronegatives compared to none in HIV seropositive. Opportunistic infection like Herpes zoster was present in 50% of HIV seropositive patients compared to 1.25% in HIV seronegatives (Table 22). This is similar to a study[1] conducted in Ethiopia where 27% of patients had Herpes zoster in the past among HIV seropositives compared to 4% in HIV

seronegatives. 12.50% and 4.17% were diabetics among HIV seropositive and HIV seronegative patients.

Malnutrition as evidenced by BMI was significantly low (BMI 15.8 ± 2.23 kg/m^2) among HIV sropositive patients compared to seronegative patients (BMI 17.4 ± 4.03 kg/m^2). This can be attributable to low socioeconomic status with superadded HIV infection (Table 23).

In the present study patients had a wide spectrum of physical findings on examination. Opportunistic infections like oral candidiasis (66.67%) herpetic skin scar (50%) and genital lesions (16.67%) were more common among HIV seropositive compared to HIV seronegative (Table 24). This is comparable to study[7] conducted at Chennai where 38% of patients had oral candidiasis and 27% had herpetic scar among HIV seropositive compared to none among HIV seronegative patients.

Other findings like lymphadenopathy was seen in 33.33% in HIV seropositive compared to 27.50% in HIV seronegative. This is comparable to a study[1] done in Ethiopia where 29% of patients among HIV seropositive had lymphadenopathy compared to 22% in HIV seronegative. 54.17% of patients among HIV seropositive had pallor compared to 43.75% in HIV seronegative patients.

In the present study clinical findings on respiratory system examination showed 41.25% of patients with signs of cavitation, 91.25% with signs of fibrosis in the HIV seronegative compared to 29.17% and 70.83% in the HIV seropositives (Table 25). This is comparable to a study[1] conducted in Ethiopia

where 68.2% of patients had sign of cavitations, 56.4% had sign of fibrosis in the HIV seronegative compared to 31.8% and 43.6% in HIV seropositive patients. History of cough expectoration and haemoptysis and sign of cavitation as well as coarse crepitation in the chest were independent predictions of sputum smear positive TB. 12.50% of patients in the seronegative had sign of infiltration compared to 4.17% in HIV seropositives.

In the present study, hematological parameters such as mean hemoglobin and TLC were significantly lower in HIV seropositives (8.2%) compared to HIV seronegatives. Mean ESR (mm) levels were comparable in HIV seropositive and HIV seronegative patients (Table 26).

In this study with regard to biochemical parameters, serum creatinine, serum urea, serum sodium, serum potassium were comparable in seropositive and seronegative patients (Table 27).

In the present study on sputum examination majority of patients among HIV seropositives and HIV seronegatives were recorded as grade + for AFB and also showed higher grading (+, ++, +++) among HIV seronegatives compared to HIV seropositives. While, 25% and 20.83% among HIV seropositive patients were recorded as scanty and grade 0 compared to HIV sronegatives in the first sputum sample. Similar findings were noted in the sample 2. This reflects that sputum positive for AFB were recorded at a lower grade among HIV seropositive due to low CD4 counts and less number of patients with cavitatory lesions (Table 28).

In this study, the chest x-ray findings showed, fibrosis, cavitation and fibrocavitatory lesions were more common in the HIV seronegative compared to HIV seropositive patients. However, infiltrative lesions and miliary mottling were more common in HIV seropositives compared to HIV seronegative patients (Table 29).

This is comparable to study[1] conducted in Ethiopia where 50% of patients had fibrosis and 13.3% had miliary mottling among HIV seropositive compared to HIV seronegatives. Another study[7] in Chennai showed, 83.33% of patients had fibrocavitatory lesions among HIV seronegatives compared to 11.15% in HIV seropositives. Another study[101] showed cavitary lesions were less common (29% vs 36.1%) among HIV seropositive patients as compared to HIV seronegative patients.

Another study[7] done in Chennai showed 91% and 39% of patients among HIV seronegative had diffuse infiltrates and cavitation compared to 65% and 18% in HIV seropositives.

In this study, the x-ray findings showed upper zone involvement was more among HIV seronegative with regard to infiltration (58.75%), cavitation (27.5%) and fibrosis (35%) compared to 29.17%, 4.17% and 8.33% in HIV seropositive respectively (Table 30).

Among infiltrative lesions, 45.83% and 29.17% of patients had middle and lower zone involvement among HIV seropositive as compared to 36.25% and 15% in seronegative patients (Table 30).

This is comparable to a study[101] done in Lucknow where upper zone infiltration was less common (12.5% vs 54.7%) while middle zone and lower zone (25% vs 7.6) (20.8% vs 6.7) involvement were more common among HIV seropositive as compared to HIV seronegative patients of PTB.

Among HIV seropositive patients typical radiological features of post primary TB that is, upper zone infiltrates were less common, while atypical features such as mid and lower zone infiltrates were more common in seropositive patients. It has been shown that in mild immuno suppression the appearance is often classical while in severe immune suppression, it is atypical.

Miliary mottling was seen in 12.5% among HIV seropositive compared to 1% in HIV seronegative. This could be probably due to more dissemination due to low CD4 count and severe immune suppression (Table 29). This is similar to a study[63] done in Chetput, Chennai showed 11% patients among HIV seropositives had miliary mottling compared to 1% among HIV seronegatives.

CONCLUSION

Seroprevalence of HIV among pulmonary tuberculosis patients was found to be 23.08%. Seroprevalence rate among males were high (79.17%) compared to females and was highest (50%) in the age group between 31 to 40 years. Also, seropositivity was high among agricultural workers and labourers.

HIV seropositive PTB patients commonly present with fever, weight loss and loss of appetite while cough with expectoration, hemoptysis, breathlessness were more common with HIV seronegative patients.

On examination, anaemia, undernourishment, lymphadenopathy and the presence of opportunistic infections like oral candidiasis, herpes zoster and genital lesions were more predominant among HIV seropositives compared to HIV seronegatives.

On respiratory system examination, signs of fibrosis and cavitation were more predominant in HIV seronegative compared to HIV seropositives.

Mean Hb and TLC were significantly low among HIV seropositives compared to HIV seronegatives. Sputum smear examinations for AFB showed higher grades among HIV seronegatives and low grades among HIV seropositives. Chest X-ray showed varied presentation. Cavitation, fibrosis and fibrocavitatory lesions were predominantly seen among HIV seronegatives while infiltration and miliary mottling was present in HIV seropositives. Upper zone infiltration, cavitation and fibrosis were more commonly involved among HIV seronegatives compared to HIV seropositives.

SUMMARY

The global impact of the converging dual epidemics of TB and HIV is one of the major public health challenges. The increasing rate of HIV infection in many countries has had an impact on TB epidemiology. While TB prevalence has remained stable, TB incidence continues to rise, especially in countries most severely affected by the HIV epidemic as well as those facing political turmoil, migration, poverty and unemployment and where intravenous drug abuse is rampant.

As the prevalence of pulmonary tuberculosis is increasing among HIV seropositive patients with a wide range of immune status and clinical presentations, the present study was undertaken to assess the clinical and laboratory profile of sputum positive pulmonary tuberculosis among HIV seropositive and HIV seronegative patients.

The present one year cross sectional study was conducted in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum on 104 patients with sputum positive pulmonary tuberculosis patients during the period of January 2009 to December 2009. Routine investigations such as blood group, haemogram that is, haemoglobin, total count, differential count, erythrocyte sedimentation rate, sputum smears for AFB, chest X-ray were done.

Seroprevalence of HIV among pulmonary tuberculosis patients was 23.08%. Seroprevalence rate among males were high (79.17%) compared to

females and the majority (50%) of the patients had age between 31 to 40 years. Also seropositivity was high among agricultural workers and labourers.

Cough with expectoration (100%), fever (95.83%), weight loss (83.33%) and loss of appetite (70.83%) were more predominant in HIV seropositives compared to HIV seronegatives. On examination anaemia, undernourishment, lymphadenopathy and the presence of opportunistic infections like oral candidiasis, herpes zoster stain and and genital lesions were more predominant among HIV seropositives compared to HIV seronegatives. Signs of fibrosis and cavitation were more predominant in HIV seronegative compared to HIV seropositives.

Mean Hb and TLC were significantly low among HIV seropositives compared to HIV seronegatives. Sputum smear examinations for AFB showed higher grades among HIV seronegatives and low grades among HIV seropositives. Chest X-ray showed varied presentation. Cavitation, fibrosis and fibrocavitatory lesions were predominantly seen among HIV seronegatives while infiltration and military mottling was present in HIV seropositives. Upper zone infiltration, cavitation and fibrosis were more commonly involved among HIV seronegatives compared to HIV seropositives.

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ANNEXURE I - CONSENT FORM

"CLINICAL AND LABORATORY PROFILE OF SPUTUM POSITIVE PULMONARY TUBERCULOSIS AMONG HIV SEROPOSITIVE AND HIV SERONEGATIVE PATIENTS – A CROSS SECTIONAL STUDY"

Objective and purpose of the study

This research is intended to study clinical and laboratory profile of pulmonary tuberculosis. The principal investigator of the study is Dr. ***** ****** under the guidance of Dr. ***** ***** Professor, Department of Medicine, J. N. Medical College, Belgaum. Your co-operation will be of great help to patients with Pulmonary tuberculosis in future.

Procedure

If you agree to be part of the research study you will be asked the relevant history and will be subjected to relevant clinical examination and investigations. You will also have to give blood samples and undergo other necessary investigations like sputum examination and chest X-ray.

Risk and Benefits

The only risk and possible discomfort you might get is while taking blood from your arm for the investigations which may cause swelling, pain, redness, bruising or infection (rarely happens) at the site from where the blood is drawn.

Alternatives

Taking part in this study is voluntary. You may choose not to take part in this study, or if you decide to take part you can later change your mind and

withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsor may stop your participation in this study any time. If you choose not to take part in the study you will receive the standard treatment like other patients.

Privacy and Confidentiality

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy

Does not apply to this research

Financial incentives for participation

You will not be paid / offered any gifts /incentives for participating in the study.

Authorization to publish the results

The results of the study would be forwarded to the KLE University, Belgaum as part of requirement towards the completion of MD degree, review and publishing.

If you have any questions about your rights as a participant you may call Principal and Chairman, J.N.M.C Ethical Committee for Human Research.

In case of the queries during study or in future you may contact following person.

Principal investigator : Dr. ****** ******* Mobile No.: ***** *****

Guide : Dr. ****** ******* Tele. No. : ***** *****

Consent Statement

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read, or it has been read to me, this entire consent form, and have had all my questions answered.

Name of the Participant: _____ Signature or _____
Thumb print

Name of the Witness _____ Signature _____

Name of the Investigator _____ Signature _____

Date: Place:

ANNEXURE II – PROFOMA

"CLINICAL AND LABORATORY PROFILE OF SPUTUM POSITIVE PULMONARY TUBERCULOSIS AMONG HIV SEROPOSITIVE AND HIV SERONEGATIVE PATIENTS – A CROSS SECTIONAL STUDY"

Patient Name: I.P number:

Age: Sex:

Date of admission: Date of discharge:

Address: Occupation:

Chief complaints

Complaints	Duration
Cough with expectoration	
Fever	
Night sweats	
Haemoptysis	
Weight loss	

History of Present Illness

Treatment History

Past History

Family History

History of High risk behaviour

Personal history

Smoking :

Alcohol intake :

Tobacco chewing :

Any other :

GENERAL PHYSICAL EXAMINATION

Built and nourishment:		Palpabral conjunctiva	:
Lymphadenopathy	:	Oral cavity	:
Cyanosis	:	Clubbing	:
Weight	:	BMI	:

Vital Signs

Temperature	:	Pulse rate	:
Blood pressure	:	Respiratory rate	:

SYSTEMIC EXAMINATION

Respiratory system

Inspection :

Palpation :

Percussion :

Auscultation :

Special tests for cavity

Post tussive crepitations :

Post tussive succussion :

Whispering pectoriloguy :

Egophony

CVS :

Per abdomen :

CNS :

INVESTIGATIONS

Complete blood count

Hb% :

TC :

DC : N - L - E - M –

B -

ESR : HIV :

Blood Sugar

FBS : PPBS :

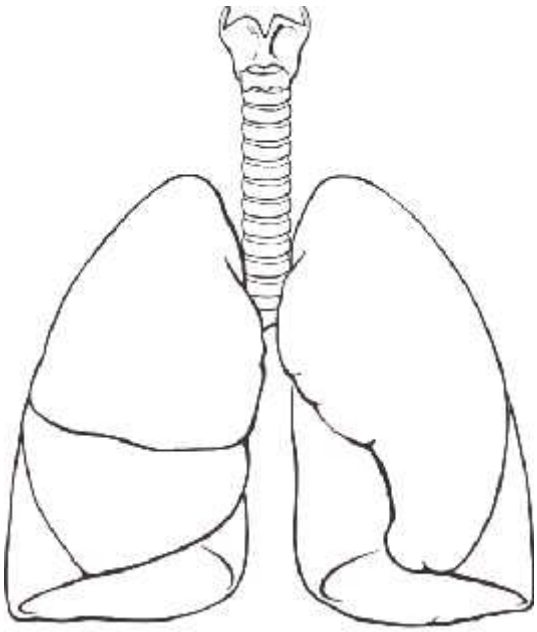
Sputum examination

Microscopy

Ziehl Neelsen Stain

	Date	**Grading of AFB**
Sample 1		
Sample 2		
Sample 3		

Chest X-ray :



Fundoscopy :

Treatment given :

ANNEXURE III - MASTER CHART

Sl. No.	IP. No.	Sex	Age (Years)	Marital Status	Occupation	Symptoms										Past History					Risk factors for HIV				General Physical Examination									Systemic examination			
						Cough with expectoration	Fever	Night Sweats	Haemoptysis	Weight loss > 10%	Loss of appetite	Diarrhoea	Chest pain	Breathlessness	Generalised weakness	HIV status	Contact with tuberculosis	Herpez Zoster	Hypertension	Diabetes mellitus	Multiple sexual partners	H/o Blood transfusion	Sexually transmitted disease	Intrvenous drug abuse	Height (Cms)	Weight (Kg)	Body mass index (Kg/m^2)	Pallor	Lymphadenopathy	Oral candidiasis	Herpetic Scar	Genital lesions	Cyanosis	Signs of Cavitation	Signs of Consolidation	Crepitations	Rhonchi
1	302408	M	36	Mr	D	P	P	A	P	P	P	A	A	A	A	A	P	A	A	A	A	A	A	A	176	46	14.85	P	P	A	A	A	A	P	A	P	A
2	300517	M	58	Mr	Ag	P	A	A	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	168	52	18.42	P	P	A	A	A	A	P	A	P	A
3	303392	M	60	Mr	Ag	P	P	A	A	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	165	46	16.90	A	A	A	A	A	A	A	P	P	A
4	303685	M	48	Mr	L	P	P	P	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	170	58	20.07	A	P	A	A	A	A	A	A	P	A
5	303681	F	42	Mr	HW	P	A	A	P	A	A	A	A	A	A	A	P	A	A	A	A	A	A	A	166	48	17.42	A	A	A	A	A	A	A	A	P	A
6	304245	M	52	Mr	L	P	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	161	66	25.46	A	A	A	A	A	A	P	A	P	A
7	300473	M	51	Mr	L	P	A	P	A	P	P	A	P	A	A	A	A	A	A	P	A	A	A	A	164	40	14.87	P	A	A	A	A	A	P	A	P	A
8	303681	M	47	Mr	D	P	P	A	A	P	P	A	A	A	A	A	A	A	A	A	P	A	A	A	170	50	17.30	P	A	P	A	A	A	A	A	P	A
9	304705	M	25	S	S	P	A	A	A	A	A	A	A	P	A	A	A	A	A	A	A	A	A	A	168	46	16.30	A	A	A	A	A	A	P	A	P	A
10	303940	M	45	Mr	L	P	P	A	A	P	P	A	A	A	A	P	A	P	A	A	A	P	A	A	167	51	18.29	A	A	A	P	A	A	A	A	P	A
11	305104	F	25	W	HW	P	P	A	A	P	A	P	P	A	A	A	A	A	A	A	A	A	P	A	163	46	17.31	P	P	A	A	P	A	A	P	A	A
12	305342	M	30	Mr	L	P	P	P	A	A	A	A	A	A	P	A	A	A	A	A	A	A	A	A	159	38	15.03	A	P	A	A	A	A	P	A	P	A
13	300518	F	18	S	S	P	P	P	A	P	A	A	A	P	P	A	P	A	A	A	A	A	A	A	168	30	10.63	P	P	A	A	A	A	A	A	P	A
14	302790	M	73	Mr	Ag	P	P	A	A	P	P	A	A	A	A	A	A	A	A	P	A	A	A	A	171	68	23.26	P	A	A	A	A	A	A	A	P	A
15	308585	F	28	Mr	HW	P	A	A	A	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	166	48	17.42	P	P	A	A	A	A	P	A	P	A
16	314656	M	19	S	S	P	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	167	28	10.04	P	P	A	A	A	A	A	A	P	A
17	306766	F	50	Mr	HW	P	A	A	A	P	A	A	A	A	P	A	A	A	P	P	A	A	A	A	169	86	30.11	A	P	A	A	A	A	P	A	P	A
18	306254	M	26	S	S	P	A	A	A	P	P	A	A	A	P	A	P	A	A	A	A	A	A	A	165	48	17.63	A	P	A	A	A	A	P	A	P	A
19	308762	F	30	Mr	HW	P	A	A	P	A	A	A	A	A	A	A	P	A	A	A	A	A	A	A	159	44	17.40	A	A	A	A	A	A	A	A	P	A
20	309873	M	45	Mr	Ag	P	A	A	A	P	P	A	A	A	A	A	P	A	A	P	A	A	A	A	164	67	24.91	A	A	A	A	A	A	P	A	P	A
21	309873	M	45	Mr	Ag	P	A	A	P	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	158	56	22.43	A	A	A	A	A	A	P	A	P	A
22	308585	F	34	Mr	HW	P	P	P	A	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	157	36	14.61	A	A	A	A	A	A	P	A	P	A
23	309735	M	35	Mr	D	p	p	A	A	P	P	A	P	A	A	P	A	P	A	A	P	A	A	A	173	45	15.04	P	P	P	P	A	A	A	A	P	A
24	309905	M	46	Mr	D	P	A	P	A	P	P	A	A	A	A	A	A	A	P	P	A	A	A	A	167	38	13.63	P	A	A	A	A	A	P	A	P	A
25	300088	M	23	S	S	P	A	A	A	P	P	A	A	A	P	A	P	A	A	A	A	A	A	A	159	39	15.43	P	P	A	A	A	A	P	A	P	A
26	309724	M	32	Mr	B	P	P	A	A	P	P	A	A	A	A	A	A	A	A	A	A	P	A	A	164	48	17.82	P	P	P	A	P	A	P	A	P	P
27	331592	M	38	Mr	B	P	P	A	A	A	A	A	A	A	A	A	P	A	A	A	A	A	A	A	166	46	16.69	A	A	A	A	A	A	A	A	P	A
28	310071	M	23	S	S	P	P	P	A	P	P	A	A	A	P	A	A	A	A	A	A	A	A	A	154	38	16.02	A	A	A	A	A	A	P	A	P	A
29	312511	M	45	Mr	Ag	P	A	A	A	P	P	A	A	A	A	A	P	A	P	P	A	A	A	A	172	56	18.93	A	A	A	A	A	A	P	A	P	A
30	313836	M	21	S	S	P	P	P	A	P	P	A	P	A	A	A	P	A	A	A	A	A	A	A	158	24	9.61	P	A	A	A	A	A	A	A	P	A
31	317691	M	32	Mr	L	P	P	A	A	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	167	47	16.85	P	A	P	A	A	A	A	P	P	A
32	317974	M	28	Mr	L	P	P	P	A	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	176	44	14.35	P	P	A	A	A	A	A	A	P	A
33	313856	M	33	Mr	L	P	P	P	A	P	P	A	P	A	A	A	P	A	A	A	A	A	A	A	169	28	9.80	P	P	A	A	A	A	A	A	P	A
34	316082	M	55	Mr	B	P	A	A	P	P	P	A	A	A	A	A	A	A	P	A	A	A	A	A	166	56	20.32	A	A	A	A	A	A	A	A	P	A
35	316669	M	60	Mr	Ag	P	P	P	A	P	P	A	A	A	A	A	A	A	P	A	A	A	A	A	169	61	21.36	A	A	A	A	A	A	P	A	P	A
36	315669	F	28	Mr	SE	P	P	A	A	A	P	A	A	A	P	A	A	A	A	A	A	A	A	A	156	38	15.61	P	A	A	A	A	A	P	A	P	A
37	365161	M	38	Mr	Ag	P	P	A	A	A	A	A	A	P	A	A	A	A	A	A	A	A	A	A	162	46	17.53	A	A	A	A	A	A	A	A	P	A
38	318397	M	42	Mr	Ag	P	P	P	A	A	A	A	A	A	A	A	A	A	P	A	A	A	A	A	165	65	23.88	A	A	A	A	A	A	A	A	P	A
39	319723	M	38	Mr	Ag	P	P	A	A	A	P	P	A	A	P	A	A	P	A	A	P	A	A	A	167	49	17.57	P	A	P	P	A	A	A	A	P	A

Sl. No.	Investigations																															
											AFB		CXR																			
													Infiltration						Cavitation						Fibrosis							
													Right			Left			Right			Left			Right			Left				
	Haemoglobin (gm%)	Total count	ESR	HIV ELISA	Urea	Serum creatinine	Serum Sodium	Serum Potassium	Peripherial smear	CD4 Count	Sample 1	Sample 2	Upper	Middle	Lower	Upper	Middle	Lower	Upper	Middle	Lower	Upper	Middle	Lower	Upper	Middle	Lower	Upper	Middle	Lower	Fibrocavitatory lesion	Miliary Pattern
1	8.0	7800	102	NR	18	1.0	140	5.0	MHA	-	2+	3+	-	-	-	+	-	-	+	-	-	-	-	-	+	-	-	-	-	-	P	A
2	9.8	11100	78	NR	40	1.1	151	5.4	NHA	-	1+	2+	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	P	A
3	12.2	10400	48	NR	32	1.1	134	2.9	NHA	-	SC	1+	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	A	A
4	13.0	5050	108	NR	34	1.4	137	3.8	NHA	-	1+	2+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	A	A
5	13.3	14000	62	NR	30	0.9	132	3.0	NHA	-	1+	2+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
6	10.0	12200	78	NR	20	1.0	138	3.4	NHA	-	1+	2+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	P	A
7	10.3	18200	104	NR	42	1.4	138	4.0	NHA	-	1+	2+	-	-	-	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	A	A
8	8.0	7895	45	R	31	0.4	137	3.2	DA	180	0	1+	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
9	10.0	13600	23	NR	30	0.9	136	2.9	NHA	-	1+	2+	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	P	A
10	8.9	8142	86	R	24	1.0	142	3.8	NHA	220	SC	SC	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
11	7.7	8623	46	R	40	1.1	138	4.0	DA	114	1+	1+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
12	9.0	18000	48	NR	86	2.0	142	5.2	NHA	-	1+	2+	+	-	-	+	-	+	+	-	-	-	-	+	+	-	-	-	-	-	P	A
13	8.9	12600	128	NR	16	0.6	145	3.5	NHA	-	SC	1+	+	-	-	-	-	-	-	-	-	-	-	+	-	-	-	+	-	-	P	A
14	9.7	8000	114	NR	21	0.8	138	3.8	NHA	-	2+	3+	+	-	+	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	A	A
15	9.6	14700	63	NR	24	0.7	143	3.7	MHA	-	2+	3+	-	+	-	+	-	+	-	-	-	-	+	-	-	-	-	-	+	-	P	A
16	10.0	7600	68	NR	32	1.0	138	3.3	NHA	-	1+	2+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
17	9.0	13000	101	NR	46	1.4	140	3.4	NBP	-	SC	1+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	+	P	A
18	10.9	16000	64	NR	34	1,3	137	3.9	NBP	-	1+	2+	+	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	P	A
19	13.0	6800	80	NR	24	0.8	138	3.9	NBP	-	1+	1+	+	-	+	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	A	A
20	10.0	17100	72	NR	33	1.6	147	4.3	NBP	-	3+	3+	+	-	-	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	P	A
21	12,6	15600	60	NR	22	0.6	142	4.1	NBP	-	1+	2+	-	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	P	A
22	11.4	16330	72	NR	34	1.4	135	3.8	NBP	-	1+	2+	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	P	A
23	5.5	3000	112	R	28	0.6	139	4.4	DA	86	0	SC	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	P
24	9.3	4566	102	NR	86	2.1	151	5.8	NBP	-	1+	1+	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	P	A
25	7.5	12560	116	NR	43	1.3	140	3.9	DA	-	1+	1+	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	+	-	-	P	A
26	8.2	5650	39	R	25	0.7	144	3.7	NBP	117	SC	SC	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-	-	P	A
27	11.4	16300	68	NR	36	1.2	142	4.1	NBP	-	1+	1+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
28	9.6	8600	68	NR	24	1.0	147	4.8	NBP	-	1+	2+	-	-	-	+	-	-	-	-	-	-	+	-	-	-	-	-	-	-	A	A
29	10.2	15390	102	NR	46	2.0	138	5.0	NBP	-	1+	2+	-	+	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	P	A
30	7.2	4500	86	NR	22	0.9	139	3.7	DA	-	1+	1+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
31	8.0	3900	88	R	16	1.0	125	3.4	MHA	35	1+	SC	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	A	A
32	8.0	6060	111	NR	86	1.2	130	3.6	MHA	-	1+	1+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	A	A
33	6.8	18800	44	NR	37	1.8	146	4.6	DA	-	2+	3+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
34	9.9	6800	102	NR	23	1.0	140	3.4	NBP	-	2+	2+	+	-	-	+	-	-	-	-	-	-	+	-	-	-	-	-	-	-	A	A
35	10.9	8458	68	NR	34	1.0	142	4.0	NBP	-	1+	2+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
36	9.0	21000	78	NR	46	1.4	137	3.8	NBP	-	1+	2+	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	P	A
37	13.5	12400	28	NR	15	0.8	136	4.4	NBP	-	1+	1+	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
38	11.5	15000	48	NR	24	0.6	142	3.5	NBP	-	1+	1+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
39	7.3	7980	62	R	21	0.9	126	3.0	DA	46	SC	1+	-	+	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	A	A

ANNEXURE III - MASTER CHART

Sl. No.	IP. No.	Sex	Age (Years)	Marital Status	Occupation	Symptoms: Cough with expectoration	Fever	Night Sweats	Haemoptysis	Weight loss > 10%	Loss of appetite	Diarrhoea	Chest pain	Breathlessness	Generalised weakness	Past History: HIV status	Contact with tuberculosis	Herpez Zoster	Hypertension	Diabetes mellitus	Risk factors for HIV: Multiple sexual partners	H/o Blood transfusion	Sexually transmitted disease	Intrvenous drug abuse	General Physical Examination: Height (Cms)	Weight (Kg)	Body mass index (Kg/m²)	Pallor	Lymphadenopathy	Oral candidiasis	Herpetic Scar	Genital lesions	Cyanosis	Systemic examination: Signs of Cavitation	Signs of Consolidation	Crepitations	Rhonchi
40	321642	F	32	Mr	HW	P	A	A	A	P	P	A	A	A	P	A	A	A	A	A	A	A	A	A	162	58	22.10	P	P	A	A	A	A	A	A	P	A
41	321645	F	46	Mr	HW	P	A	A	A	P	P	A	A	A	P	A	A	A	P	A	A	A	A	A	156	52	21.37	P	A	A	A	A	A	A	A	P	P
42	365128	F	16	S	S	P	P	A	A	P	P	A	A	A	P	A	A	A	A	A	A	A	A	A	152	38	16.45	P	P	A	A	A	A	P	A	P	P
43	323538	M	56	Mr	Ag	P	P	A	A	A	A	A	A	A	A	A	A	A	P	P	A	A	A	A	164	58	21.56	A	A	A	A	A	A	A	P	P	A
44	323266	M	48	Mr	Ag	P	P	A	A	P	P	A	A	A	P	A	A	A	P	P	A	A	A	A	160	66	25.78	P	A	P	A	A	A	A	P	P	P
45	322538	F	14	S	S	P	P	A	A	A	A	A	P	A	P	A	P	P	A	A	A	A	A	A	148	20	9.13	A	A	A	P	A	A	A	P	P	P
46	323581	M	48	Mr	L	P	P	P	A	P	P	A	A	A	P	A	P	P	A	A	A	A	A	A	168	38	13.46	P	P	P	P	A	A	P	A	P	A
47	323582	M	46	Mr	L	P	P	A	A	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	164	58	21.56	P	P	A	A	A	A	P	A	P	A
48	323448	F	28	Mr	Sr	P	A	A	A	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	156	48	19.72	A	P	A	A	A	A	P	A	P	A
49	324908	M	40	Mr	SE	P	A	A	A	P	A	A	A	A	P	A	A	A	A	A	A	A	A	A	170	48	16.61	A	A	A	A	A	A	A	P	P	A
50	324108	M	36	Mr	B	P	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	164	38	14.86	A	P	A	A	A	A	A	A	P	A
51	325476	F	55	Mr	HW	P	P	P	A	P	P	A	A	A	P	A	A	A	A	A	A	A	A	A	165	61	22.41	P	A	A	A	A	A	A	A	P	A
52	325879	F	28	Mr	HW	P	P	A	A	A	A	A	P	A	A	A	A	A	A	A	A	A	A	A	162	38	14.48	A	A	A	A	A	A	A	A	P	A
53	331459	F	34	W	HW	P	P	A	A	P	P	P	A	A	P	A	A	P	A	A	A	A	A	A	158	28	11.22	P	P	P	P	A	A	A	A	P	A
54	334623	M	43	Mr	Ag	P	A	A	A	P	P	A	A	A	P	A	A	A	P	A	A	A	A	A	170	48	16.61	P	P	A	A	A	A	P	A	P	A
55	328629	F	32	Mr	HW	P	P	A	A	P	P	A	A	A	P	A	P	A	A	A	A	A	A	A	163	47	17.69	P	A	A	A	A	A	A	A	P	P
56	329830	M	26	Mr	S	P	P	P	A	P	P	A	A	P	P	A	A	A	A	A	A	A	A	A	160	36	14.06	P	A	A	A	A	A	A	A	P	A
57	329840	M	55	Mr	SE	P	P	A	A	A	A	A	A	A	P	A	A	A	P	A	A	A	A	A	168	56	19.84	P	A	A	A	A	A	A	A	P	A
58	325632	M	53	Mr	L	P	P	A	A	A	P	A	A	A	A	A	A	A	P	P	A	A	A	A	171	62	21.2	A	A	A	A	A	A	A	A	P	A
59	331618	M	35	Mr	D	P	P	A	A	P	P	A	A	A	A	A	A	P	A	A	P	A	P	A	168	49	17.36	P	A	P	P	P	A	A	A	A	A
60	372419	F	32	Mr	L	P	P	P	A	P	P	A	A	A	P	P	A	P	A	A	A	P	A	A	166	46	16.69	P	A	P	P	A	A	P	A	P	A
61	336426	M	47	Mr	SE	P	P	A	A	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	172	38	12.84	P	P	A	A	A	A	A	P	P	P
62	316906	M	45	Mr	Ag	P	P	A	A	P	P	A	A	A	P	A	A	A	A	A	A	P	A	A	168	46	16.3	A	A	P	A	A	A	A	P	A	A
63	331925	M	38	Mr	Ag	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	166	48	17.42	A	A	A	A	A	A	P	A	P	A
64	334112	F	18	S	S	P	P	A	A	P	P	A	A	A	P	A	A	A	A	A	A	A	A	A	156	32	13.15	A	A	A	A	A	A	A	P	P	P
65	365128	F	44	Mr	HW	P	P	A	A	P	P	A	A	A	P	A	P	A	A	A	A	A	A	A	169	40	14.01	P	A	P	A	A	A	P	A	P	A
66	333360	M	58	Mr	Ag	P	P	A	A	P	P	A	A	A	P	A	A	A	P	A	A	A	A	A	164	51	18.96	P	A	A	A	A	A	P	A	P	A
67	334635	M	51	Mr	D	P	P	A	A	P	P	A	A	A	P	P	A	A	P	P	A	P	A	A	166	51	18.51	P	A	P	A	A	A	P	A	P	A
68	335219	M	35	Mr	B	P	P	A	A	P	P	A	A	A	P	A	A	P	A	A	P	A	P	A	170	40	13.84	A	A	P	P	P	A	P	A	P	A
69	335203	F	24	Mr	S	P	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	154	58	24.46	A	A	A	A	A	A	A	A	P	A
70	335370	M	28	Mr	L	P	P	A	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	160	46	17.97	A	A	A	A	A	A	A	A	P	A
71	334097	M	45	Mr	Ag	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	168	46	16.3	A	A	A	A	A	A	A	A	P	P
72	337365	M	38	Mr	B	P	P	A	A	P	P	A	A	A	P	A	A	A	A	A	P	A	A	A	172	38	12.84	A	A	P	A	A	A	A	A	P	A
73	337716	M	56	Mr	B	P	P	A	A	P	P	A	A	A	P	P	A	A	A	A	P	A	A	A	170	50	17.3	A	A	P	A	A	A	A	A	A	A
74	337497	F	43	Mr	HW	P	P	A	A	P	A	A	A	A	A	P	A	A	A	A	P	A	A	A	169	46	16.11	A	A	A	A	A	A	A	A	P	A
75	335905	M	51	Mr	SE	P	P	A	A	A	A	A	A	A	A	A	P	A	P	A	A	A	A	A	166	54	19.6	A	A	A	A	A	A	A	A	P	A
76	337769	M	31	Mr	L	P	P	A	A	A	A	A	A	A	A	P	A	P	A	A	P	A	A	A	169	38	13.3	A	A	A	P	A	A	A	A	P	A
77	337979	F	41	Mr	SE	P	P	A	A	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	164	48	17.85	A	A	A	A	A	A	P	A	A	A
78	339285	M	38	Mr	L	P	A	A	A	A	A	A	A	A	P	A	A	P	A	A	A	A	A	A	170	51	17.65	A	A	P	P	A	A	A	A	P	A

Sl. No.	Investigations: Haemoglobin (gm%)	Total count	ESR	HIV ELISA	Urea	Serum creatinine	Serum Sodium	Serum Potassium	Peripherial smear	CD4 Count	AFB: Sample 1	AFB: Sample 2	CXR Infiltration Right: Upper	Middle	Lower	Infiltration Left: Upper	Middle	Lower	Cavitation Right: Upper	Middle	Lower	Cavitation Left: Upper	Middle	Lower	Fibrosis Right: Upper	Middle	Lower	Fibrosis Left: Upper	Middle	Lower	Fibrocavitatory lesion	Miliary Pattern
40	9.2	12400	102	NR	18	0.8	138	3.3	DA	-	2+	3+	-	+	-	-	+	-	-	-	-	-	-	-	+	-	-	-	-	-	A	A
41	8.0	10000	68	NR	16	1.0	138	4.1	MHA	-	2+	2+	+	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
42	9.0	13400	72	NR	20	0.8	143	4.0	MHA	-	2+	3+	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	P	A
43	10.3	16300	32	NR	22	0.8	140	5.0	NBP	-	1+	1+	+	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
44	8.2	17600	44	NR	86	2.1	151	5.4	DA	-	2+	2+	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
45	13.8	16300	112	NR	26	1.4	134	2.9	NBP	-	1+	2+	+	+	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	A	A
46	7.0	4400	72	R	43	1.0	137	3.8	DA	186	1+	1+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
47	9.4	11000	42	NR	26	1.0	132	3.0	NBP	-	1+	1+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	A	A
48	10.0	6600	48	NR	18	0.8	138	3.4	NBP	-	1+	1+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	P	A
49	10.2	13400	60	NR	17	1.3	138	4.0	NBP	-	SC	1+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
50	10.2	10200	66	NR	18	1.0	137	3.2	NBP	-	1+	2+	-	+	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	A	A
51	9.4	14500	48	NR	24	0.8	138	3.6	NBP	-	1+	1+	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	A	A
52	11	14,600	68	NR	16	0.8	140	3	NBP	-	1+	1+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
53	9	8,980	39	R	60	1.5	138	3.9	NBP	311	0	1+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	P
54	16	15300	68	NR	18	1	142	4	NBP	-	1+	1+	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	P	A
55	8	6,800	102	NR	16	0.8	142	4.1	DA	-	2+	3+	+	+	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	A	A
56	9.6	12,800	46	NR	18	1	136	3.8	NBP	-	2+	3+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	P
57	10	18,000	36	NR	42	1.2	147	4.1	NBP	-	1+	2+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
58	9	14,800	28	NR	16	0.4	137	3.3	NBP	-	1+	2+	+	+	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	A	A
59	7	2,950	56	R	20	0.6	137	2.9	DA	21	SC	1+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
60	9	9,051	43	R	46	1.2	130	3	MHA	397	SC	SC	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
61	8.2	10,400	40	NR	16	1	137	3.7	MHA	-	1+	2+	+	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
62	8.9	4,600	44	R	24	0.9	130	3.6	NBP	87	1+	1+	-	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
63	12	12,300	60	NR	33	1	136	4.1	NBP	-	1+	1+	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	P	A
64	11	21,600	48	NR	86	1.8	128	3.3	NBP	-	2+	3+	+	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
65	8	12,800	100	NR	86	1.6	148	5.2	DA	-	2+	2+	+	-	-	+	-	-	-	-	+	-	-	-	-	-	+	-	-	-	P	A
66	9	10,400	70	NR	46	1.1	138	3.4	NBP	-	1+	1+	+	+	-	+	-	-	-	-	-	-	-	+	-	-	-	-	-	+	P	A
67	8	7,800	68	R	56	1	137	2.9	DA	259	SC	1+	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	+	P	A
68	8.4	4,100	102	R	36	0.6	145	3.8	NBP	217	1+	2+	-	+	-	-	-	-	-	-	+	-	-	+	-	-	-	-	-	-	A	A
69	12	16,500	34	NR	20	0.8	138	4.5	NBP	-	1+	1+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
70	12	14,000	32	NR	22	1	140	3.7	NBP	-	SC	1+	+	+	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	A	A
71	5.6	6,800	44	NR	42	1	132	3	DA	-	1+	1+	+	+	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	A	A
72	8.3	8,047	46	R	30	1	134	3	NBP	64	1+	1+	-	+	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	-	A	A
73	8.6	8,303	56	R	18	1	148	5	NBP	448	0	SC	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
74	8.9	7,644	101	R	43	1.4	137	4	NBP	84	1+	1+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	P
75	10	6,400	34	NR	20	0.9	135	3.2	NBP	-	1+	1+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
76	8.1	8,445	22	R	40	1.1	140	4	NBP	111	1+	1+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
77	8.2	6,900	44	R	24	0.9	133	4.2	NBP	121	1+	1+	-	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	P	A
78	9	9,201	36	R	26	0.7	137	3.1	NBP	151	0	1+	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A

ANNEXURE III - MASTER CHART

Sl. No.	IP. No.	Sex	Age (Years)	Marital Status	Occupation	Symptoms: Cough with expectoration	Fever	Night Sweats	Haemoptysis	Weight loss > 10%	Loss of appetite	Diarrhoea	Chest pain	Breathlessness	Generalised weakness	Past History: HIV status	Contact with tuberculosis	Herpez Zoster	Hypertension	Diabetes mellitus	Risk factors for HIV: Multiple sexual partners	H/o Blood transfusion	Sexually transmitted disease	Intrvenous drug abuse	General Physical Examination: Height (Cms)
79	338038	M	55	Mr	Ag	P	P	A	P	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	168
80	337958	M	36	Mr	Ag	P	P	A	A	P	P	A	A	A	A	A	A	A	A	A	P	A	A	A	164
81	338998	M	35	Mr	D	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	172
82	339095	F	48	Mr	SE	P	A	A	A	P	A	A	A	A	A	A	A	A	P	P	A	A	A	A	167
83	339630	M	50	Mr	Ag	P	P	A	A	P	A	A	A	A	A	A	A	P	A	A	A	A	A	A	168
84	323212	M	36	Mr	Ag	p	p	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	173
85	323826	M	55	Mr	Ag	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	166
86	323764	M	21	S	S	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	165
87	339030	M	33	Mr	L	P	P	A	A	A	P	A	A	A	P	A	A	A	A	A	A	A	A	A	168
88	333266	M	38	Mr	L	P	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	173
89	333212	M	41	Mr	L	P	P	P	A	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	166
90	330701	F	35	Mr	HW	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	164
91	344300	M	55	Mr	Ag	P	P	A	A	A	A	A	A	A	P	P	A	A	A	A	A	P	A	A	172
92	365121	F	19	S	S	P	P	A	A	A	P	A	A	A	P	A	A	A	A	A	A	A	A	A	162
93	334142	M	27	Mr	Ag	P	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	170
94	332486	M	32	Mr	D	P	A	A	A	A	P	A	A	A	P	A	A	A	A	A	A	A	A	A	166
95	335160	M	18	S	S	P	A	A	A	P	P	A	A	A	P	A	A	A	A	A	A	A	A	A	158
96	336799	M	36	Mr	Ag	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	168
97	335478	M	41	Mr	SE	P	A	A	A	P	P	A	A	A	P	A	P	A	A	A	A	A	A	A	166
98	337254	M	40	Mr	SE	P	P	P	A	A	A	A	A	A	P	A	A	A	A	A	A	A	A	A	166
99	344994	M	51	Mr	SE	P	P	A	A	P	P	A	A	A	A	A	A	P	A	A	P	A	A	A	172
100	333452	M	36	Mr	L	p	p	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	173
101	333676	M	55	Mr	L	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	166
102	323986	M	21	S	Sr	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	165
103	339042	M	33	Mr	Ag	P	P	A	A	A	P	A	A	A	P	A	A	A	A	A	A	A	A	A	168
104	333261	M	38	Mr	Ag	P	P	P	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	A	173

Sl. No.	Weight (Kg)	Body mass index (Kg/m^2)	Pallor	Lymphadenopathy	Oral candidiasis	Herpetic Scar	Genital lesions	Cyanosis	Systemic examination: Signs of Cavitation	Signs of Consolidation	Crepitations	Rhonchi	Investigations: Haemoglobin (gm%)	Total count	ESR	HIV ELISA	Urea	Serum creatinine	Serum Sodium	Serum Potassium	Peripherial smear	CD4 Count
79	58	20.55	P	A	A	A	A	A	P	A	P	A	10	11,400	36	NR	26	1.2	136	3	NBP	-
80	38	14.13	A	A	A	A	A	A	A	A	P	A	8.9	8,067	40	R	46	1.6	136	4.9	NBP	106
81	42	14.2	A	A	A	A	A	A	P	A	P	A	12	15,600	36	NR	46	1.1	138	3.6	NBP	-
82	68	24.38	P	A	A	A	A	A	P	A	P	A	10	16,700	36	NR	48	1.2	138	4.5	NBP	-
83	48	17.01	P	P	A	P	A	A	P	A	P	A	9	8,210	48	R	46	1.2	146	4	NBP	179
84	52	17.37	P	A	A	A	A	A	P	A	P	A	8	8,900	36	NR	32	1.1	137	3.7	DA	-
85	38	13.79	P	A	A	A	A	A	A	A	p	A	6	11,200	48	NR	24	0.8	138	3.6	DA	-
86	38	13.96	A	A	A	A	A	A	A	A	P	A	10	11,600	32	NR	12	0.7	129	3.6	NBP	-
87	42	14.88	A	P	A	A	A	A	A	A	P	A	11	13,600	38	NR	18	0.6	137	4	NBP	-
88	54	18.04	A	A	A	A	A	A	P	A	P	P	12	16,700	66	NR	20	0.9	134	3.6	NBP	-
89	52	18.87	P	A	A	A	A	A	A	A	P	A	10	8,600	72	NR	16	0.8	138	4.1	NBP	-
90	33	12.27	A	A	A	A	A	A	A	A	A	A	11	12,700	44	NR	18	0.6	140	3.8	NBP	-
91	40	13.52	A	P	A	A	A	A	A	A	A	A	9	8,600	38	R	16	0.8	136	3.9	NBP	147
92	38	14.48	A	A	A	A	A	A	A	A	P	A	13	16,000	28	NR	26	0.9	137	3.5	NBP	-
93	41	14.19	A	A	A	A	A	A	A	A	A	A	10	11,260	44	NR	17	0.8	138	3.3	NBP	-
94	43	15.6	A	P	A	A	A	A	A	A	A	A	11	14,600	32	NR	16	0.9	140	3.8	NBP	-
95	46	18.43	P	A	A	A	A	A	A	A	A	A	8	4,600	88	NR	20	0.8	139	3.8	DA	-
96	54	19.13	A	A	A	A	A	A	A	A	A	A	11	12,600	44	NR	16	0.9	140	4	NBP	-
97	44	15.97	P	A	A	A	A	A	P	A	A	A	9	10,800	46	NR	26	1	141	4	NBP	-
98	52	18.87	A	A	A	A	A	A	A	A	A	A	14	14,690	44	NR	18	1	138	4	NBP	-
99	34	11.49	P	P	P	P	A	A	A	A	A	A	7	8,452	100	R	16	0.9	136	3.9	DA	460
100	52	17.37	P	A	A	A	A	A	P	A	P	A	8	8,900	36	NR	32	1.1	137	3.7	DA	-
101	38	13.79	P	A	A	A	A	A	A	A	p	A	6	11,200	48	NR	24	0.8	138	3.6	DA	-
102	38	13.96	A	A	A	A	A	A	A	A	P	A	10	11,600	32	NR	12	0.7	129	3.6	NBP	-
103	42	14.88	A	P	A	A	A	A	A	A	P	A	11	13,600	38	NR	18	0.6	137	4	NBP	-
104	54	18.04	A	A	A	A	A	A	P	A	P	P	12	16,700	66	NR	20	0.9	134	3.6	NBP	-

Sl. No.	AFB Sample 1	AFB Sample 2	CXR Infiltration Right Upper	Middle	Lower	Infiltration Left Upper	Middle	Lower	Cavitation Right Upper	Middle	Lower	Cavitation Left Upper	Middle	Lower	Fibrosis Right Upper	Middle	Lower	Fibrosis Left Upper	Middle	Lower	Fibrocavitatory lesion	Miliary Pattern
79	2+	2+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	A	A
80	1+	2+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
81	1+	1+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	P	A
82	1+	2+	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	+	-	P	A
83	1+	1+	-	-	+	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	P	A
84	1+	2+	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	P	A
85	1+	2+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
86	2+	2+	+	+	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
87	1+	2+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
88	1+	2+	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	P	A
89	2+	2+	+	+	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
90	1+	2+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
91	1+	1+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	P	A
92	1+	1+	+	+	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	A	A
93	2+	2+	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
94	2+	2+	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
95	2+	2+	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
96	2+	1+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
97	1+	1+	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	P	A
98	1+	1+	+	+	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
99	1+	1+	+	+	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	A	A
100	1+	2+	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	P	A
101	1+	2+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
102	2+	2+	+	+	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
103	1+	2+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	A	A
104	1+	2+	-	-	-	-	-	-	+	-	-	-	-	-	+	-	-	-	-	-	P	A

Group	Height	Weight	BMI	Hb	TLC
NR	176	46	14.85	8.0	7800
NR	168	52	18.42	9.8	11100
NR	165	46	16.90	12.2	10400
NR	170	58	20.07	13.0	5050
NR	166	48	17.42	13.3	14000
NR	161	66	25.46	10.0	12200
NR	164	40	14.87	10.3	18200
R	170	50	17.30	8.0	7895
NR	168	46	16.30	10.0	13600
R	167	51	18.29	8.9	8142
R	163	46	17.31	7.7	8623
NR	159	38	15.03	9.0	18000
NR	168	30	10.63	8.9	12600
NR	171	68	23.26	9.7	8000
NR	166	48	17.42	9.6	14700
NR	167	28	10.04	10.0	7600
NR	169	86	30.11	9.0	13000
NR	165	48	17.63	10.9	16000
NR	159	44	17.40	13.0	6800
NR	164	67	24.91	10.0	17100
NR	158	56	22.43	12,6	15600
NR	157	36	14.61	11.4	16330
R	173	45	15.04	5.5	3000
NR	167	38	13.63	9.3	4566
NR	159	39	15.43	7.5	12560
R	164	48	17.82	8.2	5650
NR	166	46	16.69	11.4	16300
NR	154	38	16.02	9.6	8600
NR	172	56	18.93	10.2	15390
NR	158	24	9.61	7.2	4500
R	167	47	16.85	8.0	3900
NR	176	44	14.35	8.0	6060
NR	169	28	9.80	6.8	18800
NR	166	56	20.32	9.9	6800
NR	169	61	21.36	10.9	8458
NR	156	38	15.61	9.0	21000
NR	162	46	17.53	13.5	12400
NR	165	65	23.88	11.5	15000
R	167	49	17.57	7.3	7980
NR	162	58	22.10	9.2	12400
NR	156	52	21.37	8.0	10000
NR	152	38	16.45	9.0	13400
NR	164	58	21.56	10.3	16300
NR	160	66	25.78	8.2	17600
NR	148	20	9.13	13.8	16300
R	168	38	13.46	7.0	4400
NR	164	58	21.56	9.4	11000
NR	156	48	19.72	10.0	6600
NR	170	48	16.61	10.2	13400
NR	164	38	14.86	10.2	10200
NR	165	61	22.41	9.4	14500
NR	162	38	14.48	11.4	14,600
R	158	28	11.22	9	8,980
NR	170	48	16.61	16	15300

NR	163	47	17.69	8	6,800
NR	160	36	14.06	9.6	12,800
NR	168	56	19.84	10	18,000
NR	171	62	21.20	9	14,800
R	168	49	17.36	7	2,950
R	166	46	16.69	9	9,051
NR	172	38	12.84	8.2	10,400
R	168	46	16.30	8.9	4,600
NR	166	48	17.42	12	12,300
NR	156	32	13.15	11	21,600
NR	169	40	14.01	8	12,800
NR	164	51	18.96	9	10,400
R	166	51	18.51	8	7,800
R	170	40	13.84	8.4	4,100
NR	154	58	24.46	12	16,500
NR	160	46	17.97	11.6	14,000
NR	168	46	16.30	5.6	6,800
R	172	38	12.84	8.3	8,047
R	170	50	17.30	8.6	8,303
R	169	46	16.11	8.9	7,644
NR	166	54	19.60	10	6,400
R	169	38	13.30	8.1	8,445
R	164	48	17.85	8.2	6,900
R	170	51	17.65	9	9,201
NR	168	58	20.55	10.2	11,400
R	164	38	14.13	8.9	8,067
NR	172	42	14.20	12	15,600
NR	167	68	24.38	10.2	16,700
R	168	48	17.01	9	8,210
NR	173	52	17.37	8	8,900
NR	166	38	13.79	6	11,200
NR	165	38	13.96	10.4	11,600
NR	168	42	14.88	11	13,600
NR	173	54	18.04	12	16,700
NR	166	52	18.87	10	8,600
NR	164	33	12.27	11	12,700
R	172	40	13.52	9	8,600
NR	162	38	14.48	13	16,000
NR	170	41	14.19	10.2	11,260
NR	166	43	15.60	11	14,600
NR	158	46	18.43	8	4,600
NR	168	54	19.13	11	12,600
NR	166	44	15.97	9	10,800
NR	166	52	18.87	14.2	14,690
R	172	34	11.49	7	8,452
NR	173	52	17.37	8	8,900
NR	166	38	13.79	6	11,200
NR	165	38	13.96	10.4	11,600
NR	168	42	14.88	11	13,600
NR	173	54	18.04	12	16,700

Right			Left				Right			Left				Right			Left		
Upper	Middle	Lower	Upper	Middle	Lower	Infiltration	Upper	Middle	Lower	Upper	Middle	Lower	Cavitation	Upper	Middle	Lower	Upper	Middle	Lower
-	-	-	+	-	-	LU	+	-	-	-	-	-	RU	+	-	-	-	-	-
-	-	-	-	-	-	A	+	-	-	-	-	-	RU	+	-	-	-	-	-
-	-	-	+	-	+	UL	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	-	-	-	-	-	-	A	+	-	-	-	-	-
+	+	-	-	-	-	RU,RM	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	-	-	-	+	-	-	LU	-	-	-	+	-	-
-	-	-	-	-	-	A	+	-	+	-	-	-	RU,RL	-	-	-	-	-	-
-	-	+	+	-	-	UL,RL	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	+	-	-	-	-	-	RU	+	-	-	-	-	-
+	-	+	-	-	-	RU,RL	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	+	-	LM	-	-	-	-	-	-	A	-	-	-	-	-	-
+	-	-	+	-	+	RU,LU,LL	+	-	-	-	-	+	RU,LL	+	-	-	-	-	-
+	-	-	-	-	-	RU	-	-	-	-	-	+	LL	-	-	-	+	-	-
+	-	+	+	-	+	BU,BL	-	-	-	-	-	-	A	-	-	-	-	-	-
-	+	-	+	-	+	RM,LU,LL	-	-	-	-	+	-	LM	-	-	-	-	+	-
-	+	-	-	-	-	RM	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	-	-	-	-	-	+	LL	-	-	-	-	-	+
+	-	-	-	-	-	RU	-	+	-	-	-	-	RM	-	+	-	-	-	-
+	-	+	+	-	+	BLU	-	-	-	-	-	-	A	-	-	-	-	-	-
+	-	-	-	-	-	RU	-	-	-	+	-	-	LU	-	-	-	+	-	-
-	-	-	-	-	-	A	-	+	-	-	-	-	RM	-	+	-	-	-	-
-	-	-	-	-	-	A	+	-	-	-	-	-	RU	+	-	-	-	-	-
-	-	-	-	-	-	A	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	+	-	-	-	-	-	RU	+	-	-	-	-	-
-	-	-	-	-	-	A	-	-	-	-	+	-	LM	+	-	-	+	-	-
-	-	-	-	-	-	A	-	+	-	-	-	-	RM	+	+	-	-	-	-
+	-	-	-	-	-	RU	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	+	-	-	LU	-	-	-	-	+	-	LM	-	-	-	-	-	-
-	+	-	-	-	-	RM	+	-	-	-	-	-	RU	+	-	-	-	-	-
+	-	-	-	-	-	RU	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	+	-	LL	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	-	-	-	-	-	-	A	-	-	-	+	-	-
+	-	-	-	-	-	RU	-	-	-	-	-	-	A	-	-	-	-	-	-
+	-	-	+	-	-	BU	-	-	-	-	+	-	LM	-	-	-	-	-	-
-	+	-	-	-	-	RM	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	+	-	-	-	-	-	RU	+	-	-	-	-	-

+	-	-	+	-	-	BU	-	-	-	-	-	-	A	-	-	-	-	-	-
+	-	-	-	-	-	RU	-	-	-	-	-	-	A	-	-	-	-	-	-
-	+	+	-	-	+	RM,BL	-	-	-	-	-	-	A	-	-	-	-	-	-
-	+	-	-	+	-	BM	-	-	-	-	-	-	A	+	-	-	-	-	-
+	+	+	+	+	-	BU,BM,RL	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	+	-	-	-	-	-	RU	+	-	-	-	-	-
+	+	-	+	-	-	BU,RM	-	-	-	-	-	-	A	-	-	-	-	-	-
+	-	-	-	+	-	RU,LM	-	-	-	-	-	-	A	-	-	-	-	-	-
+	+	+	+	+	+	BU,BM,BL	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	-	+	-	-	-	-	RM	-	-	-	-	-	-
-	-	-	-	-	-	A	-	-	-	+	-	-	LU	-	-	-	-	-	-
-	-	-	-	-	-	A	-	-	-	+	-	-	LU	-	-	-	+	-	-
+	-	-	-	-	-	RU	-	-	-	-	-	-	A	-	-	-	-	-	-
-	+	-	-	-	-	RM	-	-	-	-	-	-	A	+	-	-	-	-	-
-	-	-	-	-	-	A	-	-	-	-	-	-	P	+	-	-	-	-	-
+	-	-	-	-	-	RU	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	+	-	-	-	-	-	RU	+	-	-	-	-	-
+	+	+	+	+	+	BUML	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	+	-	LM	-	-	-	-	-	-	A	-	-	-	-	-	-
+	+	+	+	+	+	BUML	-	-	-	-	-	-	A	-	-	-	-	-	-
-	+	-	-	-	-	RM	-	-	-	-	-	-	A	-	-	-	-	-	-
+	-	-	+	-	-	BU	-	-	+	-	-	-	RL	-	-	-	-	-	-
+	+	-	-	+	-	BM,RU	-	-	-	-	-	-	A	-	-	-	-	-	-
-	+	-	-	+	-	BM	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	+	-	-	-	-	-	RU	+	-	-	-	-	-
+	+	-	+	-	-	BU,RM	-	-	-	-	-	-	A	-	-	-	-	-	-
+	-	-	+	-	-	BU	-	-	+	-	-	-	RL	-	-	+	-	-	-
+	+	-	+	-	-	BU, RM	-	-	-	-	-	+	LL	-	-	-	-	-	+
-	-	-	-	-	+	LL	-	-	-	-	-	+	LL	-	-	-	-	-	+
-	+	-	-	-	-	RM	-	-	+	-	-	+	BL	-	-	-	-	-	-
+	-	-	-	-	-	RU	-	-	-	-	-	-	A	-	-	-	-	-	-
+	+	+	+	+	+	BUML	-	-	-	-	-	-	A	-	-	-	-	-	-
+	+	+	+	+	+	BUML	-	-	-	-	-	-	A	-	-	-	-	-	-
-	+	-	-	-	-	RM	-	+	+	-	-	-	RM,RL	-	-	-	-	-	-
+	+	-	-	-	-	RUM	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	-	-	-	-	-	-	A	-	-	-	-	-	-
+	+	-	-	-	-	RU,RM	-	-	-	-	-	-	A	-	-	-	-	-	-
+	+	-	-	-	-	RU,RM	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	-	+	-	-	-	-	RM	-	+	-	-	-	-

-	-	-	+	+	-	LU,LM	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	-	-	-	-	-	-	A	-	-	-	+	-	-
-	+	-	-	-	-	RM	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	-	-	-	+	-	-	LU	-	-	-	+	-	-
-	-	-	-	-	-	A	-	-	-	-	+	-	LM	-	-	-	-	+	-
-	-	+	-	-	-	RL	-	+	-	-	-	-	RM	-	+	-	-	-	-
-	-	-	-	-	-	A	+	-	-	-	-	-	RU	+	-	-	-	-	-
+	-	-	-	-	-	RU	-	-	-	-	-	-	A	-	-	-	-	-	-
+	+	-	+	+	-	BUM	-	-	-	-	-	-	A	-	-	-	-	-	-
+	+	-	-	-	-	RU,RM	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	+	-	-	-	-	-	RU	+	-	-	-	-	-
+	+	-	+	+	-	BUM	-	-	-	-	-	-	A	-	-	-	-	-	-
+	-	-	-	-	-	RU	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	-	-	-	+	-	-	LU	-	-	-	+	-	-
+	+	+	+	+	+	BUML	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	+	+	-	LU,LM	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	+	+	-	LU,LM	-	-	-	-	-	-	A	-	-	-	-	-	-
+	-	-	+	-	-	BU	-	-	-	-	-	-	A	-	-	-	-	-	-
+	-	-	-	-	-	RU	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	+	-	-	-	-	-	RU	+	-	-	-	-	-
+	+	-	+	+	-	BUM	-	-	-	-	-	-	A	-	-	-	-	-	-
+	+	+	+	+	+	BUML	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	+	-	-	-	-	-	RU	+	-	-	-	-	-
+	-	-	-	-	-	RU	-	-	-	-	-	-	A	-	-	-	-	-	-
+	+	-	+	+	-	BUM	-	-	-	-	-	-	A	-	-	-	-	-	-
+	+	-	-	-	-	RU,RM	-	-	-	-	-	-	A	-	-	-	-	-	-
-	-	-	-	-	-	A	+	-	-	-	-	-	RU	+	-	-	-	-	-

Fibrosis	Fibrocavitatory
RU	P
RU	P
A	A
RU	A
A	A
LU	P
A	A
A	A
RU	P
A	A
A	A
RU	P
LU	P
A	A
LM	P
A	A
LL	P
RM	P
A	A
LU	P
RM	P
RU	P
A	A
RU	P
BU	P
RU,RM	P
A	A
A	A
RU	P
A	A
A	A
LU	A
A	A
A	A
A	A
RU	P

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RU	A
A	A
RU	P
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LU	P
A	A
RU	A
RU	A
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RL	P
LL	P
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LM	P
RM	P
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RU	P
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A	A
RU	P
A	A
A	A
A	A
RU	P

ANNEXURE III - KEY TO MASTER CHART

-	-	Absent
+	-	Present
A	-	Absent
AFB	-	Acid fast bacilli
Ag	-	Agriculture
B	-	Business
Cm	-	Centimeter
D	-	Driver
DA	-	Dimorphic anaemia
ESR	-	Erythrocyte sedimentation rate
F	-	Female
H/o	-	History of
HIV	-	Human immunodeficiency virus
HW	-	House wife
IP. No.	-	In patient number
Kg	-	Kilogram
L	-	Labourer
M	-	Male
m	-	Meter
MHA	-	Microcytic hyperchromic anaemia
Mr	-	Married
NHA	-	Normocytic hyperchromic anaemia

NR	-	Non reactive
P	-	Present
R	-	Reactive
S	-	Single
S. No.	-	Serial number
SE	-	Service
ST	-	Student
W	-	Widow