A dissertation on

"PROSPECTIVE STUDY ON ELECTROCARDIOGRAPHIC CHANGES IN SPUTUM POSITIVE PULMONARY TUBERCULOSIS BEFORE AND AFTER ANTITUBERCULOUS THERAPY"

Dissertation submitted to

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CHENNAI, TAMIL NADU

With partial fulfillment of the regulations required For the award of degree of M.D. GENERAL MEDICINE

BRANCH- I

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COIMBATORE

May 2019

CERTIFICATE

This is to certify that this dissertation titled "**PROSPECTIVE STUDY ON ELECTROCARDIOGRAPHIC CHANGES IN SPUTUM POSITIVE PULMONARY TUBERCULOSIS BEFORE AND AFTER ANTITUBERCULOUS THERAPY**" has been done by **Dr. BALASUBRAMANIYAM.. J** under my guidance. Further certified that this work is an original study of bonafide cases.

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DECLARATION

I, **Dr. J. BALASUBRAMANIYAM**, declare that the Dissertation titled **"PROSPECTIVE STUDY ON ELECTROCARDIOGRAPHIC CHANGES IN SPUTUM POSITIVE PULMONARY TUBERCULOSIS BEFORE AND AFTER ANTITUBERCULOUS THERAPY"** Submitted to the Dr. M. G. R. Medical university, Guindy, Chennai is an original work done by me during the academic period from MAY 2o17 – MAY 2o18 at the Department of Medicine, Coimbatore Medical College Hospital, Coimbatore, under the guidance and direct supervision of **Dr. USHA. S** in partial fulfillment of the rules & regulations of the Dr. M. G. R. Medical University for MD Medicine post graduate degree.

All the details of the patients, the materials and methods used are true to the best of my knowledge.

I assure that this dissertation has not been submitted to or evaluated by any other Medical University.

Dr. BALASUBRAMANIYAM.J

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The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation Proposal is accepted and you are permitted to proceed with the above Study.

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ABBREVIATIONS

TB	-	Tuberculosis
ESR	-	Erythrocyte Sedimentation Rate
AFB	-	Acid Fast Bacilli
ATT	-	Anti-tuberculous Therapy
CSF	-	Cerebrospinal Fluid
CBNAAT	-	Cartridge-based nucleic acid amplification test
MTB/Rif	-	Mycobacterium tuberculosis/ Rifampicin
INH	-	Isoniazid
NAAT-TB	-	Nucleic Acid Amplification Test
BCG	-	Bacille Calmette Guerin
TST	-	Tuberculin skin test
IGRA	-	Interferon Gamma release assay
ESAT-6	-	Early Secretory Antigenic Target 6kDa
CFP	-	Culture Filtrate Protein 10
LPA	-	Line Probe Assay
CXR	-	Chest X-Ray

PMDT	-	Programmatic Management of Drug-resistent Tuberculosis
EPTB	-	Extrapulmonary Tuberculosis
IP	-	Intensive Phase
CP	-	Continuation Phase
MDR	-	Multi Drug Resistant
RR TB	-	Rifampicin Resistant Tuberculosis
BDQ	-	Bedaquiline
RNTCP	-	Revised National Tuberculosis Control Programme
RBBB	-	Right Bundle Branch Block

INTRODUCTION

Tuberculosis caused by Mycobacterium Tuberculosis remain a major health issue worldwide , roughly one third of worlds population has been infected with Mycobacterium Tuberculosis with new infections occurring in about 1% of the population each year An estimated 1o.4 million people fell ill with TB in 2o16: 9o% were adults, 65% were male . Tuberculosis is the second most common cause of death from infectious diseases, India had the largest total incidence with an estimated 2.0 million new cases. Cardiovascular involvement occurs in one to two percent of the patients with pulmonary tuberculosis and usually affects the pericardium and myocardium rarely valves are involved, very less work has been done to determine cardiac manifestations in pulmonary tuberculosis. The objective was to study the electrocardiographic changes in sputum positive pulmonary tuberculosis patients before and after anti-tuberculosis treatment with six months follow up to know the influence of anti- tuberculosis treatment over electrocardiographic changes and cardiac manifestations in pulmonary tuberculosis patients.

AIM OF THE STUDY

1) To study the electrocardiographic changes in untreated cases of sputum positive pulmonary tuberculosis.

2) To follow up for six months and study whether the electrocardiographic changes are markedly decreased or normalized after anti-tuberculosis therapy

REVIEW OF LITERATURE

Tuberculosis is caused by Mycobacterium tuberculosis. Its remains a worldwide health problem inspite of the fact that organism was discovered 1oo years back. Tuberculosis affects primarily the lungs but it can affect also intestine, meninges, bones, joints, lymphnodes, skin .Sometimes it affects animals called BOVINE tuberculosis. The tubercle bacilli was discovered by ROBERT KOCH in 1882 The disease was called "CONSUMPTION" in the past because of the way it would consume from within anyone who became infected .It can affect any age group. In 1993 WHO declared TB as global health problem About 1/3 of population was infected with tuberculosis bacilli, Most maternal deaths occurred due to tuberculosis. The burden of tuberculosis was more from Asia and Africa. TB is the ninth leading cause of death worldwide and the leading cause from a single infectious agent, ranking above HIV/AIDS. In 2o16, there were an estimated 1.3 million TB deaths among HIV-negative people (down from 1.7 million in 2000) and an additional 374000 deaths among HIV-positive people. An estimated 1o.4 million people fell ill with TB in 2o16: 9o% were adults, 65% were male, 1o% were people living with HIV (74% in Africa) and 56% were in five countries: India, Indonesia, China, the Philippines and Pakistan. Drug-resistant TB is a continuing threat. In 2016, there were 600000 new cases with resistance to rifampicin (RRTB), the most effective first-line drug, of which 490 000 had multidrug-resistant TB (MDR-TB). Almost half (47%) of these cases were in India, China and the Russian Federation. Globally, the TB mortality rate is falling

at about 3% per year. TB incidence is falling at about 2% per year and 16% of TB cases die from the disease; by 2o2o, these figures need to improve to 4–5% per year and 1o%, respectively, to reach the first (2o2o) milestones of the End TB Strategy. Most deaths from TB could be prevented with early diagnosis and appropriate treatment. Millions of people are diagnosed and successfully treated for TB each year, averting millions of deaths (53 million 2ooo–2o16), but there are still large gaps in detection and treatment. In 2o16, 6.3 million new cases of TB were reported (up from 6.1 million in 2o15), equivalent to 61% of the estimated incidence of 1o.4 million; the latest treatment outcome data show a global treatment success rate of 83%, similar to recent years. There were 476 774 reported cases of HIV-positive TB (46% of the estimated incidence), of whom 85% were on antiretroviral therapy (ART). A total of 129 689 people were started on treatment for drug-resistant TB, a small increase from 125 629 in 2o15 but only 22% of the estimated incidence THE NATIONAL TUBERCULOSIS PROGRAME was started on 1997 was not able to achieve high cure rates in developing countries .Reasons are poverty, economic recession, malnutrition, family problems, overcrowding, Tobacco, alcohol abuse, air pollution, diabetes. To make situation worse tuberculosis and HIV lethal combination, one making the other worse.

Epidemiological indices:

Presumptive case: Refers to patient who present with sign and symptom suggestive of tuberculosis

Case definition: a bacteriologically confirmed TB case is one from whom a biological specimen is positive by smear microscopy, culture, A clinically diagnosed tb who does not fulfil the criteria for bacteriological confirmation but has been diagnosed by physician who has decided to give a full course of TB treatment.

Treatment completed:

A TB patient who completed treatment without evidence of failure BUT with no record to show that sputum smear or culture results in the last month of treatment and on at least one previous occasion were negative, either because tests were not done or because results are unavailable.

Treatment failed: A TB patient whose sputum smear or culture is positive at month 5 or later during treatment.

Died: TB patient who dies for any reason before starting or during the course of treatment.

Lost to follow-up: A TB patient who did not start treatment or whose treatment was interrupted for 2 consecutive months or more.

Not evaluated : A TB patient for whom no treatment outcome is assigned. This includes cases "transferred out" to another treatment unit as well as cases for whom he treatment outcome is unknown to the reporting unit.

Treatment success: The sum of cured and treatment completed .

Classification based on history of previous TB treatment:

Classifications based on history of previous TB treatment are slightly different from those previously defined. They focus only on history of previous treatment and are Independent of bacteriological confirmation or site of disease.

New patients: Patients who have never been treated for TB or have taken anti-TB drugs for less than 1 month.

Previously treated patients: Patients who received 1 month or more of anti-TB drugs in the past.

They are further classified by the outcome of their most recent course of treatment as follows:

1) Relapse patients have previously been treated for TB, were declared cured or treatment completed at the end of their most recent course of treatment, and are now diagnosed with a recurrent episode of TB (either a true relapse or a new episode of TB caused by reinfection).
2) Treatment after failure patients are those who have previously been treated for TB and whose treatment failed at the end of their most recent course of treatment.
3) Treatment after loss to follow-up patients have previously been treated for TB and were declared lost to follow up at the end of their most recent course of treatment. (These were previously known as treatment after default patients.)

4) Other previously treated patients are those who have previously been treated for TB but whose outcome after their most recent course of treatment is unknown or undocumented.
5) Patients with unknown previous· TB treatment history do not fit into any of the categories listed above.

Classification based on drug resistance

a. **Monoresistance:** resistance to one first-line anti-TB drug only.

b. **Polydrug resistance:** resistance to more than one first line anti-TB drug (other than both isoniazid and rifampicin).

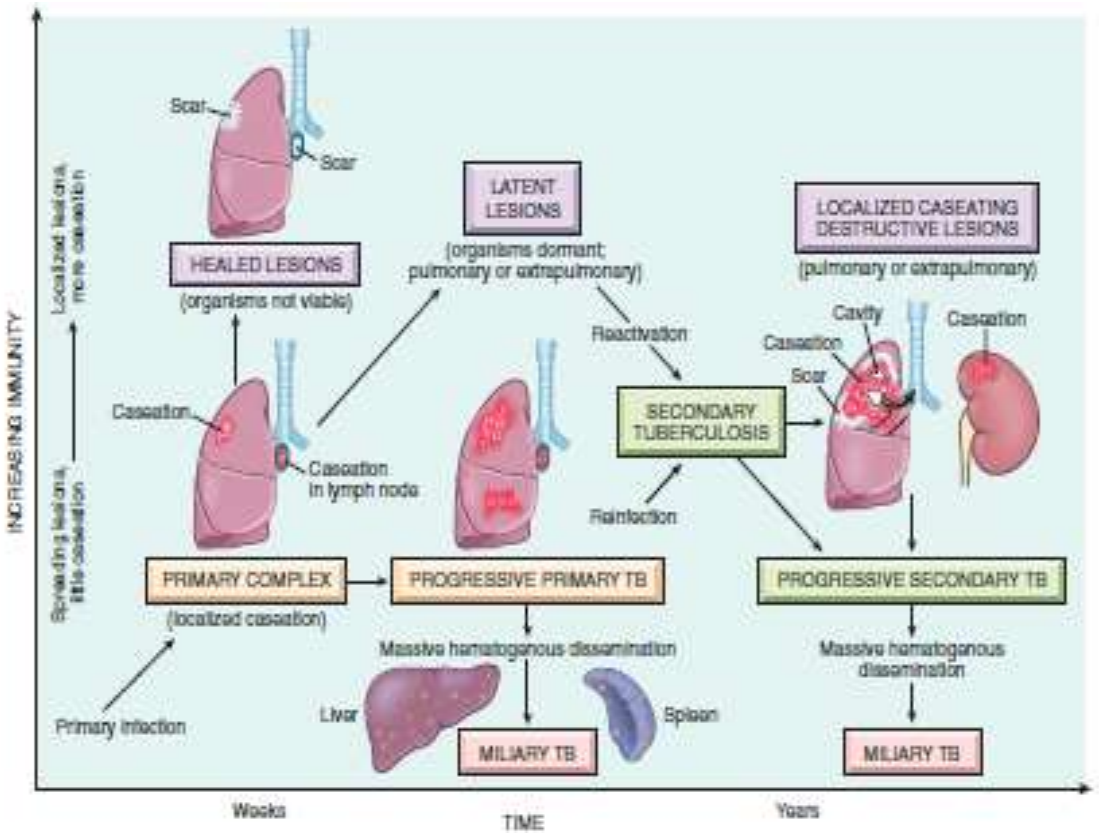
c. **Multidrug resistance:** resistance to at least both isoniazid and rifampicin.

d. **Extensive drug resistance:** resistance to any fluoroquinolone and at least one of three second-line injectable drugs (capreomycin, kanamycin and amikacin), in addition to multidrug resistance.

PATHOGENESIS

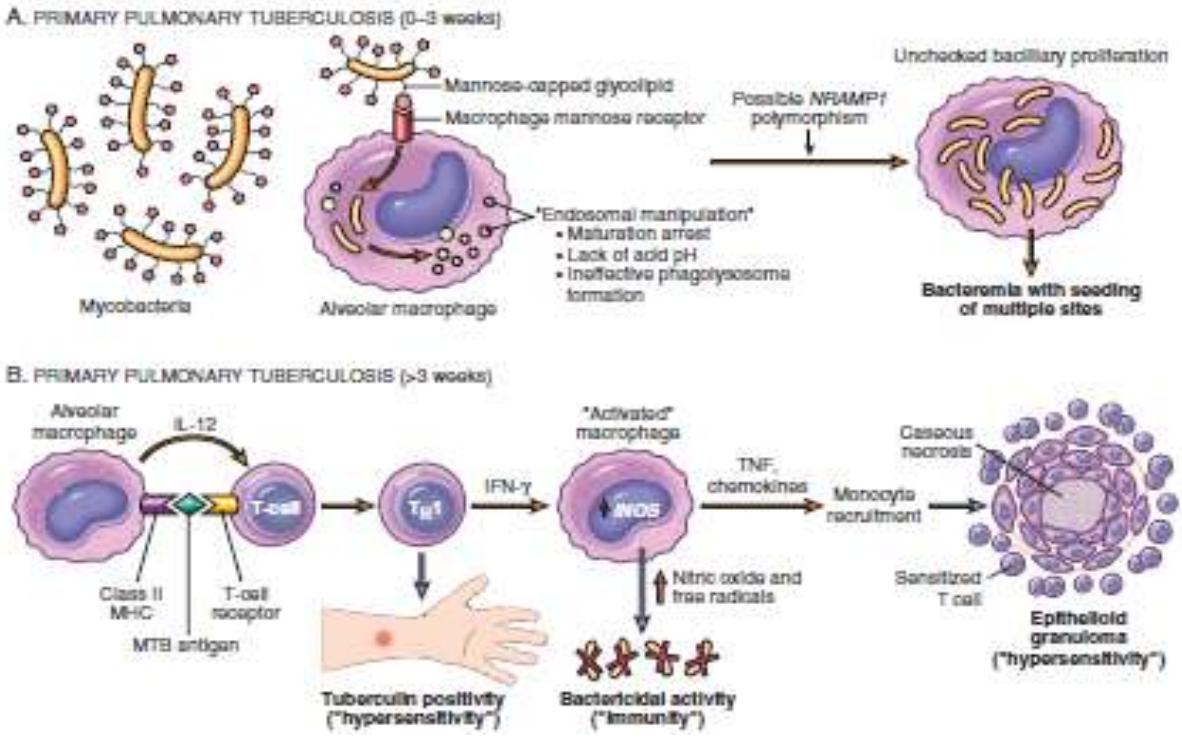
The pathogenesis of tuberculosis in the previously unexposed immunocompetent person is entered on the development of a targeted cell-mediated immunity that confers resistance to the organism and results in development of tissue hypersensitivity to tubercular antigens. The pathologic features of tuberculosis, such as caseating granulomas and cavitation, are the result of the destructive tissue hypersensitivity that is part and parcel of the host immune response. Because the effector cells for both processes are the same, the appearance of tissue hypersensitivity also signals the acquisition of immunity to the organism.

Figure - 1



The sequence of events from inhalation of the infectious inoculum to containment of the primary focus .once a virulent strain of mycobacteria gains entry into the macrophage endosomes (a process mediated by several macrophage receptors, including the macrophage mannose receptor and complement receptors that recognize several components of the mycobacterial cell walls), theorganisms are able to inhibit normal microbicidal responses by preventing the fusion of the lysosomes with the phagocytic vacuole.

Figure - 2



The prevention of phagolysosome formation allows unchecked mycobacterial proliferation. Thus, the earliest phase of primary tuberculosis (in the first 3 weeks) in the nonsensitized patient is characterized by bacillary proliferation within the pulmonary alveolar macrophages and air spaces, with resulting bacteremia and seeding of multiple sites. Despite the bacteremia, most persons at this stage are asymptomatic or have a mild flu-like illness.

- The genetic makeup of the patient may influence the course of the disease. In some people with polymorphisms of the NRAMP1 (**n**atural **r**esistance–**a**ssociated **m**acrophage **p**rotein **1**) gene, the disease may progress from this point without development of an effective immune response. NRAMP1 is a

transmembrane ion transport protein found in endosomes and lysosomes that is believed to contribute to microbial killing.

- The development of cell-mediated immunity occurs approximately 3 weeks after exposure. Processed mycobacterial antigens reach the draining lymph nodes and are presented to CD4 T cells by dendritic cells and macrophages. Under the influence of macrophage-secreted IL-12, CD4+ T cells of the TH1 subset are generated that are capable of secreting IFN-γ.
- IFN-γ released by the CD4+ T cells of the TH1 subset is crucial in activating macrophages**.** Activated macrophages, in turn, release a variety of mediators and upregulate expression of genes with important downstream effects, including TNF, which is responsible for recruitment of monocytes, which in turn undergo activation and differentiation into the "epithelioid histiocytes" that characterize the granulomatous response; expression of the inducible nitric oxide synthase *(iNoS)* gene, which results in elevated nitric oxide levels at the site of infection, with excellent antibacterial activity; and generation of reactive oxygen species, which can have antibacterial activity. You will recall that nitric oxide is a powerful oxidizing agent that results in generation of reactive nitrogen intermediates and other free radicals capable of oxidative destruction of several mycobacterial constituents, from cell wall to DNA.
- Defects in any of the steps of a TH1 response (including IL-12, IFN-γ, TNF, or nitric oxide production) result in poorly formed granulomas, absence of resistance, and disease progression. Persons with inherited

mutations in any component of the TH1 pathway are extremely susceptible to infections with mycobacteria. In summary, immunity to a tubercular infection is primarily mediated by TH1 cells, which stimulate macrophages to kill bacteria. This immune response, while largely effective, comes at the cost of hypersensitivity and the accompanying tissue destruction. Reactivation of the infection or re-exposure to the bacilli in a previously sensitized host results in rapid mobilization of a defensive reaction but also increased tissue necrosis. Just as hypersensitivity and resistance appear in parallel, so, too, the loss of hypersensitivity (indicated by tuberculin negativity in a tuberculin positive patient) may be an ominous sign that resistance to the organism has faded.

Primary Tuberculosis

Primary tuberculosis is the form of disease that develops in a previously unexposed and therefore unsensitized patient. Elderly persons and profoundly immunosuppressed patients may lose their sensitivity to the tubercle bacillus, so they may develop primary tuberculosis more than once. About 5%of those newly infected acquire significant disease. The major consequences of primary tuberculosis are that it induces hypersensitivity and increased resistance; the foci of scarring may harbour viable bacilli for years, perhaps for life, and thus be the nidus for reactivation at a later time when host defenses are compromised; and uncommonly, it may lead to progressive primary tuberculosis.

This complication occurs in patients who are immunocompromised or have nonspecific impairment of host defenses, as characteristic in malnourished children or in elderly persons. Certain racial groups, such as the Inuit, also are more prone to the development of progressive primary tuberculosis. The incidence of progressive primary tuberculosis is particularly high in HIV-positive patients with an advanced degree of immunosuppression (i.e., CD4+ counts below 2oo cells/..L). Immunosuppression results in an inability to mount a CD4+ T cell–mediated immunologic reaction that would contain the primary focus; because hypersensitivity and resistance are most often concomitant factors, the lack of a tissue hypersensitivity reaction results in the absence of the characteristic caseating granulomas (nonreactive tuberculosis).

MORPHOLOGY

In countries in which bovine tuberculosis and infected milk have largely disappeared, primary tuberculosis almost always begins in the lungs. Typically, the inhaled bacilli implant in the distal air spaces of the lower part of the upper lobe or the upper part of the lower lobe, usually close to the pleura. As sensitization develops, a 1- to 1.5-cm area of gray-white inflammatory consolidation emerges, the **Ghon focus.** In most cases the center of this focus undergoes caseous necrosis. Tubercle bacilli, either free or within phagocytes, travel in lymph drainage to the regional nodes, which also often caseate. This combination of parenchymal lesion and nodal involvement is referred to as the Ghon complex. During the first few weeks, there is also lymphatic and hematogenous dissemination to other parts of the body. In approximately 95% of cases, development of cellmediated immunity

controls the infection. Hence, the Ghon complex undergoes progressive fibrosis, often followed by radiologically detectable calcification **(Ranke complex),** and despite seeding of other organs, no lesions develop.on histologic examination, sites of active involvement are marked by a characteristic granulomatous inflammatory reaction that forms both caseating and noncaseating granulomas, which consist of epithelioid histiocytes and multinucleate giant cells.

Secondary Tuberculosis (Reactivation Tuberculosis)

Secondary tuberculosis is the pattern of disease that arises in a previously sensitized host. It may follow shortly after primary tuberculosis, but more commonly it arises from reactivation of dormant primary lesions many decades after initial infection, particularly when host resistance is weakened. It also may result from exogenous reinfection because of waning of the protection afforded by the primary disease or because of a large inoculum of virulent bacilli. Whatever the source of the organism, only a few patients (less than 5%) with primary disease subsequently develop secondary tuberculosis.

Secondary pulmonary tuberculosis is classically localized to the apex of one or both upper lobes. The reason is obscure but may relate to high oxygen tension in the apices. Because of the pre-existence of hypersensitivity, the bacilli excite a prompt and marked tissue response that tends to wall off the focus. As a result of this localization, the regional lymph nodes are less prominently involved early in the disease than they are in primary tuberculosis. on the other hand, cavitation occurs readily in the secondary form, leading to erosion into and dissemination

along airways. Such changes become an important source of infectivity, because the patient now produces sputum containing bacilli. Secondary tuberculosis should always be an important consideration in HIV-positive patients who present with pulmonary disease of note, although an increased risk of tuberculosis exists at all stages of HIV disease, the manifestations differ depending on the degree of immunosuppression. For example, patients with less severe immunosuppression (CD4+ counts greater than 3oo cells/mm3) present with “usual” secondary tuberculosis (apical disease with cavitation) while those with more advanced immunosuppression (CD4+ counts below 2oo cells/mm3) present with a clinical picture that resembles progressive primary tuberculosis (lower and middle lobe consolidation, hilar lymphadenopathy, and noncavitary disease). The extent of immunosuppression also determines the frequency of extrapulmonary involvement, rising from 1o% to 15% in mildly immunosuppressed patients to greater than 5o% in those with severe immune deficiency.

MORPHOLOGY

The initial lesion usually is a small focus of consolidation, less than 2 cm in diameter, within 1 to 2 cm of the apical pleura**.** Such foci are sharply circumscribed, firm, gray-white to yellow areas that have a variable amount of central caseation and peripheral fibrosis. In favorable cases, the initial parenchymal focus undergoes progressive fibrous encapsulation, leaving only fibrocalcific scars. Histologically, the active lesions show characteristic coalescent tubercles with central caseation. Although tubercle bacilli can be demonstrated by appropriate methods in early exudative and caseous phases of granuloma

formation, it is usually impossible to find them in the late, fibrocalcific stages. Localized, apical, secondary pulmonary tuberculosis may heal with fibrosis either spontaneously or after therapy, or the disease may progress and extend along several different pathways. Progressive pulmonary tuberculosis may ensue. The apical lesion enlarges with expansion of the area of caseation. Erosion into a bronchus evacuates the caseous center, creating a ragged, irregular cavity lined by caseous material that is poorly walled off by fibrous tissue. Erosion of blood vessels results in hemoptysis. With adequate treatment, the process may be arrested, although healing by fibrosis often distorts the pulmonary architecture. Irregular cavities, now free of caseation necrosis, may remain or collapse in the surrounding fibrosis. If the treatment is inadequate, or if host defenses are impaired, the infection may spread by direct expansion, by means of dissemination through airways, lymphatic channels, or within the vascular system. Miliary pulmonary disease occurs when organisms drain through lymphatics into the lymphatic ducts, which empty into the venous return to the right side of the heart and thence into the pulmonary arteries. Individual lesions are either microscopic or small, visible (2mm) foci of yellow white consolidation scattered through the lung parenchyma (the word miliary is derived from the resemblance of these foci to millet seeds). With progressive pulmonary tuberculosis, the pleural cavity is invariably involved and serous pleural effusions, tuberculous empyema, or obliterative fibrous pleuritis may develop. Endobronchial, endotracheal, and laryngeal tuberculosis may develop when infective material is spread either through lymphatic channels or from expectorated infectious material. The mucosal

lining may be studded with minute granulomatous lesions, sometimes apparent only on microscopic examination. Systemic miliary tuberculosis ensues when the organisms disseminate through the systemic arterial system to almost every organ in the body. Granulomas are the same as in the lung. Miliary tuberculosis is most prominent in the liver, bone marrow, spleen, adrenals, meninges, kidneys, fallopiantubes, and epididymis. Isolated-organ tuberculosis may appear in any one of the organs or tissues seeded hematogenously and may be the presenting manifestation of tuberculosis. organs typically involved include the meninges (tuberculous meningitis), kidneys (renal tuberculosis), adrenals, bones (osteomyelitis),and fallopian tubes (salpingitis). When the vertebrae are affected, the condition is referred to as Pott disease. Paraspinal “cold” abscesses may track along the tissue planes to present as an abdominal or pelvic mass. Lymphadenitis is the most frequent form of extrapulmonary tuberculosis, usually occurring in the cervical region (“scrofula”). Lymphadenopathy tends to be unifocal, and most patients do not have concurrent extranodal disease. HIV-positive patients, on the other hand, almost always have multifocal disease, systemic symptoms, and either pulmonary or other organ involvement by active tuberculosis. In years past, intestinal tuberculosis contracted by the drinking of contaminated milk was fairly common as a primary focus of tuberculosis. In developed countries today, intestinal tuberculosis is more often a complication of protracted advanced secondary tuberculosis, secondary to the swallowing of coughed-up infective material. Typically, the organisms are trapped in mucosal lymphoid aggregates of

the small and large bowel, which then undergo inflammatory enlargement with ulceration of the overlying mucosa, particularly in the ileum.

Clinical features of pulmonary tuberculosis:

- Cough
- Weight loss/anorexia
- Fever
- Night sweats
- Hemoptysis
- Chest pain
- Fatigue

Risk factors:

- Sharing air space with someone sick with TB disease (e.g., live, work, or play together)
- Crowded living conditions
- Residency or travel in a country with a high-incidence of TB disease
- High risk occupations including laboratory and health care jobs.
- Systemic effects of pulmonary tuberculosis:

Tuberculosis involving any site may produce systemic (i.e. not organ specific) symptoms. The frequency of fever ranges from 37 to 8o%. Loss of appetite, weight loss, weakness, night sweats, and malaise are also common The most common haematologic manifestations are increases in the peripheral blood polymophonuclear leukocyte count and anaemia. Each occurs in approximately 1o% of patients with apparently localized tuberculosis. Hyponatremia, which may

occur in 11% of patients is caused by the production of an antidiuretic hormone-like substance in affected lung tissue, Tuberculosis is associated often with other serious disorders including:

- HIV infection
- alcoholism
- drug abuse
- chronic renal failure
- diabetes mellitus
- neoplastic disease

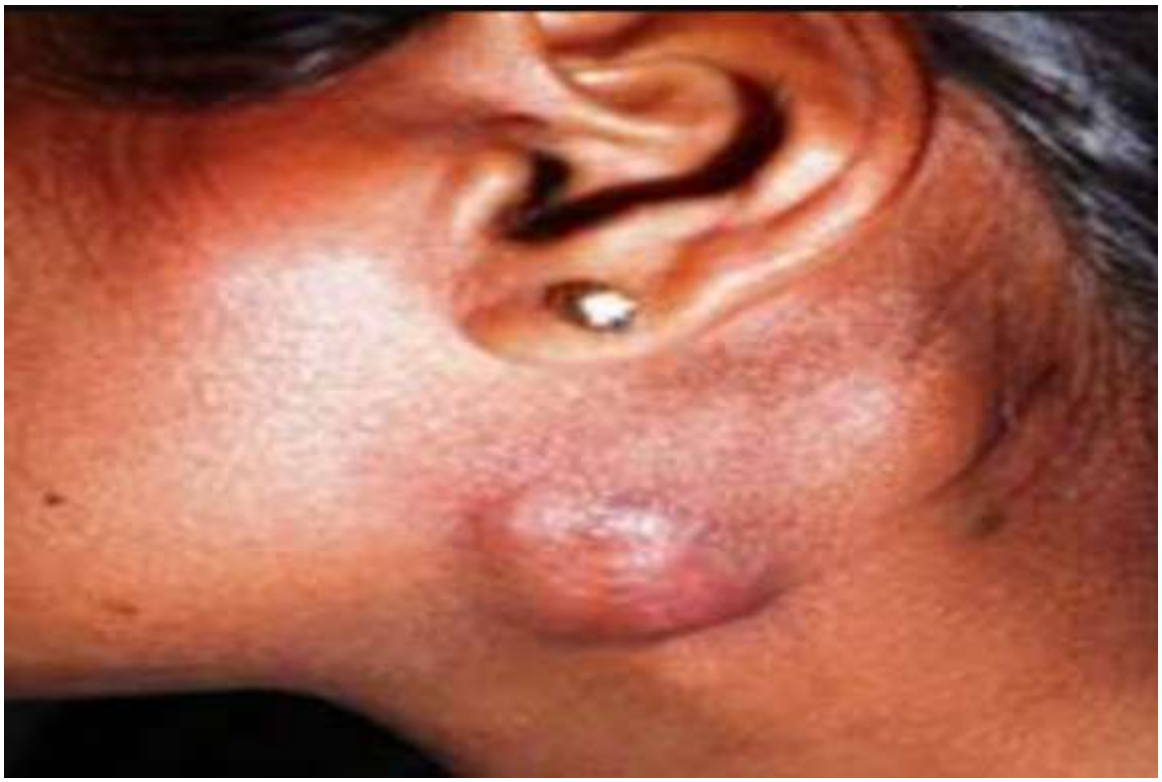
Extrapulmonary tuberculosis:

- Extrapulmonary tuberculosis in HIV-infected patients.
- Disseminated tuberculosis
- Lymph node tuberculosis
- Pleural tuberculosis
- Genitourinary tuberculosis
- Skeletal tuberculosis
- Central nervous system tuberculosis
- Abdominal tuberculosis
- Pericardial tuberculosis

Disseminated tuberculosis: Inadequacy of host defenses in containing the infection. The organism proliferates and disseminates throughout the body "miliary" tuberculosis" A finding strongly suggestive of disseminated tuberculosis is the choroidal tubercle, a granuloma in the retinal choroid.

Lymph node tuberculosis: Tuberculous lymphadenitis usually presents as painless swelling of one or more lymph nodes. Common site -posterior or anterior cervical chain of supraclavicular fossa .Rupture of the node may result in formation of a sinus tract, which is slow to heal. Intrathoracic adenopathy may compress bronchi, causing atelectasis leading to lung infection and perhaps bronchiectasis being particularly common in children.

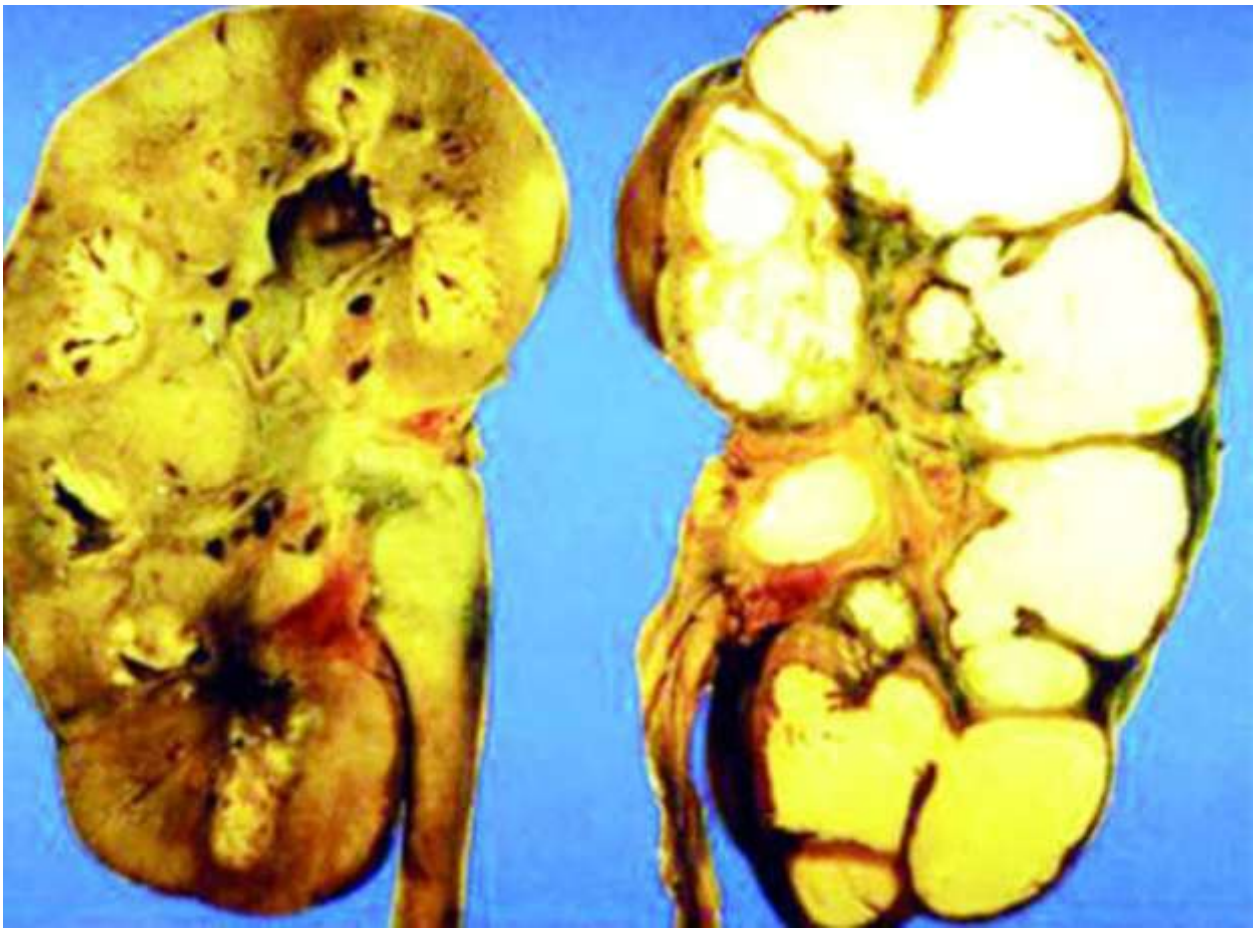
Figure - 3



Pleural tuberculosis: Early on a few organisms may gain access to the pleural space and, in the presence of cell-mediated immunity, cause a hypersensitivity response . A large number of organisms spilling into the pleural space, usually from rupture of a cavity or an adjacent parenchymal focus via a bronchopleural fistula .

Genitourinary tuberculosis: Dysuria , Hematuria ,Increase in frequency of micturition In females menstrual irregularities, pelvic pain, infertility are common ,In males scrotal Pain is more common.

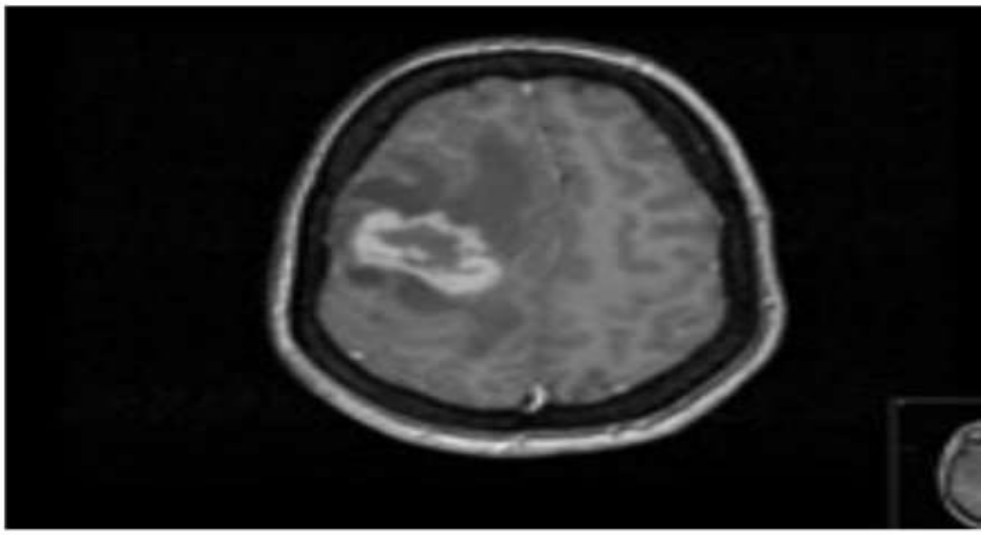
Figure - 4



Skeletal tuberculosis: Pain is the most common symptom .Swelling over the involved joint.

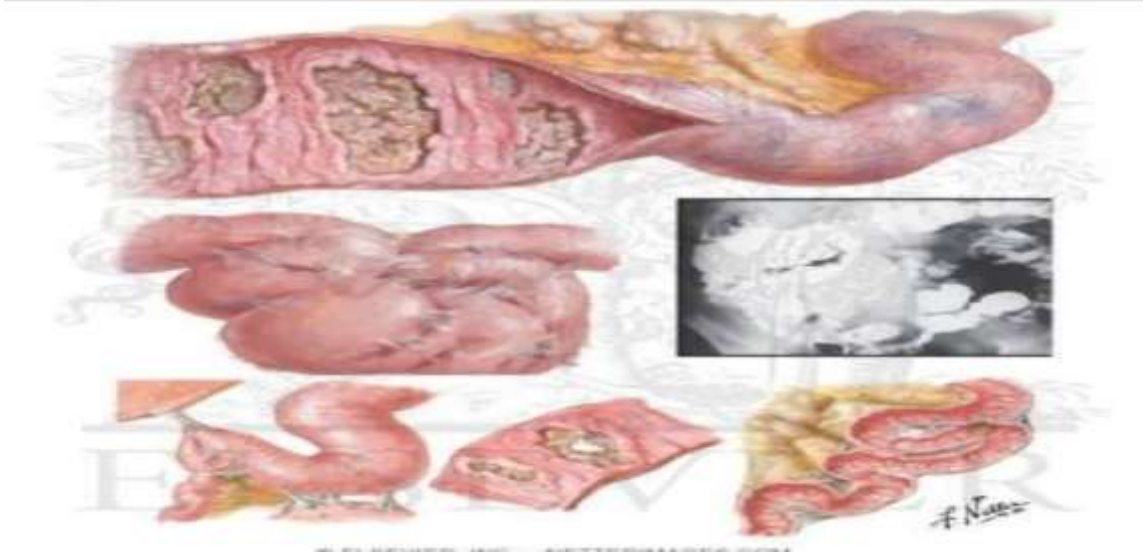
Cns tuberculosis: Meningitis commonly occur, tuberculoma, cranial nerve involvement cause headache, decreased level of consciousness and neck stiffness.

Figure - 5



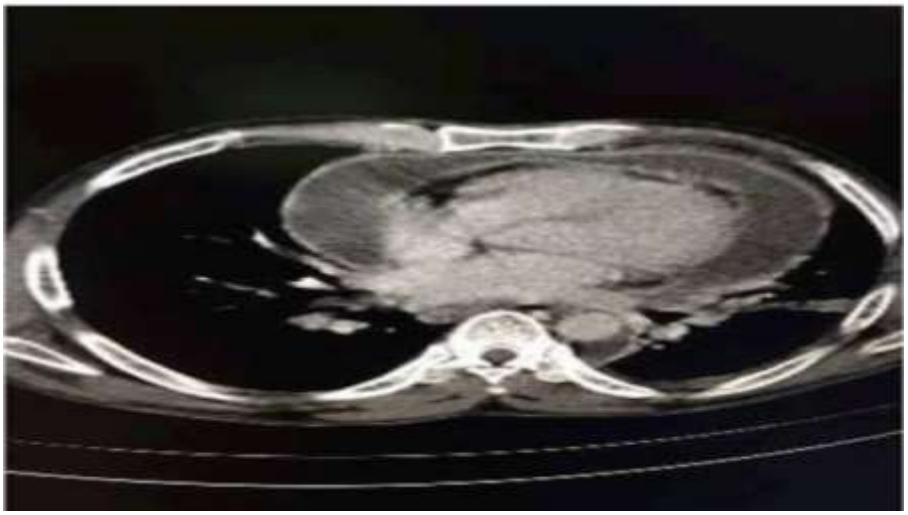
Abdominal tuberculosis: Common site Terminal ileum and caecum Tuberculous peritonitis frequently presents with pain often accompanied by abdominal swelling ,Fever, weight loss, and anorexia are also common.Rectal lesions usually present as anal fissures,fistulae or perirectal abscesses.

Figure - 6



Pericardial tuberculosis: Cardiopulmonary symptom cough, dyspnea, orthopnea, ankle swelling and chest pain .Chest pain may mimic angina.

Figure - 7



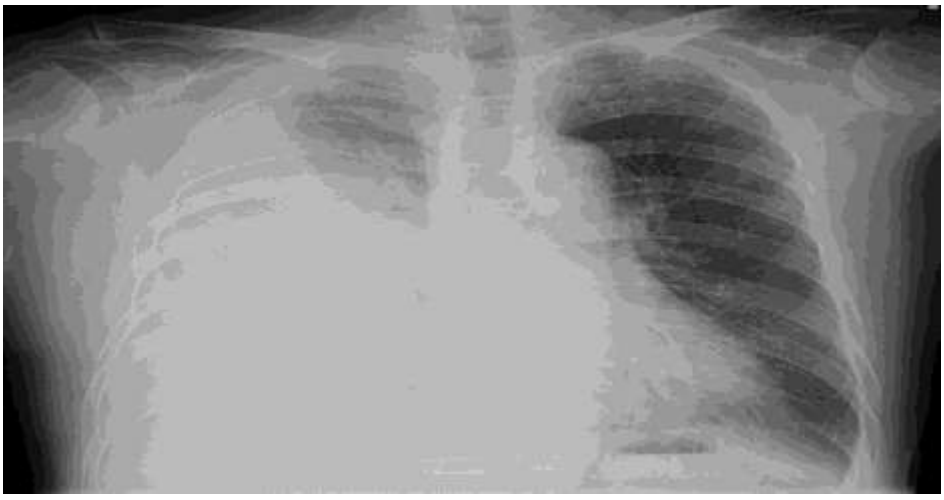
COMPLICATIONS OF PULMONARY TUBERCULOSIS:

- HEMOPTYSIS
- PNEUMOTHORAX
- PLEURAL EFFUSION
- COR PULMONALE / COPD
- TUBERCULOUS PERICARDITIS
- TUBERCULOMA
- DYSPHAGIA / DYSPHONIA
- PARAPLEGIA / PARAPERESIS
- GENITO URINARY TB
- GASTROINTESTINAL TB
- MILIARY TUBERCULOSIS
- OTHER COMPLICATIONS

HEMOPTYSIS: It may be mild, moderate or massive. It usually occurs in advanced disease. It may be the first symptom also. Mild hemoptysis occurs due to inflammation which leads to capillary breakdown – diapedesis. Mild streaks of blood is seen in the sputum. Massive hemoptysis implies coughing up of 1oo – 6oo ml of blood in 24 hours. It is due to erosion of blood vessel in tb cavity or rupture of dilated blood vessel – Rasmussen's aneurysm and aspergilloma formation. It may lead to death .5.3% of death occurs in TB due to hemoptysis and these are usually due to massive hemoptysis.

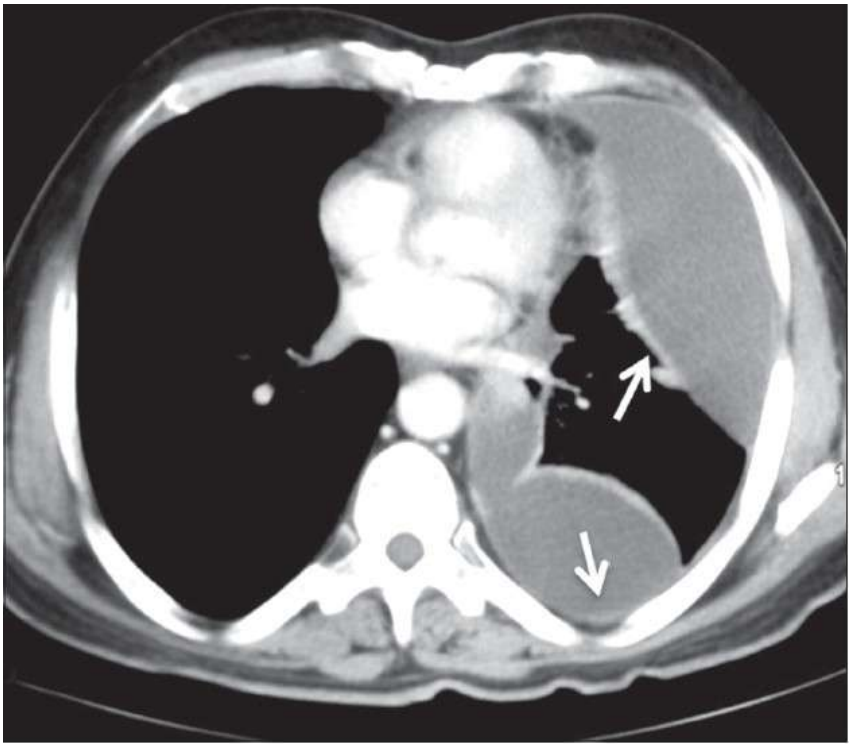
PLEURAL EFFUSION: It maybe mild moderate or massive. Mild effusion is asymptomatic. Moderate and massive pleural effusion produces symptoms depending on the rate of fluid accumulation. Clinical findings include tracheal deviation, stony dullness, straw or hemorraghic aspirate. Fluid is exudative. Determination of ADA is a useful screening test. Acid fast bacilli are seen in 1o – 25% , culture is positive in 25 – 75% cases.

Figure - 8



TUBERCULOSIS EMPYEMA: Rupture of tuberculosis lesion in pleural cavity leads to collection of purulent fluid in pleural cavity. Encystment is usuall common. Pus mainly contains polymorphs.

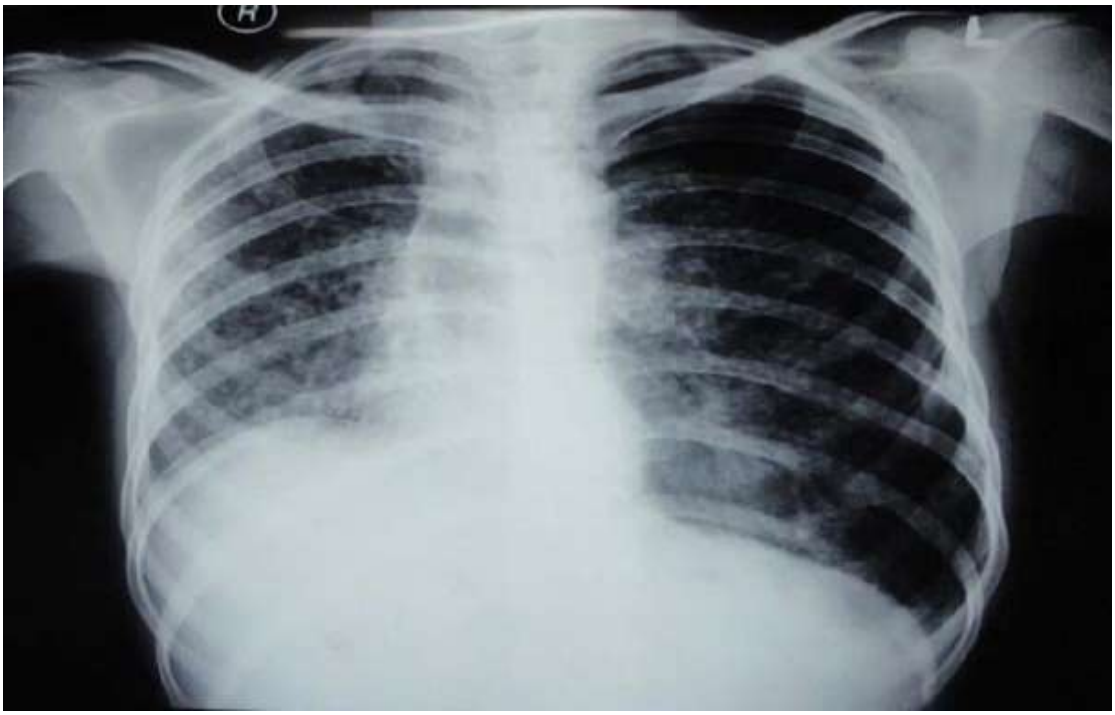
Figure - 9



CORPULMONALE / COPD: Extensive lung destruction leads to scarring of the tissue which eventually leads to corpulmonale. 5- 7 % of cases of corpulmonale in India is due to PTB. There is destruction of pulmonary vasculature, tuberculous end arteritis and vasoconstriction. Pulmonary hypertension develops in these cases. Right ventricular hypertrophy and heart failure occurs eventually. Bronchiectasis and other forms of chronic obstructive pulmonary disease is common in patients with pulmonary TB. Bronchiectasis occurs due to compression by primary complex or wall damage due to tuberculous granulation tissue or post tubercular fibrosis.

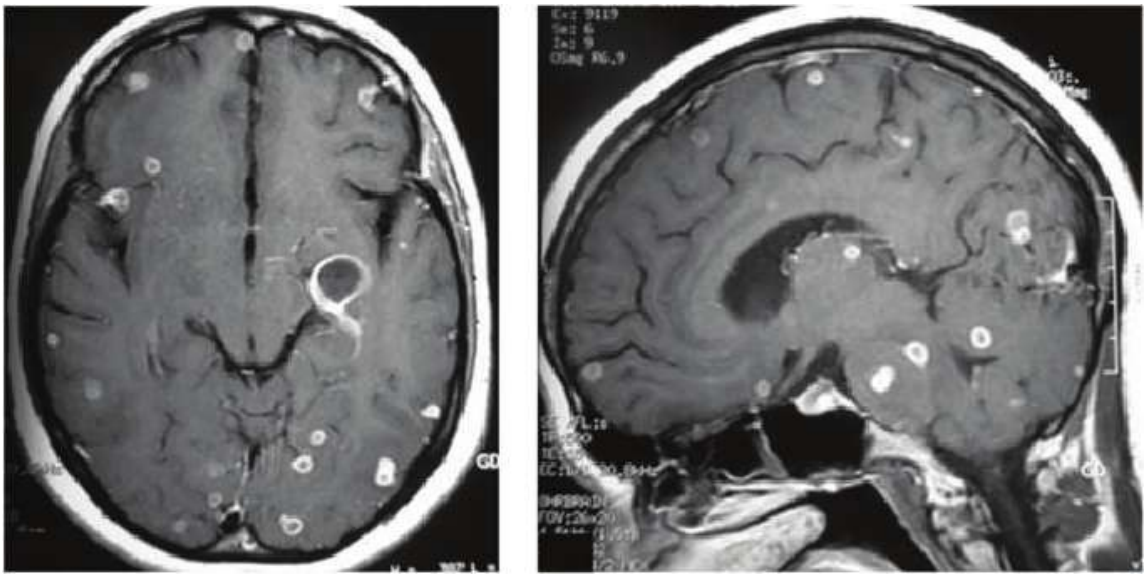
PNEUMOTHORAX : Spontaneous pneumothorax occurs due to rupture of sub pleural tuberculous lesions like bulla. Tension pneumothorax may develop. Pyopneumothorax is also a possibility. Acute chest pain, tightness, marked respiratory distress , tachycardia and cyanosis develops.

Figure - 10



TUBERCULOMA: A tuberculoma is a clinical manifestation of TB which combines tubercles into a fine lump which mimics tumors. Tuberculomas may have caseum or calcifications. Cranial nerve deficits may develop. As the histologic and clinical indications as well as tumor markers such as CA-125 are similar it is often difficult to differentiate it from cancer.

Figure - 11

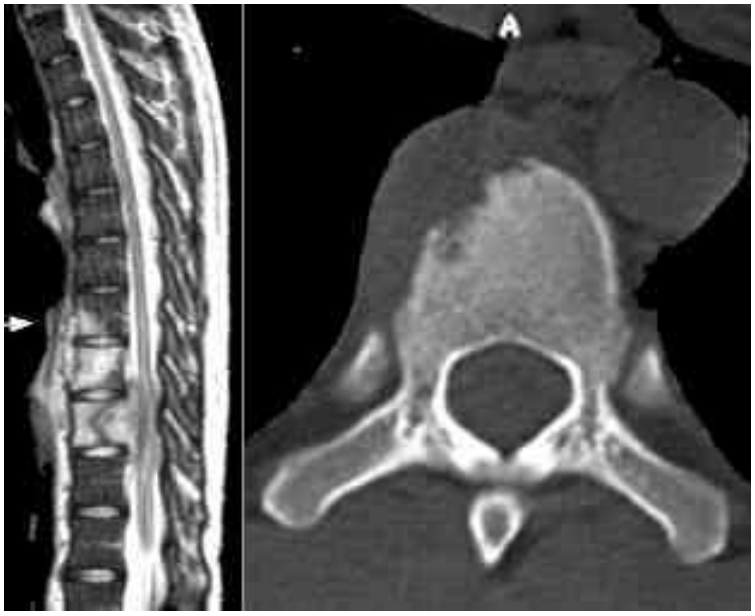


DYSPHAGIA/ DYSPHONIA: This is due to complication of TB of upper airways. It is almost usually due to advanced cavitatory lesions in advanced PTB. Ulceration may be seen in laryngoscopy.

TUBERCULOUS PERICARDITIS: Tuberculous pericarditis may be sub acute; although acute presentation with dyspnoea, pain and friction rub is possible. An effusion may develop producing cardiac symptoms – cardiac tamponade. It is more common in elderly and HIV patients. Chronic constrictive pericarditis may develop due to healing of acute fibrinous or sero fibrinous pericarditis or due to formation of granulation tissue. Case fatality rate is high as 4o% in these situations.

PARAPLEGIA / PARAPERESIS: Paraplegia is a catastrophic complication of TB spondylitis. Neurological complications occurs early in spinal TB affecting upper cervical spine and ranges from single nerve palsy to paraperesis and quadriparesis.

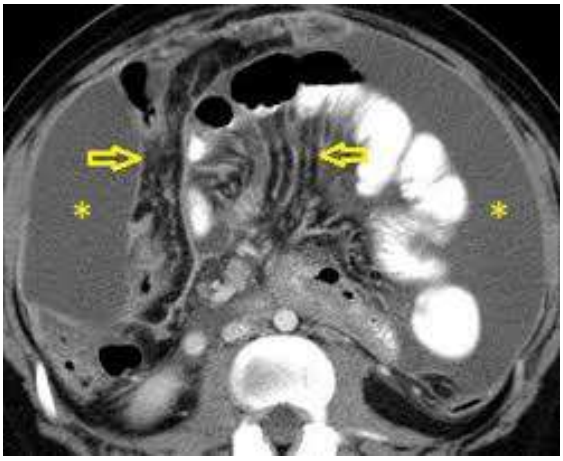
Figure - 12



GENITOURINARY TUBERCULOSIS: Patients with genito-urinary TB is usually asymptomatic until destructive lesions of kidneys develop in most cases. Severe urethral strictures leadind to hydronephrosis and renal damage may occur. In females TB affects fallopian tubes and endometrum leading to infertility, pelvic pain and menstrual abnormalities. In males TB mainly affects epidydimis producing a tender mass. Azoospermia or oligospermia can occur.

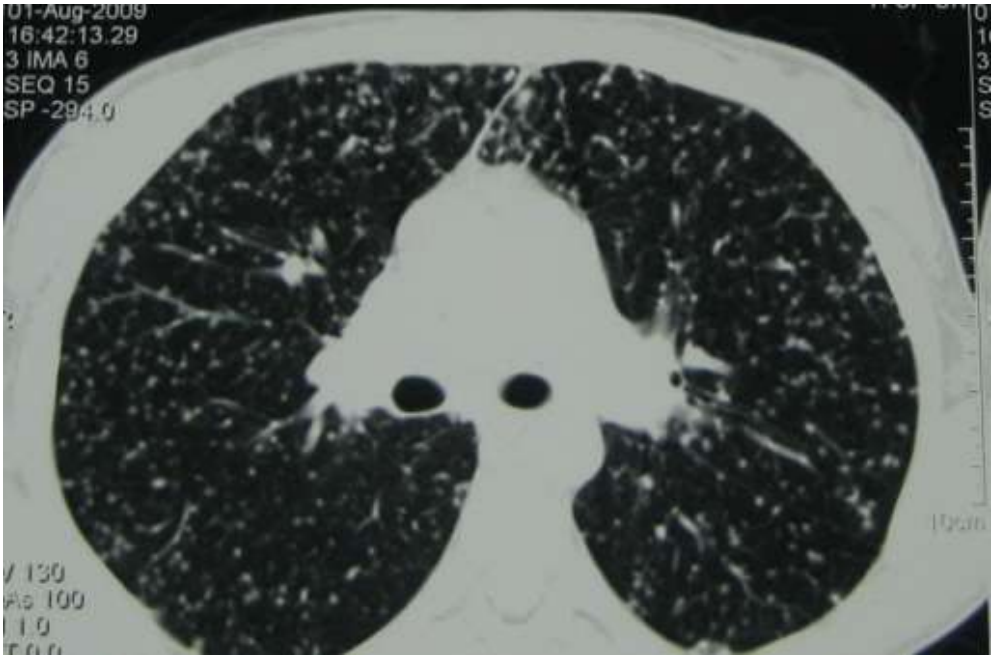
GATRO INTESTINAL TUBERCULOSIS: Pathogenesis includes swallowing of sputum with direct seeding, hemotogenous spread, ingestion of milk from cows with bovine TB. Symptoms include abdominal pain, swelling, obstruction and hematochezia. Palpable mass is a common presentation.

Figure - 13



MILIARY TUBERCULOSIS: It is due to wide spread dissemination of bacilli by hematogenous spread affecting lungs and extrapulmonary organs such as liver, spleen , kidney , brain causing tiny size lesions about 1-5 mm which looks like millet seeds on radiograph. Hence the name , miliary TB.

Figure - 14



OTHER COMPLICATIONS: TB affects eyes and causes chorioretinitis, panophthalmitis, uveitis and hyper sensitivity related phlyctenular conjunctivitis. TB of ear can cause otitis presenting as hearing loss , ottorhoea , perforation. Cutaneous manifestations of Tb includes abscesses ,chronic ulcers and scrofulderma by direct inoculation. Adrenal TB is a manifestation of disseminated disease presenting as adrenal insufficiency.

Cardiac manifestations of pulmonary tuberculosis:

Pericardial involvement in tuberculosis may result in acute pericarditis, chronic pericardial effusion, cardiac tamponade or pericardial constriction. In India, TB accounts for nearly two thirds of the cases of constrictive pericarditis.

The sub-acute stage of tuberculous pericarditis presents with features due to the presence of the pericardial fluid and those due to pericardial constriction as a result of the thickening of the visceral pericardium . Tuberculous pericarditis is always associated with a focus of tuberculosis elsewhere in the body.

The disease most commonly spreads to the pericardium by direct extension from the tracheo bronchial tree, the mediastinal or the hilar lymph nodes, the sternum or the spine. The spread may also take place by a haematogenous route from a focus in the lung.

The tuberculous involvement of the myocardium is rare, which is seen mostly in association with pericardial disease. Isolated myocardial tuberculosis is an unusual finding; the prevalence has been reported as o.14%, o.2% and 2% in various series. Tuberculosis which involves the endocardium is extremely rare.

The diagnosis is usually made during autopsy. Only few cases. The infection is a result of direct extension from the myocardium or of a haematogenous spread. The tuberculous involvement of the coronary vessels is very rare.

INVESTIGATIONS:

Mantoux Tuberculin Skin Test (TST):

o.1 ml of tuberculin purified protein derivative (- PPD) into inner surface of forearm intradermally. Read between 48-72 hrs. A positive tuberculin skin test result is supportive evidence in the diagnosis of TB in areas of low prevalence (or no vaccination); however, a negative tuberculin skin test result may occur in approximately one third of patients.

Direct demonstration of mycobacterium by staining method:

Sputum smear examination by microscopy: at least 1oooo bacilli per ml of sputum is required for positive results. Ziehl-neelsen technique is most commonly used.Grades according to the number of bacilli seen with ziehlneelsen staining

If the slide has:	No. of fields to be examined	Grading	Result
No AFB in 100 oil immersion fields	100	0	Neg
1-9 AFB per 100 oil immersion fields	100	Scanty*	Pos
10-99 AFB per 100 oil immersion fields	100	1+	Pos
1-10 AFB per oil immersion field	50	2+	Pos
More than 10 AFB per oil immersion field	20	3+	Pos

Other Staining Methods

- Cold staining methods (kinyoun”s or with gabett”s solution)
- Fluorescent staining using
- Auramine-o
- Auramine-rhodamine
- Rhodamine
- Acridine orange

Isolation of Mycobacteria by Culture:

Culture methods provide definitive diagnosis of mycobacterium by identity of the organisms and establishing the viability.The culture method is considered gold standard, it can detect as few as 1o to 1oo bacilli per ml.

Culture characters:

The growth appears in about two weeks but can be delayed up to six to eight weeks. optimum PH for growth is 6.4 to7.o. optimum temperature is 37o C. increased Co2 tension (5% to 1o%) enhances the growth7.

Culture media:

Solid media:

- Lowenstein – Jensen
- Loeffler serum slope
- Pawlowskys potato medium
- Tarshis medium (blood medium)

Liquid Media

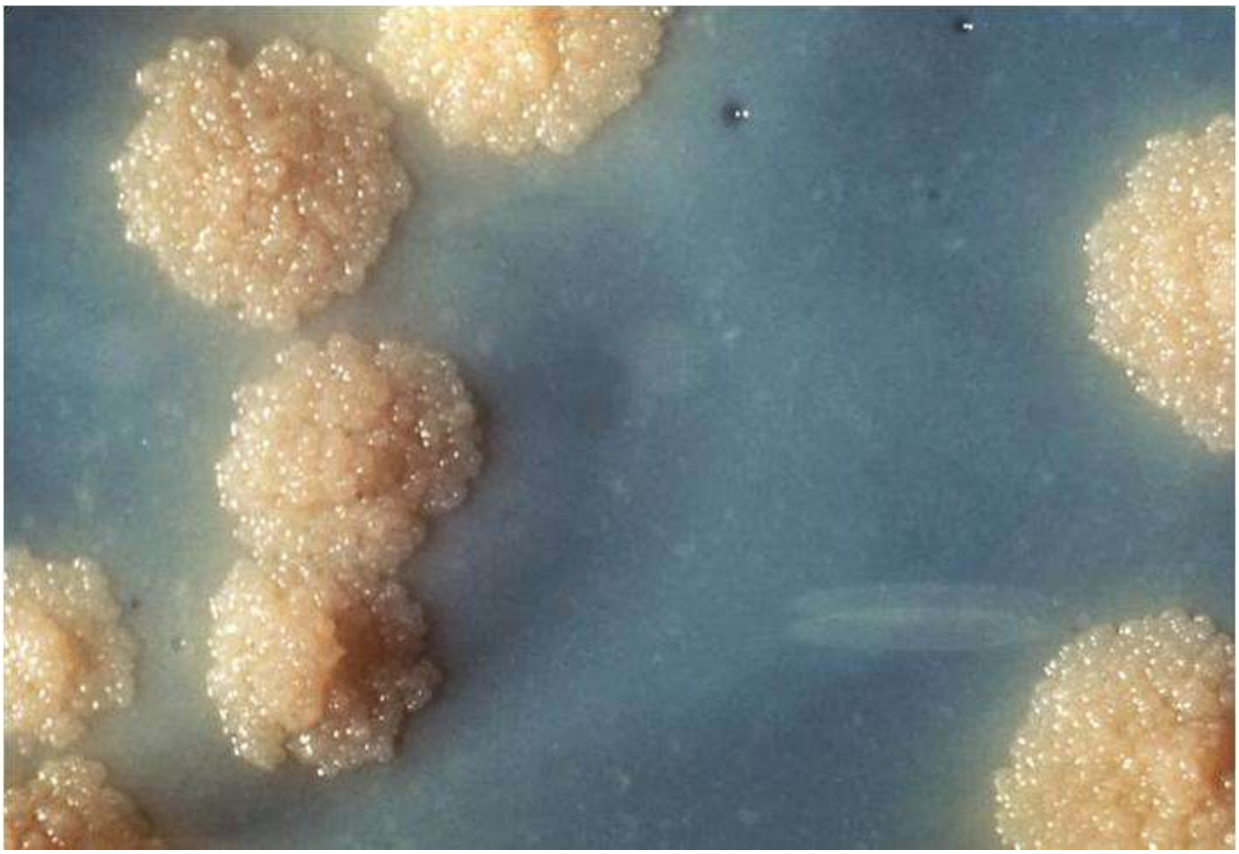
- Dubos medium
- Middle brook's medium
- Sula's medium
- Sauton's medium

Colony characteristics:

On solid media human type of tubercle bacilli give rise tofollowing character. It is discrete, raised, irregular, dry,wrinkled colonies. They are creamy white in the beginning, then develop buff colour.

M.bacilli growth on solid media:

Figure - 15



Rapid culture methods:

- BACTEC radiometric system
- Septi chek acid-fast bacilli method

It is a biphasic - culture approach for detection and isolation of mycobacterium tuberculosis

Rapid liquid tuberculosis culture also known as Mycobacteria Growth Indicator tube(MGIT) : Positive signals are obtained in 1o-12 days. MGIT can also be used as a rapid method for the detection of drug resistant strains of Mtb directly from acid-fast smear positive samples as well as from indirect drug susceptibility

Immunodiagnosis:

- Antibody detection test:Immunoglobulin IgG and IgM detection against A6o antigen most commonly used.

- Antigen detection test

  Lipoarabinomannan urine test

  Flow-through filter test

- Polymerase chain reaction sequencing (PCR) is based on amplification of mycobacterial DNA fragments. It can detect as few as 1o mycobacteria. Advantages of PCR include rapid diagnosis, improved specificity and sensitivity, and no requirement of intact immunity.This test is used for detection of rifampicin resistance.

- Identification of mycobacterium by high performance liquid chromatography.

- Gas – liquid chromatography.

- GeneXpert: Xpert MTB/RIF detects M. tuberculosis as well as rifampicin resistance conferring mutations using three specific primers and five unique molecular probes to ensure a high degree of specificity. The assay provides results directly from the sputum within 1oo minutes, Simultaneous detection of both MTB and rifampicin resistance, a marker for MDR strains Unprecedented sensitivity for detecting MTB — even in smear negative, culture positive specimens, Results in two hours; on-demand results enable physicians to treat rapidly and effectively.

- Line probe assay: LPA test provides an early diagnosis of resistance to isoniazid and rifampicin and is highly sensitive and specific for an early diagnosis of MDR-TB. Sensitivity for isoniazid is suboptimal • Based on these findings, it is concluded that the LPA test is to be implemented as first line test for diagnosis of drug resistant TB among sputum smear positive patients in high TB burden countries.

- Microscopic –observation drug-susceptibility assay The microscopic-observation drug-susceptibility (MoDS) assay is a low-cost alternate to the detection of drug resistance. MoDS detection of MDRTB was excellent

with sensitivity and specificity of 95 and 1oo%, respectively . The time to detection has been shown to be 7 days and similar to the MGIT.

- Interferon-gamma release assays (IGRAs) are diagnostic tools for latent tuberculosis infection . They are surrogate markers of Mycobacterium tuberculosis infection and indicate a cellular immune response to M. tuberculosis.

- IGRAs cannot distinguish between latent infection and active tuberculosis (TB) disease, and should not be used as a sole method for diagnosis of active TB.

- QuantiFERoN-TB Gold Test and T-SPoT-TB Test are Commercially Available IGRAs Tests.

TREATMENT oF PULMONARY TUBERCULOSIS:

GROUPING OF ANTI-TB DRUGS

Group 1: First-line oral anti-TB agents

Isoniazid (H); Rifampicin (R); Ethambutol (E); Pyrazinamide (Z)

Group 2: Injectable anti-TB agents

Streptomycin (S); Kanamycin(Km); Amikacin (Am); Capreomycin (Cm); Viomycin (Vm).

Group 3: Fluoroquinolones

Ciprofloxacin (Cfx); ofloxacin(ofx); Levofloxacin (Lvx); Moxifloxacin (Mfx); Gatifloxacin (Gfx).

Group 4: oral second-line anti-TB agents

Ethionamide (Eto); Prothionamide (Pto); Cycloserine (Cs); Terizadone (Trd); paraaminosalicylicacid (PAS)

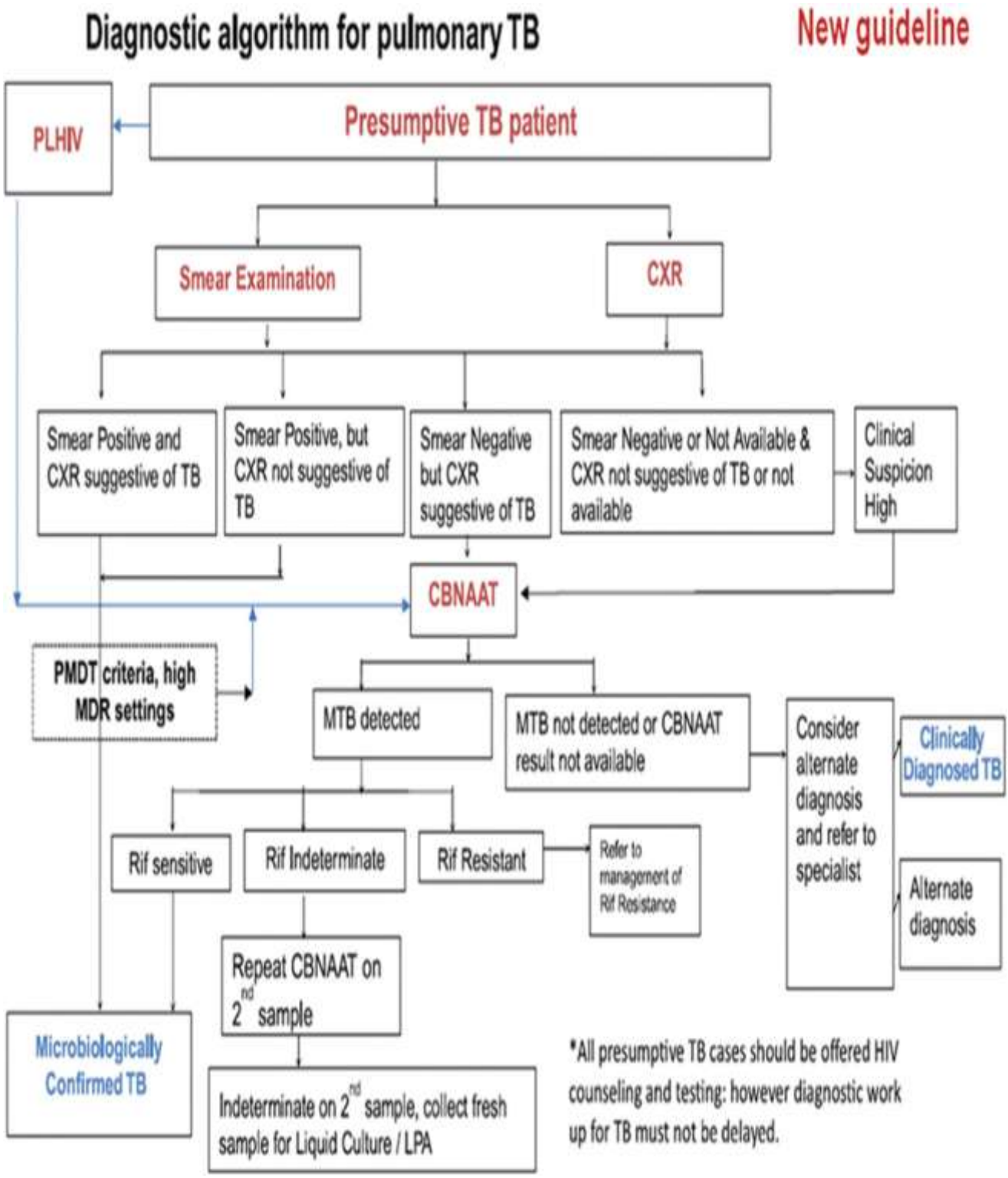
Group 5: Agents with unclear efficacy (not recommended by WHo for routine use in MDR-TB patients)

Clofazimine (Cfz); Linezolid (Lzd); Amoxicillin/Clavulanate(Amx/Clv); thioacetazone (Thz); imipenem/cilastatin (Ipm/Cln); high-dose isoniazid (high-dose H); Clarithromycin (Clr)

NEWER ANTI-TB DRUGS:

- BEDAQUILINE
- DELAMANID

Figure - 16


Diagnostic algorithm for pulmonary TB
New guideline
PLHIV
Presumptive TB patient
Smear Examination
CXR
Smear Positive and CXR suggestive of TB
Smear Positive, but CXR not suggestive of TB
Smear Negative but CXR suggestive of TB
Smear Negative or Not Available & CXR not suggestive of TB or not available
Clinical Suspicion High
CBNAAT
PMDT criteria, high MDR settings
MTB detected
MTB not detected or CBNAAT result not available
Consider alternate diagnosis and refer to specialist
Clinically Diagnosed TB
Alternate diagnosis
Rif sensitive
Rif Indeterminate
Rif Resistant
Refer to management of Rif Resistance
Repeat CBNAAT on 2nd sample
Microbiologically Confirmed TB
Indeterminate on 2nd sample, collect fresh sample for Liquid Culture / LPA
*All presumptive TB cases should be offered HIV counseling and testing; however diagnostic work up for TB must not be delayed.

New guidelines	Previous guidelines
Daily regimen	Intermittent regimen
Ethambutol in CP of both categories I and II regimen	Ethambutol in CP of category II regimen only
Fixed dose combination as per weight band	No fixed dose, limited weight band
No need of extension of IP	Extension of IP for 1 month if sputum is positive at the end of IP
Follow-up-clinical, laboratory investigation	Follow-up-laboratory only
Long-term follow-up up to 2 years	No long-term follow-up

Name of Drug	Daily dose (mg/Kg body wt)	
	Adult	Paediatrics
Isoniazid	5 mg/kg (4 - 6 mg/kg) daily	10 mg/kg (7 - 15 mg/kg) daily
Rifampicin	1o mg/kg (8 - 12 mg/kg) daily	15 mg/kg (10 - 20 mg/kg) daily
Streptomycin	15 mg/kg (12 - 18 mg/kg) daily	15 mg/kg (12 - 18 mg/kg) daily
Ethambutol	15 mg/kg (15 - 20 mg/kg) daily	20 mg/kg (15 - 25 mg/kg) daily
Pyrazinamide	25 mg/kg (20 - 30 mg/kg) daily	35 mg/kg (30 - 40mg/kg) daily

But there are significant changes in the drug regimen in the new guidelines Principle of treatment of TB has been shifted towards daily regimen with administration of daily fixed dose combination of first-line ATD as per appropriate weight ban

For new TB cases:

- Treatment in IP will consist of 8 weeks of INH, Rifampicin, Pyrazinamide and Ethambutol in daily dosages as per four weight bands categories.
- There will be no need for extension of IP.
- Only Pyrazinamide will be stopped in CP while the other three drugs will be continued for another 16 weeks as daily dosages.

For previously treated cases:IP will be of 12 weeks, where injection Streptomycin will be stopped after 8 weeks and the remaining four drugs in daily dosages as per weight band for another 4 weeks

- No need of extension of IP
- At the start of CP, Pyrazinamide will be stopped while rest of the drugs will be continued for another 2o weeks as daily dosages.

TREATMENT OUTCOMES:

1. **Cured-** A microbiologically confirmed TB at the beginning of the treatment who was smear or culture-negative at the end of complete treatment (Changed).
2. **Treatment success-** TB patients either cured or treatment completed are accounted in the treatment success (New addition).

3. **Failure-** A TB patient whose biological specimen is positive by smear or culture at the end of the treatment (Changed).
4. **Lost to follow-up-** A TB patient whose treatment was interrupted for one consecutive month or more (New addition).
5. **Not evaluated-** A TB patient for whom no treatment outcome is assigned. (known as transfer out previously).
6. **Treatment regimen changed**- Previously, it was called as switched over to MDR treatment.

MDR/RR TB CASES (without additional resistance)

- 6 - 9 months of IP which includes Kanamycin, Levofoxacin, Ethambutol, Pyrazinamide, Ethionamide and Cycloserine.
- 18 months of CP which includes Levofoxacin, Ethambutol, Ethionamide and Cycloserine (no change)

MDR/RR TB CASES

- If INH resistance is not known or DST result shows sensitivity to INH, then addition of INH in the above-mentioned regimen of ATD is to be done (New addition).
- All MDR TB cases would be subjected to Liquid Culture and DST at baseline for Kanamycin and Levofloxacin.

NEW ADDITIONS TO DRTB GUIDELINES

- If RR by CBNAAT, then add INH in the standard dose till result of LPA/LC and DST.

- For new patients diagnosed as TB and RR by CBNAAT – repeat CBNAAT and send the sample for liquid culture.

DRUG RESISTANT TB

- Introduction of new ATD under RNTCP - Bedaquiline (BDQ)
- New class of drug, diaryl-quinoline
- MoA = targets MTB-ATP synthase, which is essential for supply of energy to the bacterium
- Has strong bactericidal and sterilizing activities against MTB

Advantages

1. No cross-resistance with first and second-line ATD
2. Significant benefit in improving the time to culture conversion in MDR-TB patients.

Basic criterion

1. Adult aged ≥18 years having pulmonary MDR TB
2. Non-pregnant females

Management of TB patients with liver disorder

If the serum alanine amino transferase level is more than three times normal before initiation of treatment, the regime should be

Containing two hepatotoxic drugs:

a. INH + Rifampicin + Ethambutol for 9 months or INH + Rifampicin + Ethambutol + Streptomycin for 2 months followed by INH and Rifampicin for 7 months or
b. Rifampicin + Ethambutol + Pyrazinamide for 6 - 9 months.

Containing one hepatotoxic drug:

a. INH + Ethambutol + Streptomycin for 2 months followed by INH + Ethambutol for 1o months.
b. Containing no hepatotoxic drugs: Streptomycin + Ethambutol + FQ for 18–24 months.

MATERIALS AND METHODS

SOURCE OF STUDY:

Data consists of standard 12 lead electrocardiograph taken by the principal investigator directly from the cases of sputum positive pulmonary tuberculosis admitted in the medical ward and TB chest medicine ward and from patients treated as outpatient in Coimbatore medical college hospital

METHODOLOGY:

This is a prospective study of 1oo cases of sputum positive pulmonary tuberculosis patients of both gender and more than 2o years of age standard 12 lead ECG will be taken at the time of diagnosis less than 2 weeks of starting ATT and after completing the course of anti tuberculosis treatment and data will be analysed and studied whether ATT influences ECG changes in pulmonary tuberculosis by follow up for six months in patients admitted or treated as outpatient in Coimbatore medical college hospital during the period from may 2017 to may 2018.

INCLUSION CRITERIA:

1. Acid fast bacilli positive cases.
2. New cases with less than two weeks of ATT ensured by detailed history.
3. Both gender and age more than 2o years.

EXCLUSION CRITERIA:

Pre-existing heart diseases, copd anemia, hypertension diabetes mellitus corpulmonale and pulmonary hypertension due to other causes

RESULTS

TABLE - 1

SEX DISTRIBUTION

SEX	NO OF PATIENTS	PERCENTAGE
MALE	78	78%
FEMALE	22	22%

CHART – 1

SEX DISTRIBUTION

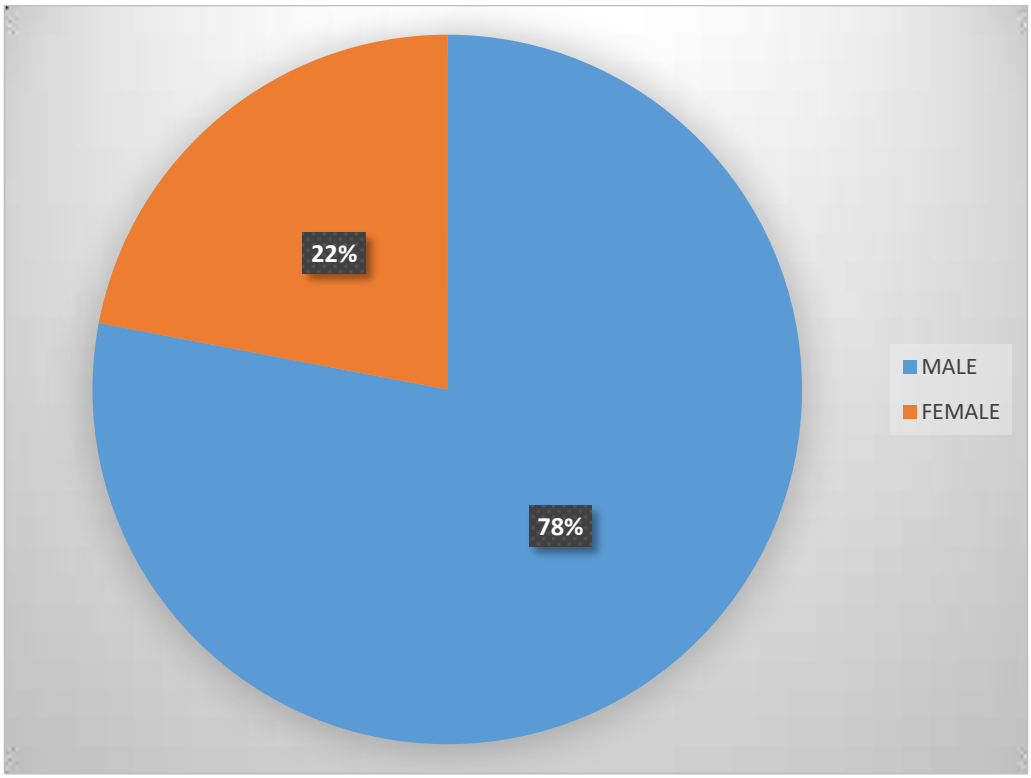


TABLE - 2

AGE WISE DISTRIBUTION

AGE IN YEARS	NO OF PATIENTS	PERCENTAGE
<30	22	22%
31-40	25	25%
41-50	22	22%
> 50	31	31%

CHART - 2

AGE WISE DISTRIBUTION

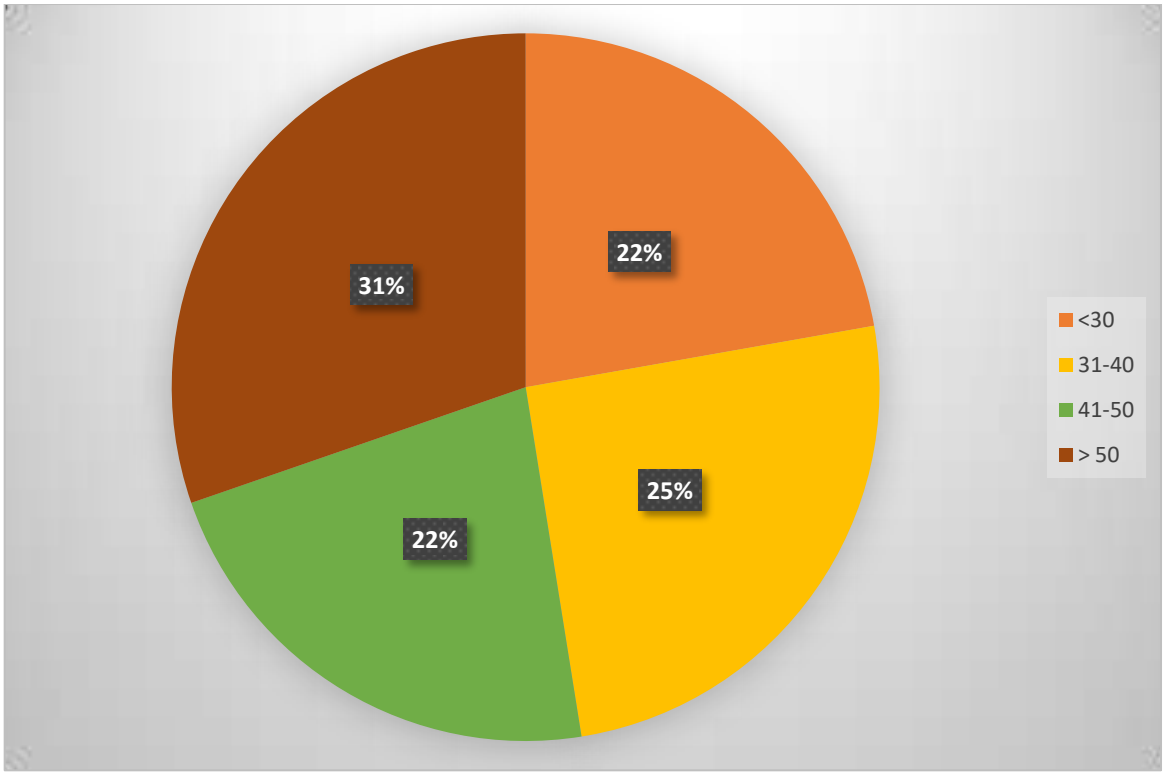


TABLE - 3

DURATI0N 0F SYMPTOMS

DURATION OF SYMPTOMS	NO OF PATIENTS	PERCENTAGE
LESS THAN 6 MONTHS	59	59%
M0RE THAN 6 MONTHS	41	41%

CHART - 3

DURATION OF SYMPTOMS

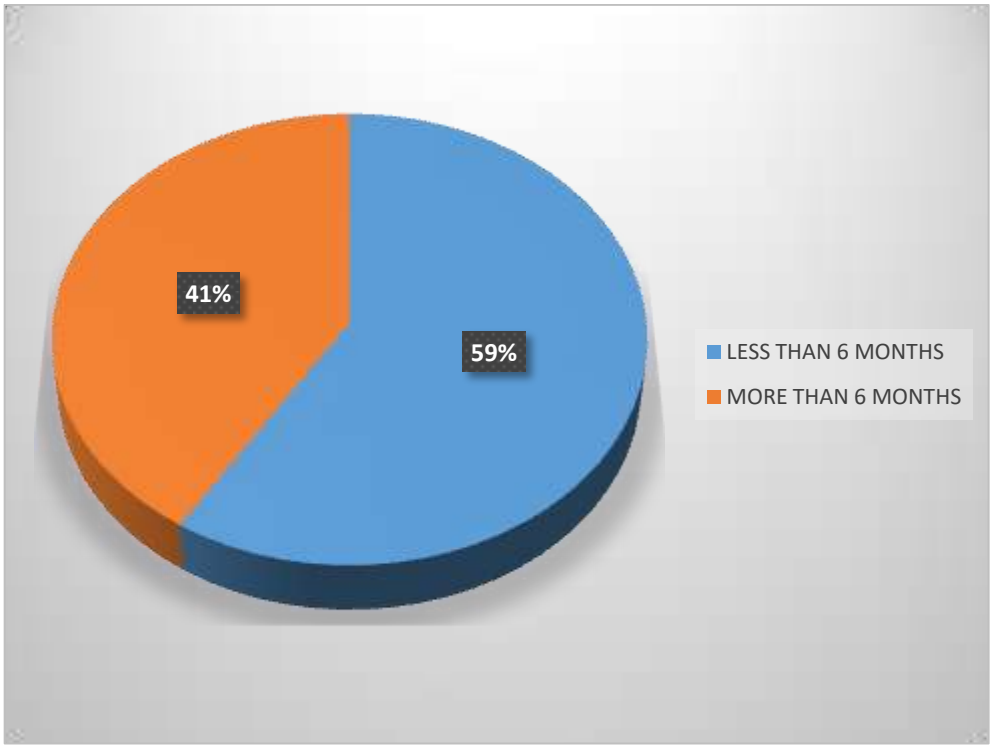


TABLE – 4

ECG CHANGES - DURATION

ECG CHANGES - BEFORE ATT	DURATION OF SYMPTOMS	
	MEAN	SD
PRESENT	3.51	0.52
ABSENT	2.32	0.41
UNPAIRED T TEST		
P VALUE - 0.012		
SIGNIFICANT		

CHART - 4

DURATION OF SYMPTOMS VS ECG CHANGES

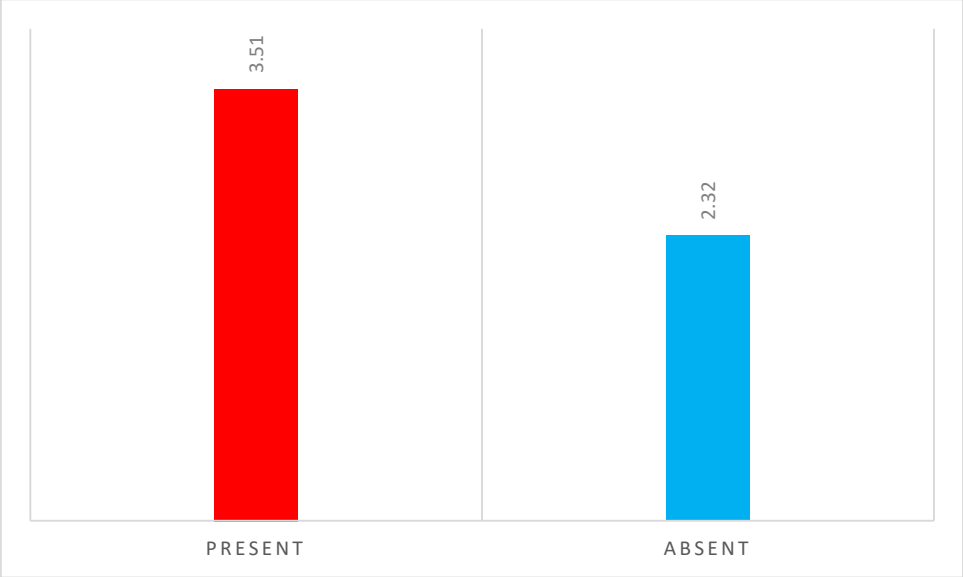


TABLE – 5

ECG CHANGES - BEFORE ATT

ECG CHANGES - BEF0RE ATT	NO OF PATIENTS	PERCENTAGE
PRESENT	66	66%
ABSENT	34	34%

CHART – 5

ECG CHANGES - BEFORE ATT

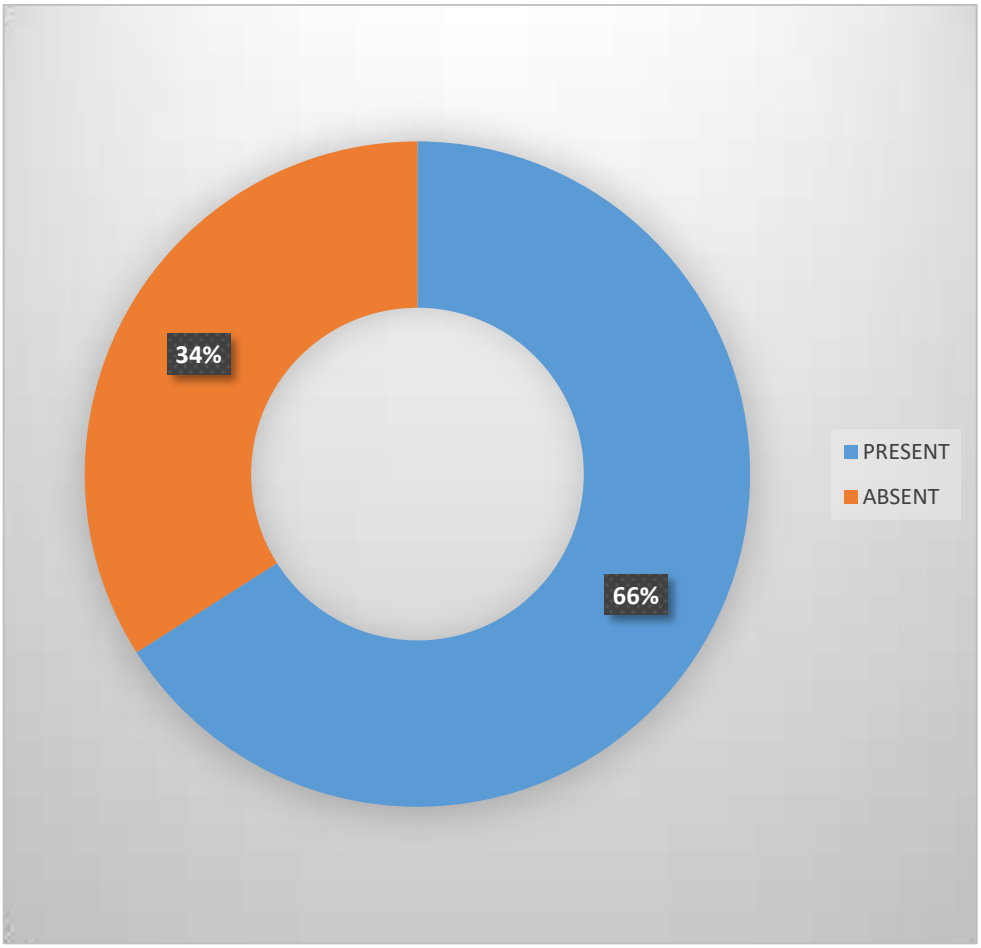


TABLE - 6

ECG CHANGES - AFTER ATT

ECG CHANGES - AFTER ATT	NO OF PATIENTS	PERCENTAGE
PRESENT	32	32%
ABSENT	68	68%

CHART - 6

ECG CHANGES - AFTER ATT

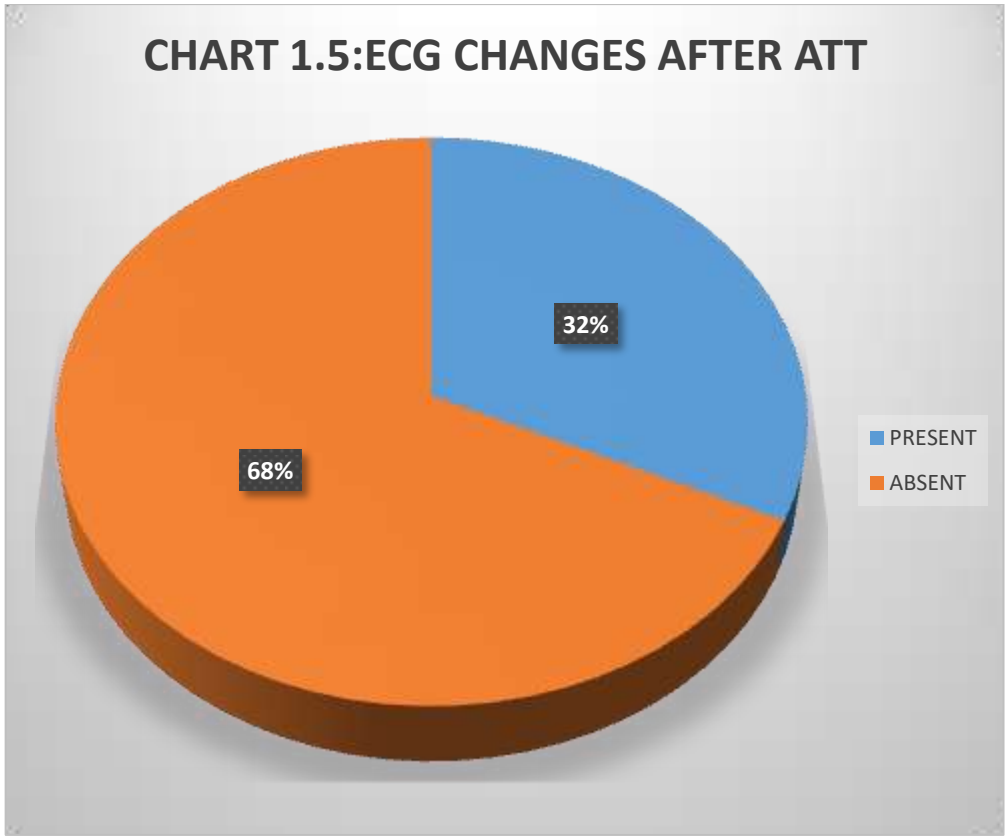


TABLE -7

ECG CHANGES – BEFORE AND AFTER ATT

ECG CHANGES	BEFORE	AFTER
PRESENT	64	32
ABSENT	36	68

CHART - 7

ECG CHANGES – BEFORE AND AFTER ATT

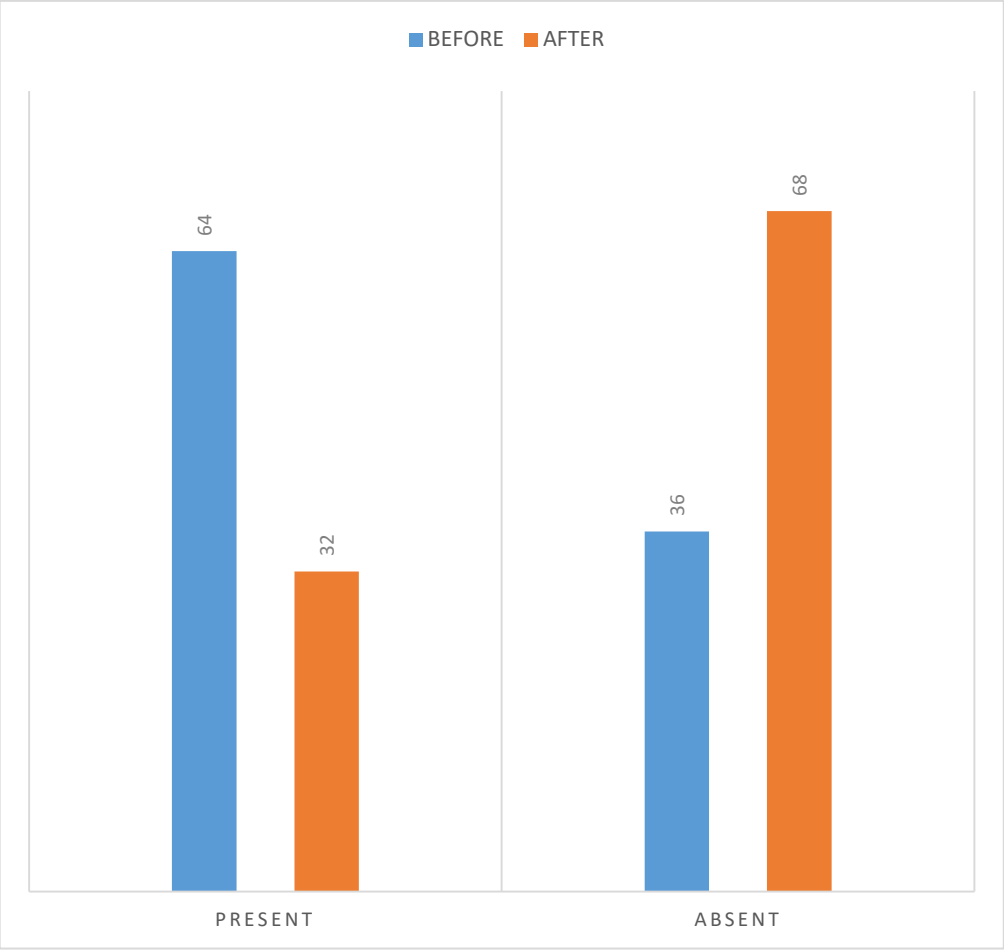


TABLE - 8

P WAVE DURATION

P WAVE DURATI0N	BEF0RE	AFTER
N0RMAL	100	100
ABN0RMAL	0	0

CHART - 8

P WAVE DURATION

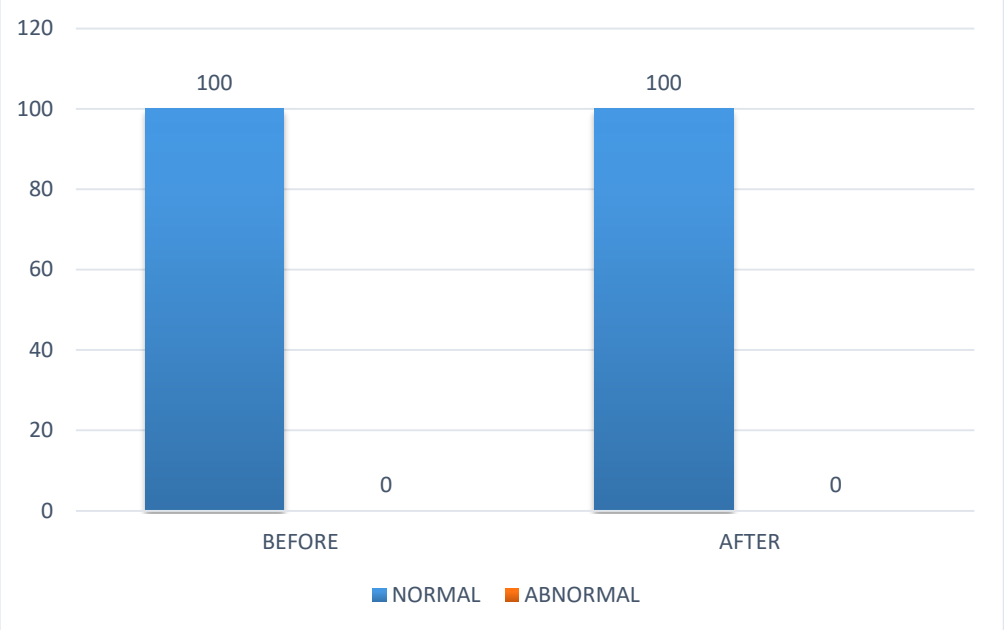


TABLE - 9

P WAVE AXIS

P WAVE AXIS	BEF0RE	AFTER
N+ 90 DEGREE	11	7
N0RMAL	89	93
P VALUE - 0.322		
N0N SIGNIFICANT		

CHART - 9

P WAVE AXIS

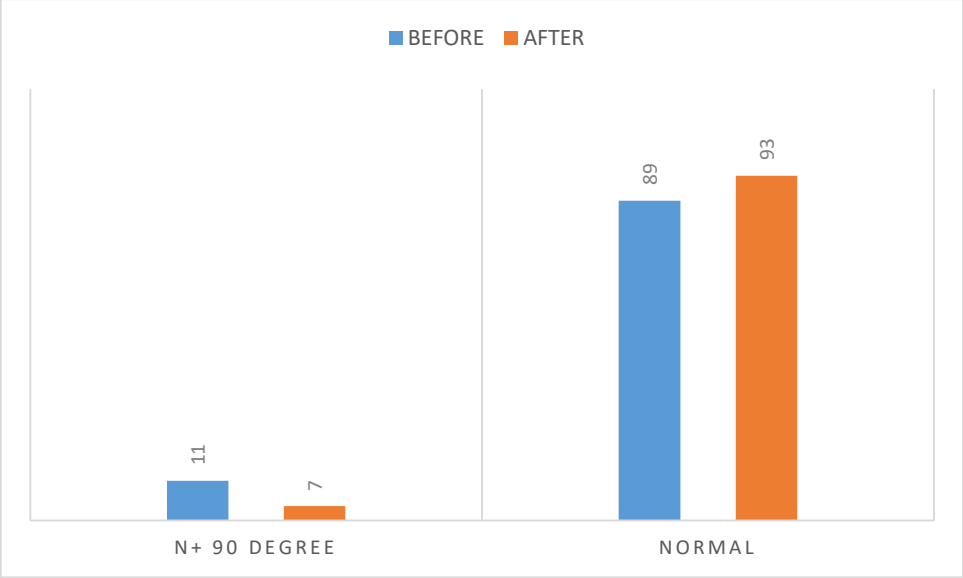


TABLE – 10

PR INTERVAL

PR INTERVAL	**BEFORE**	**AFTER**
PR0L0NGED	3	3
N0RMAL	97	97
P VALUE - 0.100		
N0N SIGNIFICANT		

TABLE – 10

PR INTERVAL

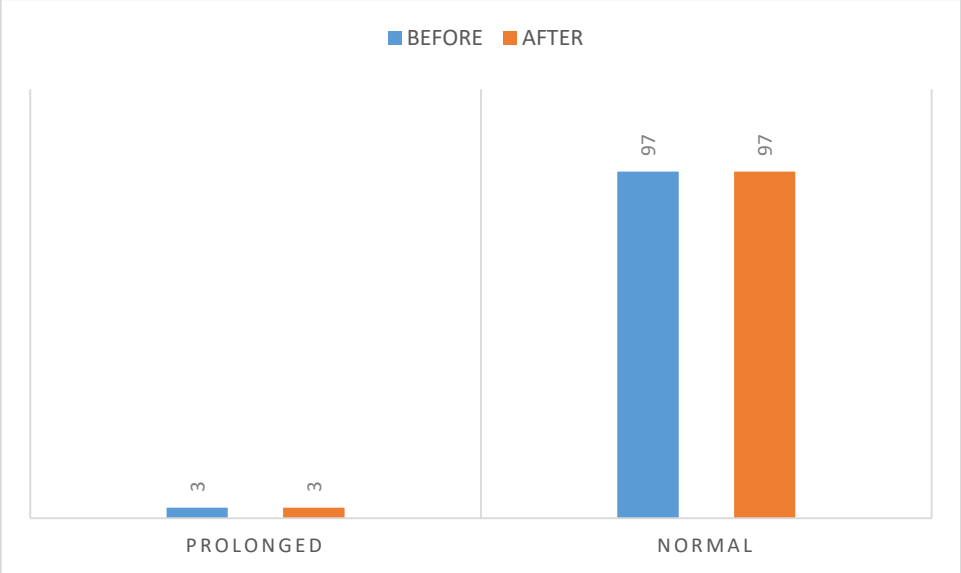


TABLE – 11

MEAN P WAVE AMPLITUDE

P WAVE AMPLITUDE	MEAN	SD
BEF0RE	2.17	0.66
AFTER	1.98	0.57
P VALUE - 0.014		
SIGNIFICANT		

CHART – 11

MEAN P WAVE AMPLITUDE

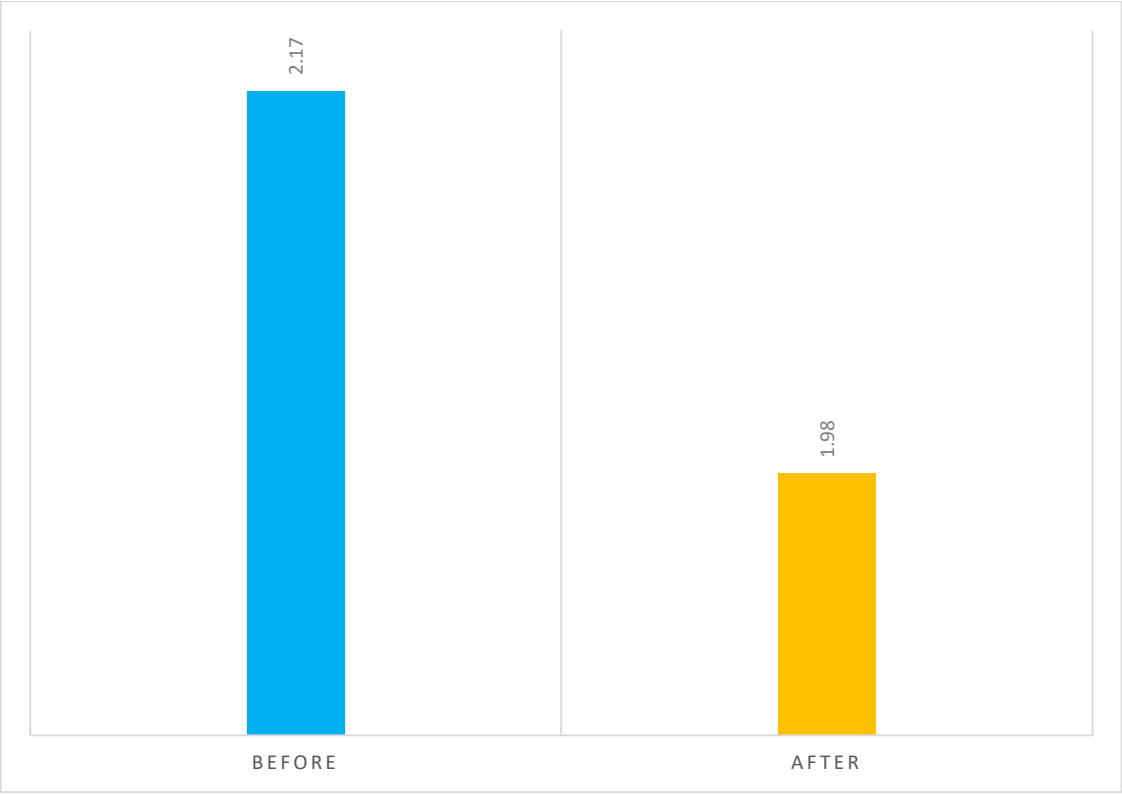


TABLE – 12

QRS DURATION

QRS DURATION	BEFORE	AFTER
N0RMAL	100	100
ABN0RMAL	0	0

CHART – 12

QRS DURATION

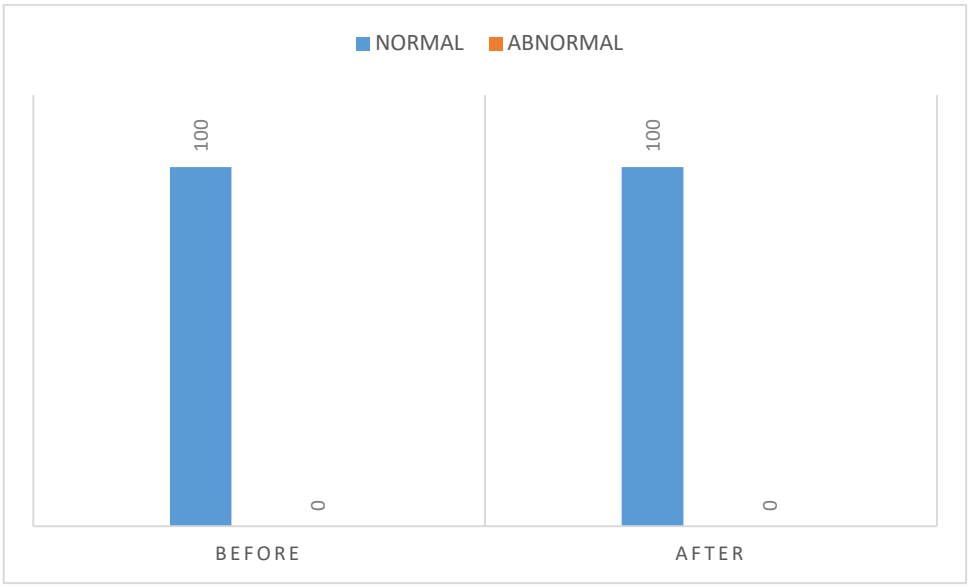


TABLE – 13

QRS AXIS

QRS AXIS	BEFORE	AFTER
N+90 DEGREES	11	5
NORMAL	89	95
P VALUE - 0.117		
NON SIGNIFICANT		

CHART – 13

QRS AXIS

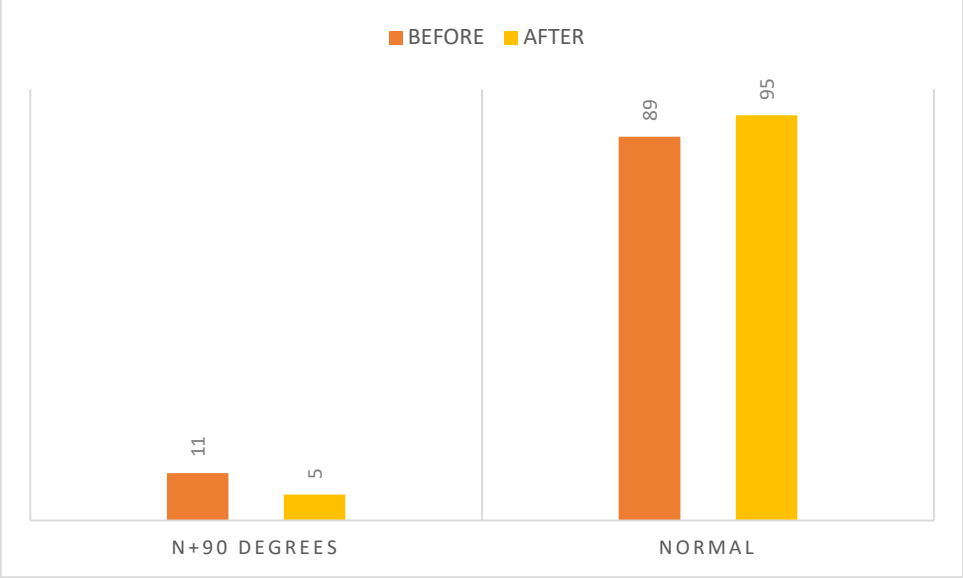


TABLE – 14

QRS AMPLITUDE

QRS AMPLITUDE	BEFORE	AFTER
LOW	15	7
NORMAL	85	93
P VALUE - 0.070		
NON SIGNIFICANT		

CHART – 14

QRS AMPLITUDE

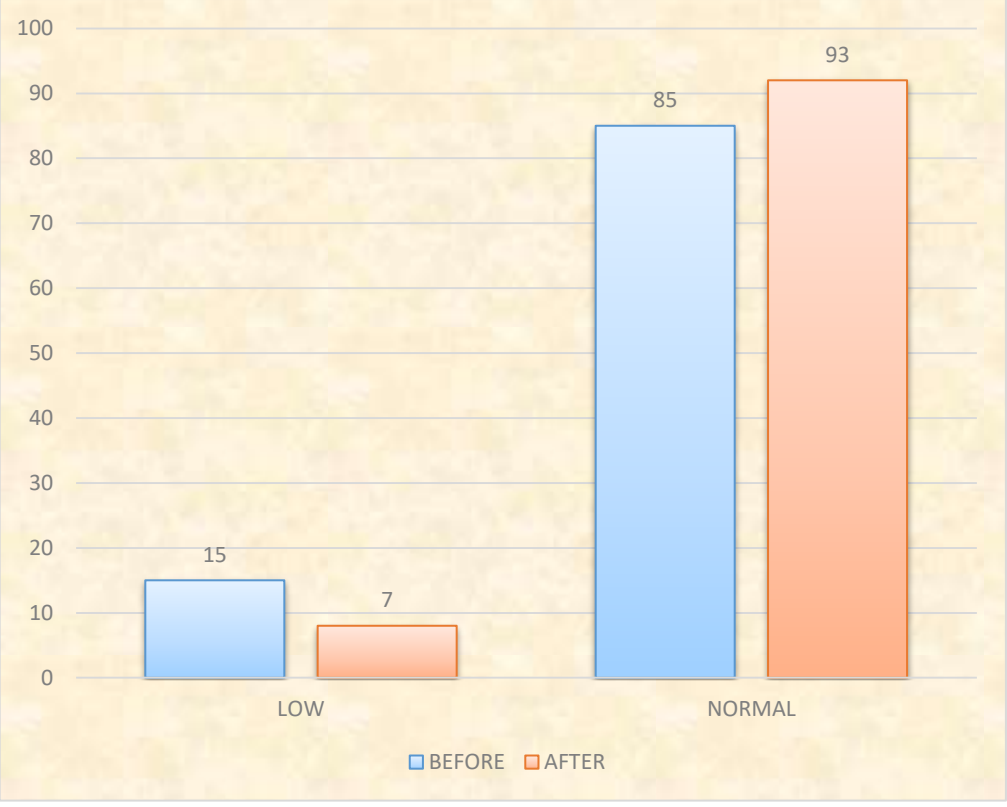


TABLE – 15

QT INTERVAL

QT INTERVAL	BEFORE	AFTER
NORMAL	100	100
ABNORMAL	0	0

CHART – 15

QT INTERVAL

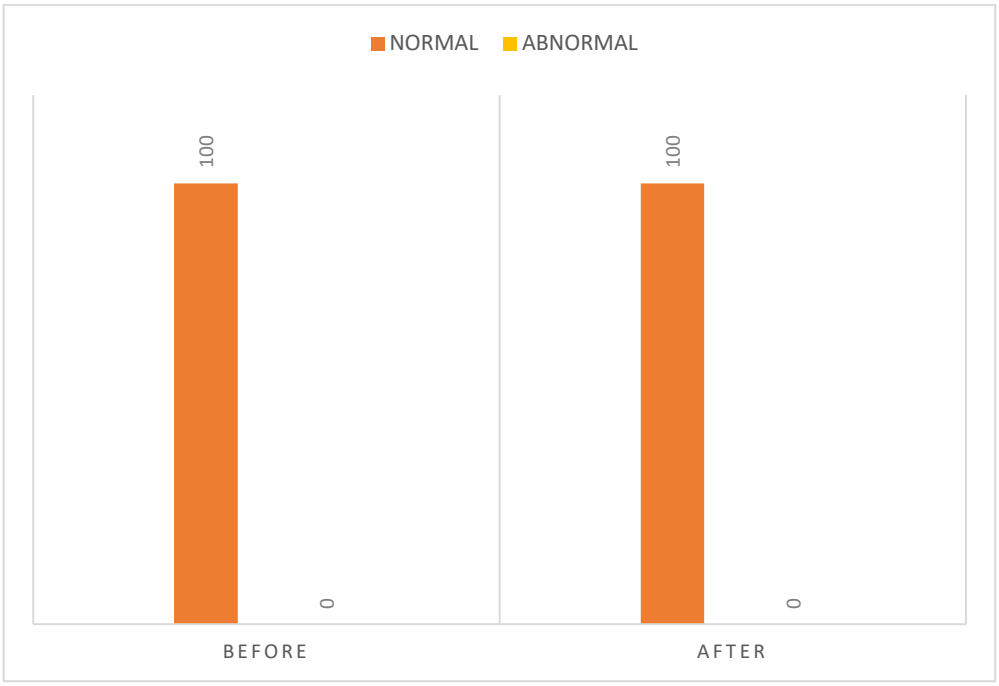


TABLE – 16

ST SEGMENT

ST SEGMENT	BEFORE	AFTER
N0RMAL	100	100
ABN0RMAL	0	0

CHART – 16

ST SEGMENT

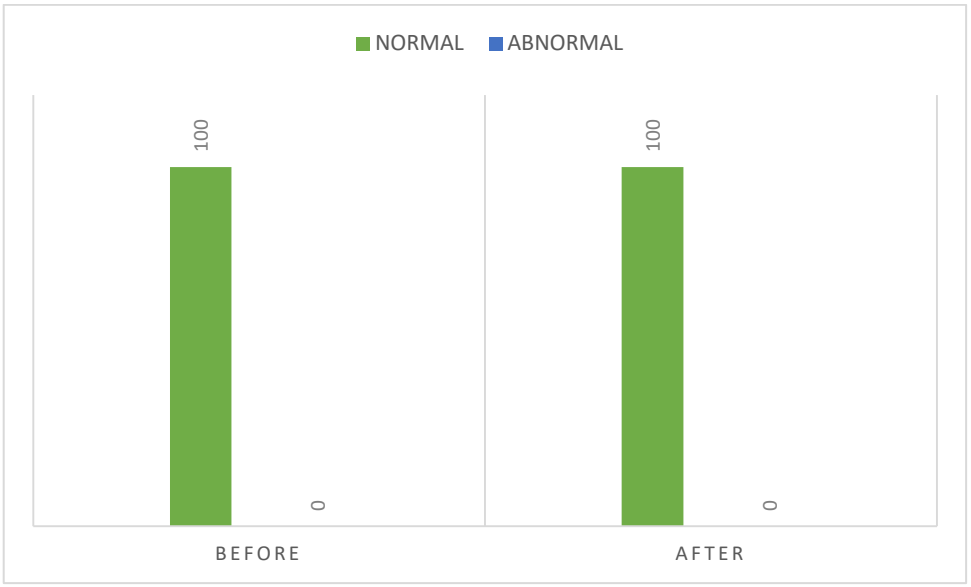


TABLE – 17

T WAVE

T WAVE	BEFORE	AFTER
INVERTED	5	1
NORMAL	95	99
P VALUE - 0.097		
NON SIGNIFICANT		

CHART – 17

T WAVE

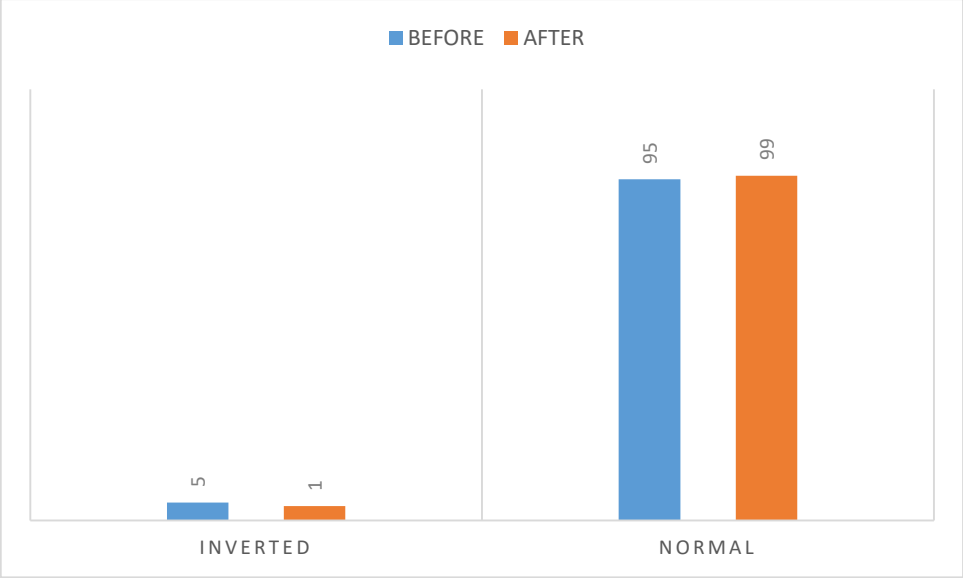


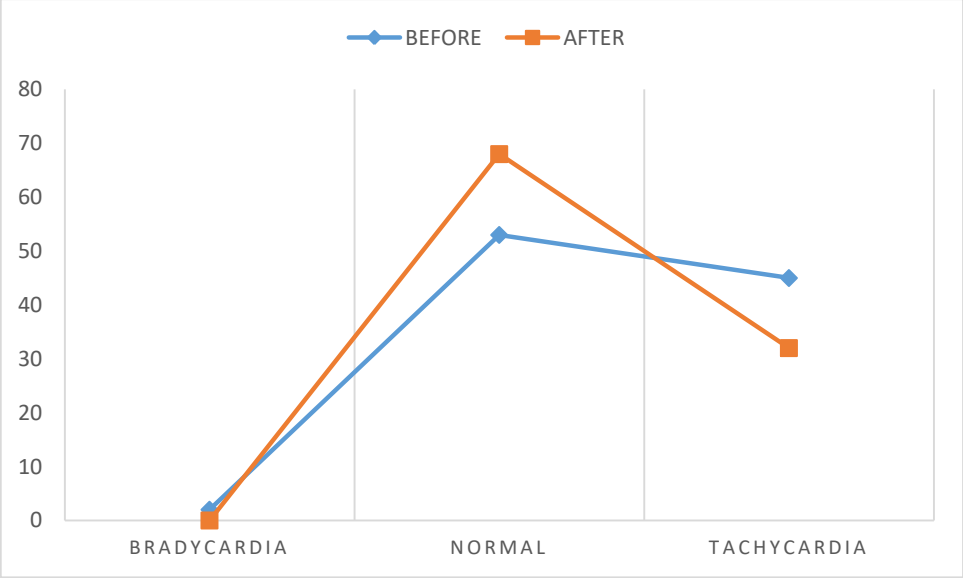
TABLE – 18

HEART RATE

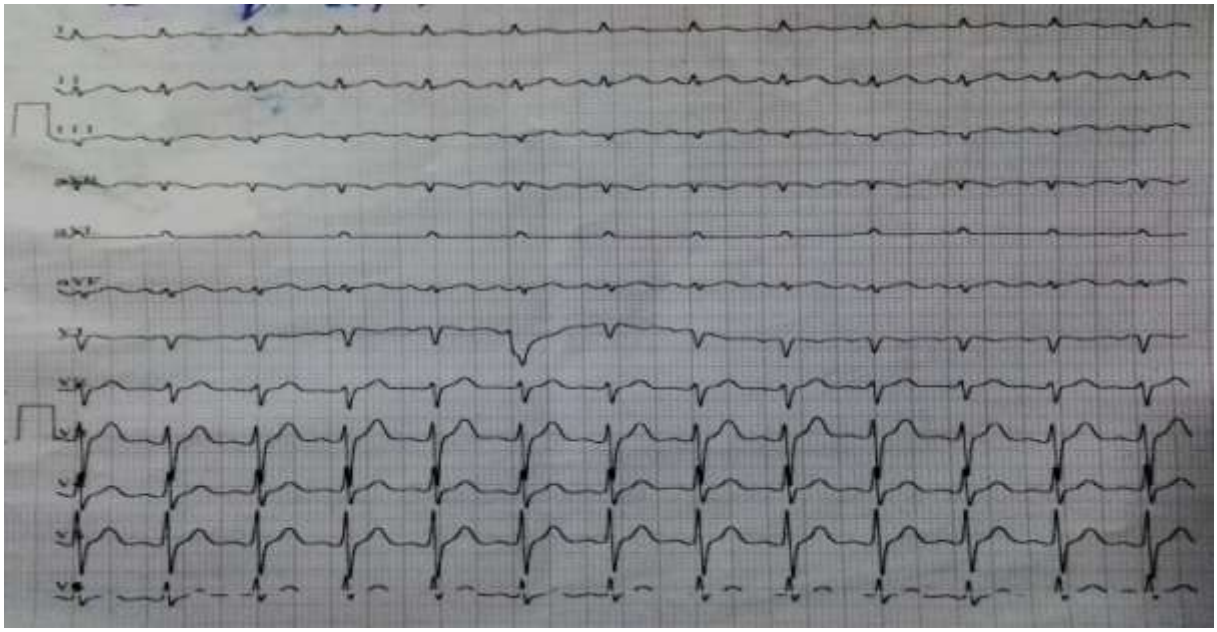
HEART RATE	BEFORE	AFTER
BRADYCARDIA	2	1
TACHYCARDIA	45	13
P VALUE - 0.041		
SIGNIFICANT		

CHART – 18

HEART RATE

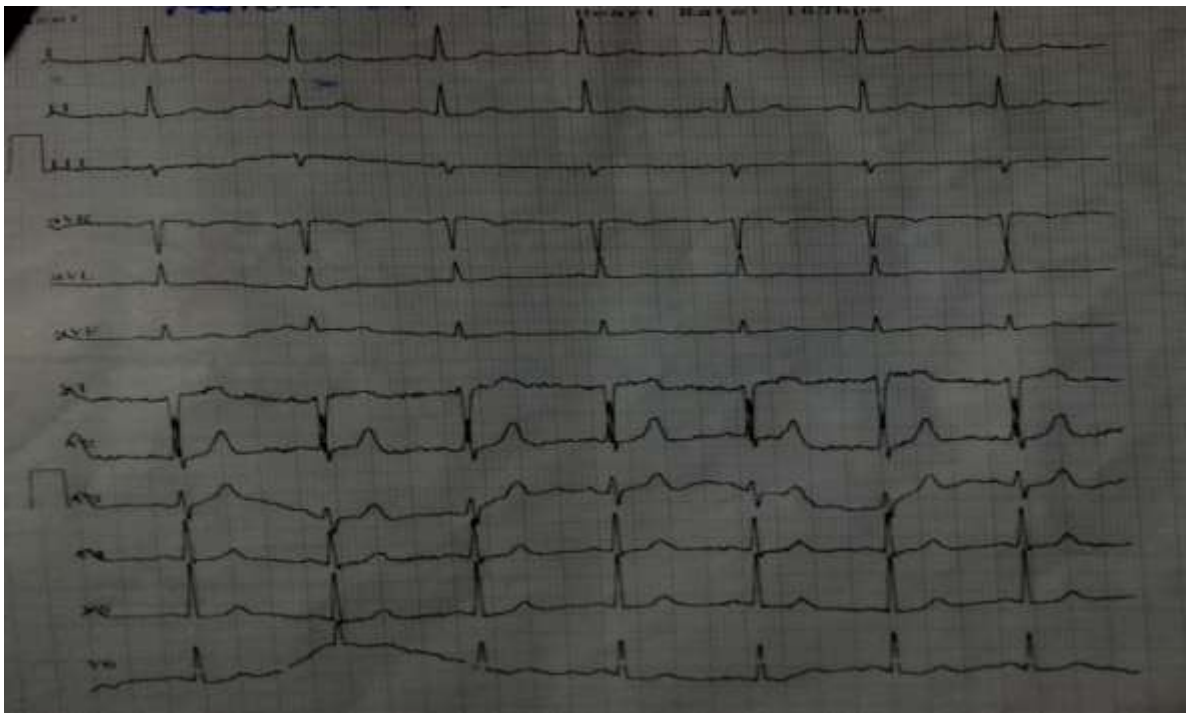


ECG - 1



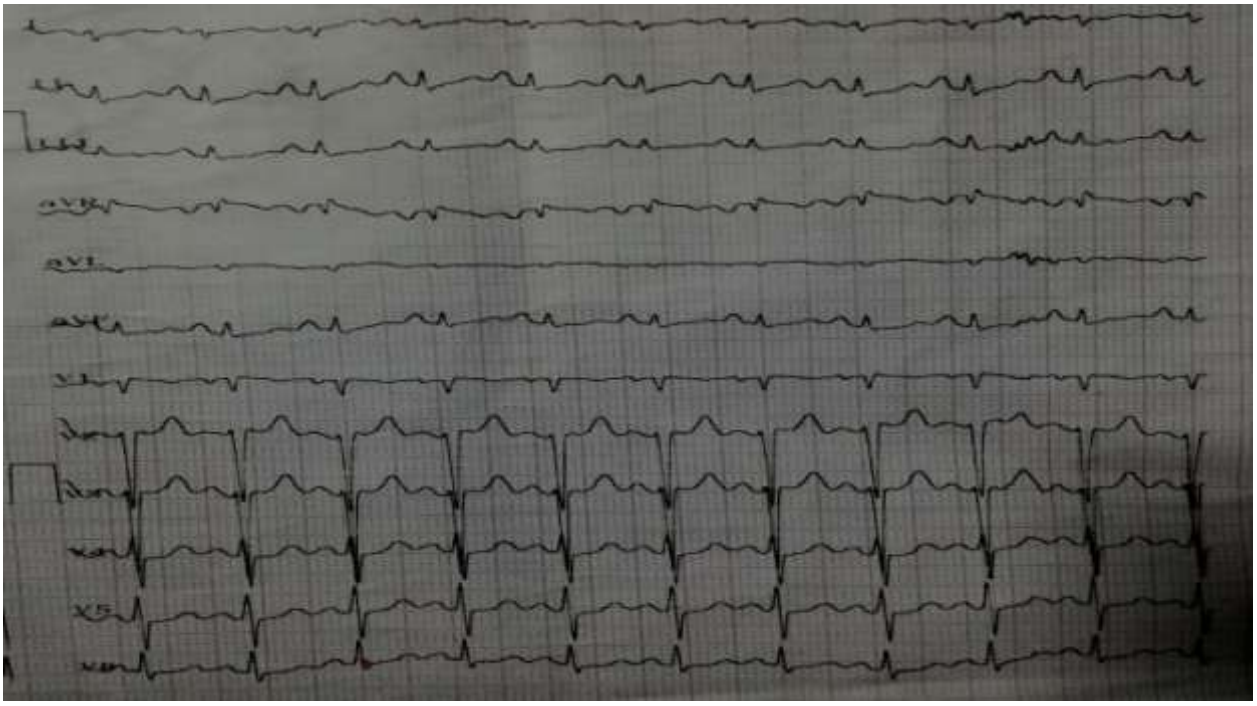
Above ECG showing" low voltage complexes and sinus tachycardia" before anti tuberculous therapy.

ECG - 2



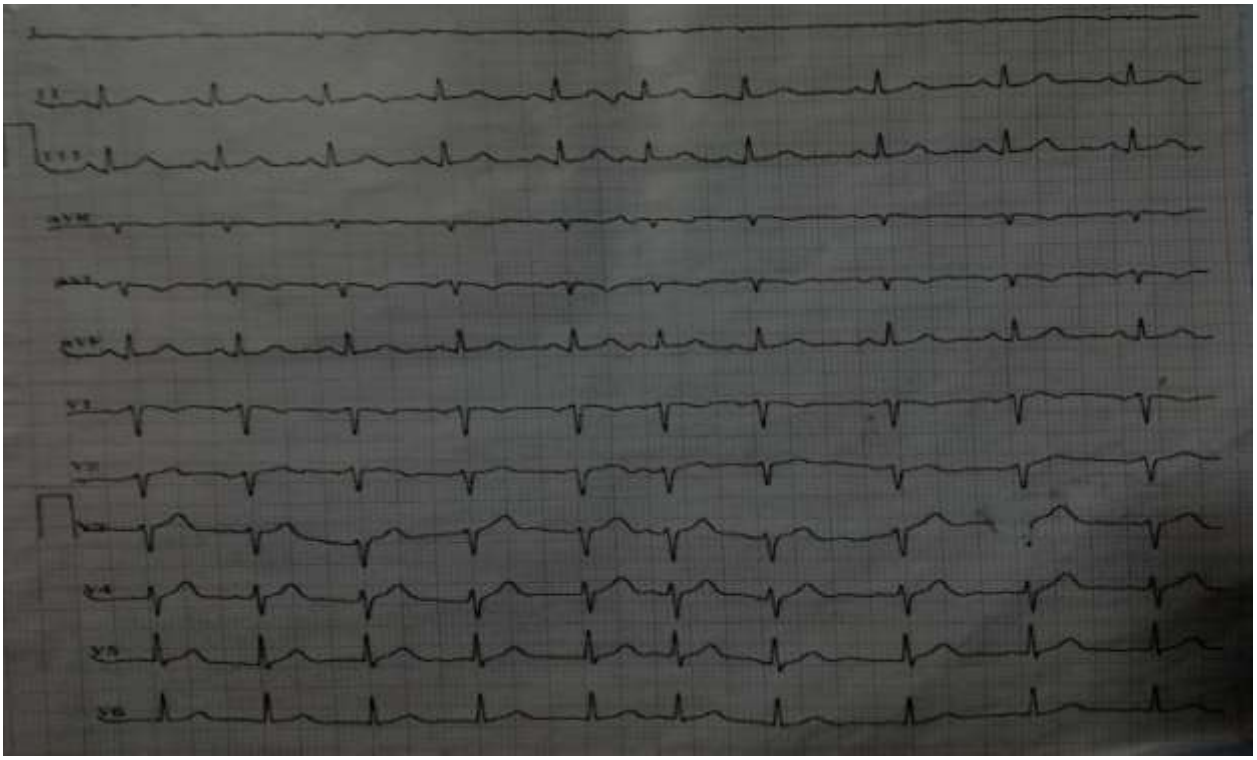
This ECG shows normal sinus rhythm and normal voltage after anti tuberculous therapy

ECG - 3



Above ECG showing" p pulmonale and low voltage complexes"

ECG - 4



Above ECG showing" sinus arrhythmia"

TABLE – 19

RHYTHM

RHYTHM	BEFORE	AFTER
ABNORMAL	10	6
NORMAL	90	94
P VALUE - 0.297		
NON SIGNIFICANT		

CHART – 19

RHYTHM

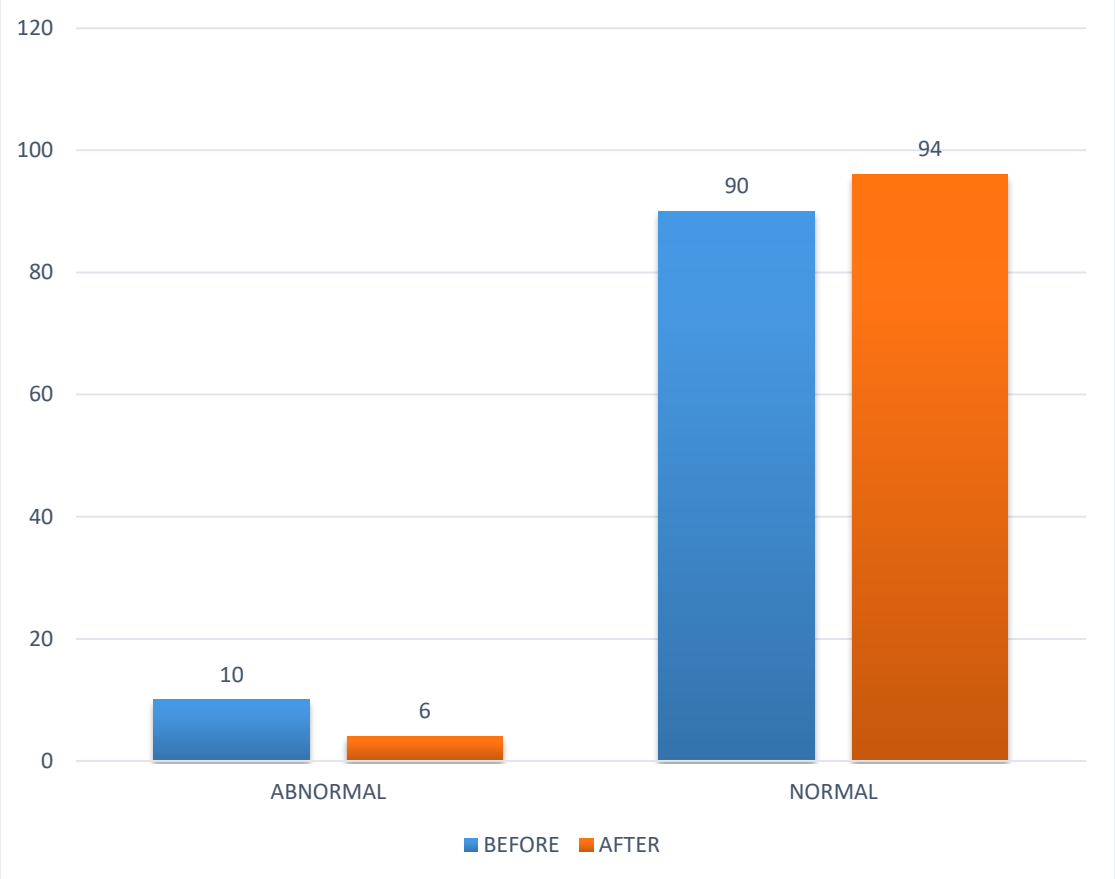


TABLE – 20

VARIOUS RHYTHM CHANGES

RHYTHM	NO OF PATIENTS	PERCENTAGE
APC	3	30%
PVC	1	10%
SINUS ARRYTHMIA	6	60%

CHART – 20

VARI0US RHYTHM CHANGES

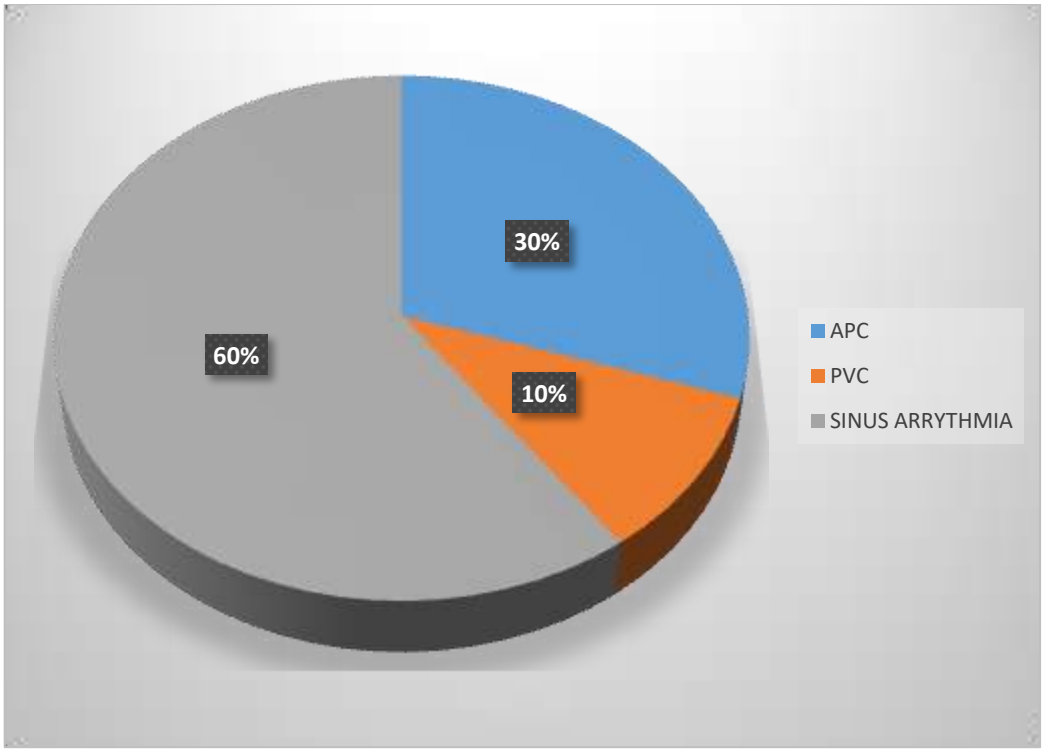


TABLE – 21

AXIS DEVIATION

AXIS DEVIATION	BEFORE	AFTER
RIGHT	11	5
NORMAL	89	95
P VALUE - 0.117		
NON SIGNIFICANT		

CHART – 21

AXIS DEVIATION

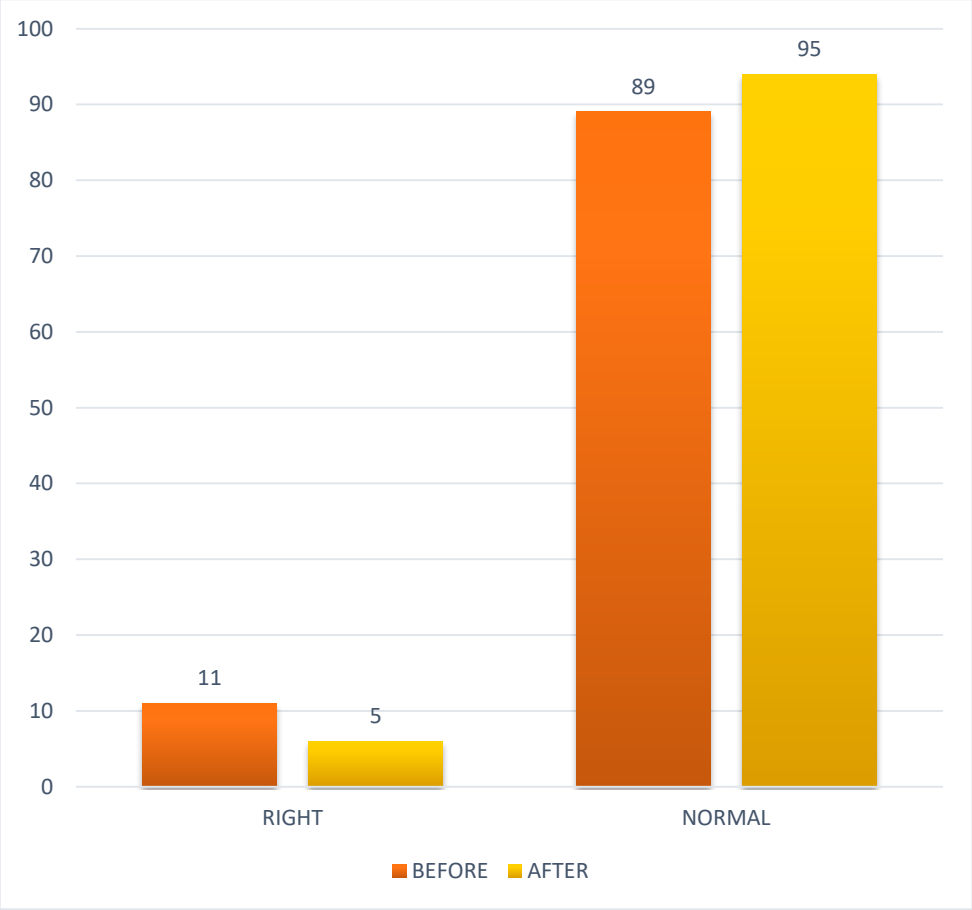


TABLE – 22

CONDUCTION BLOCK

CONDUCTION BLOCK	NO OF PATIENTS	PERCENTAGE
RBBB	4	4%
NORMAL	96	96%

CHART – 22

CONDUCTION BLOCK

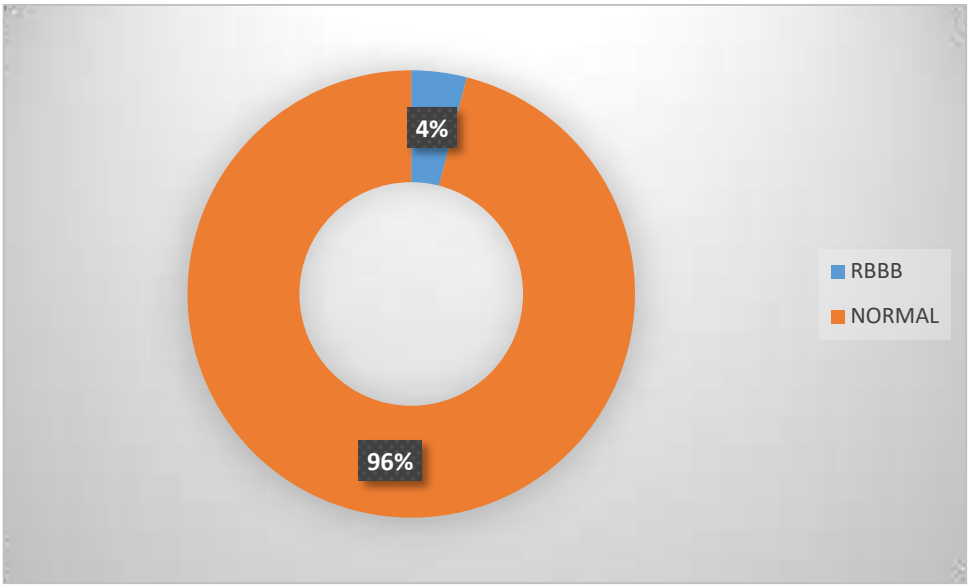


TABLE – 23

TEMPERATURE

TEMPERATURE	NO OF PATIENTS	PERCENTAGE
> 100 DEGREES	64	64%
< 100 DEGREES	36	36%

CHART – 23

TEMPERATURE

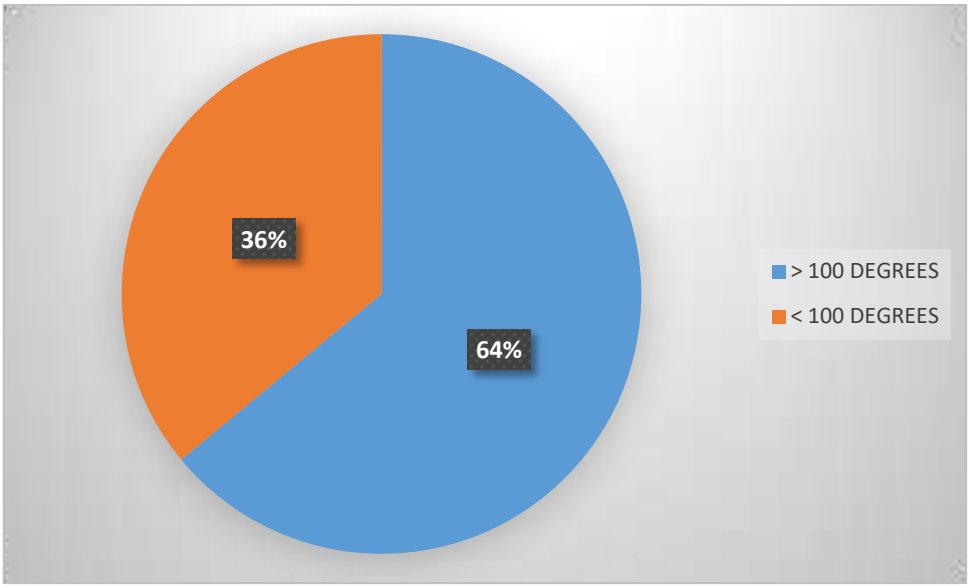


TABLE – 24

MEAN SP02 &HR WITH TEMPERATURE

BEFORE ATT	MEAN	
TEMPERATURE	SP02	HR
> 100 DEGREES	94.9	104.28
< 100 DEGREES	96.27	86
UNPAIRED T TEST		
P VALUE	0.121	0.001

CHART – 24

MEAN SP02 & HR WITH TEMPERATURE

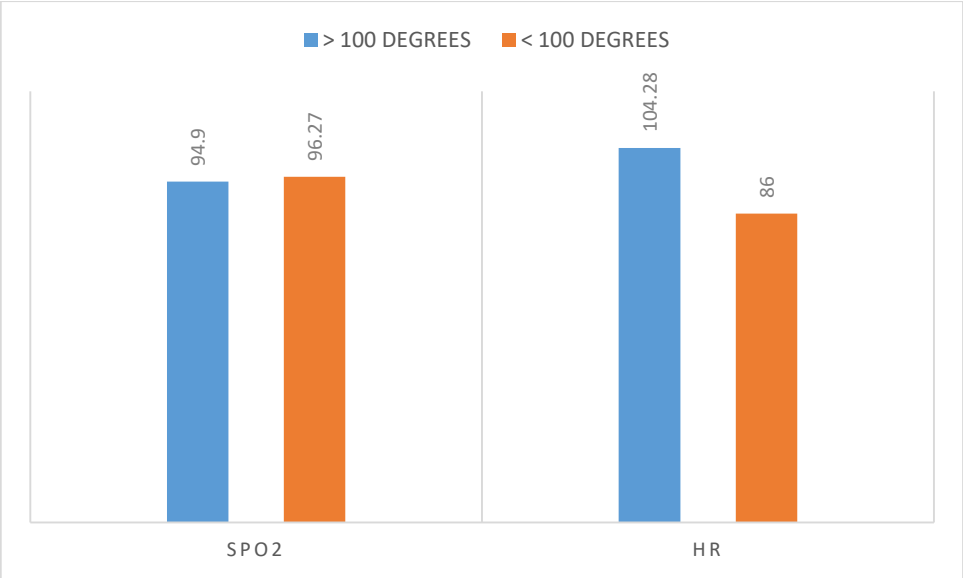


TABLE – 25

POTASSIUM VS ECG CHAGES

MEAN POTASSIUM		
ECG CHANGES	**BEFORE**	**AFTER**
PRESENT	4.01	4.02
ABSENT	3.9	3.94

CHART – 25

POTASSIUM VS ECG CHAGES

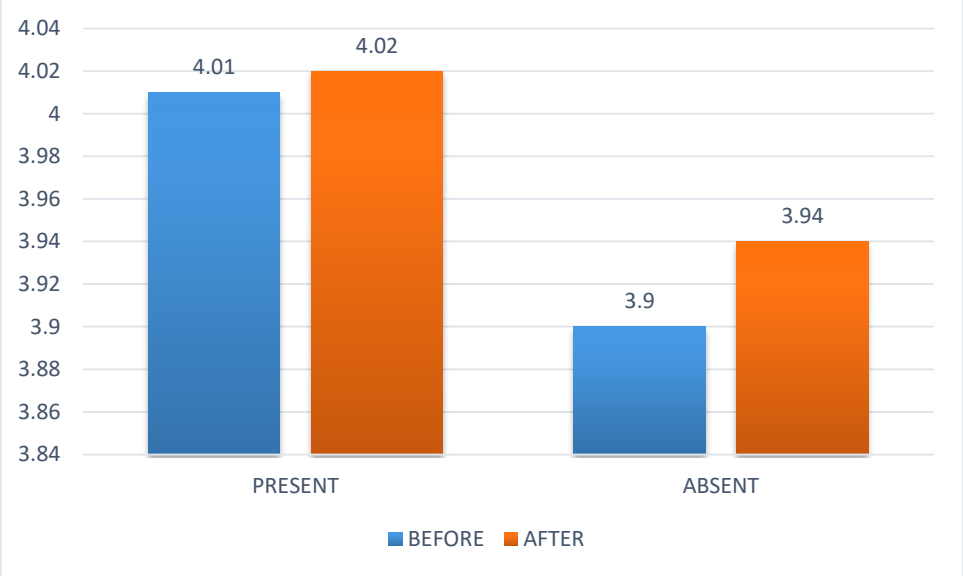


TABLE – 26

CALCIUM VS ECG CHAGES

MEAN CALCIUM		
ECG CHANGES	**BEFORE**	**AFTER**
PRESENT	9.5	9.47
ABSENT	9.62	9.56

CHART – 26

CALCIUM VS ECG CHAGES

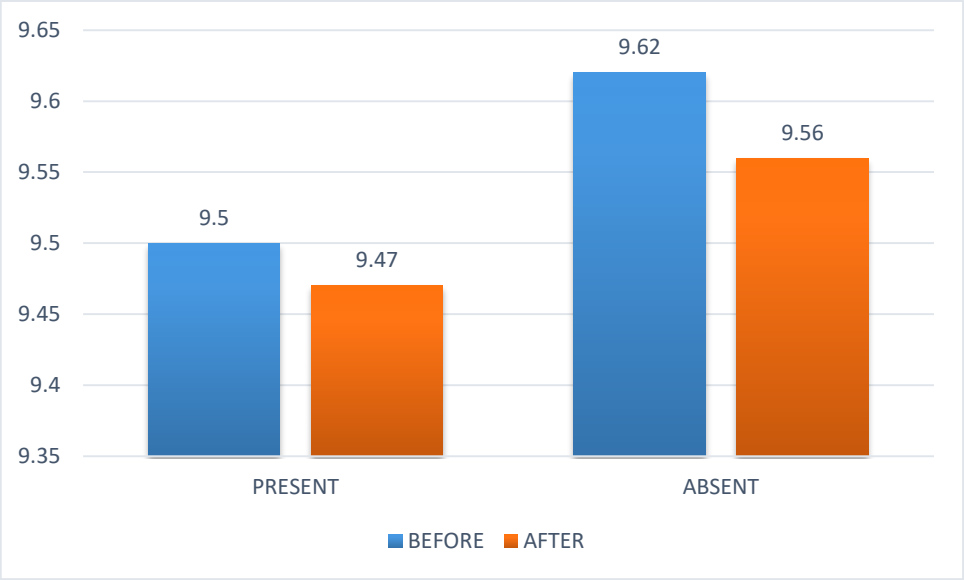


TABLE – 27

HAEMOGLOBIN LEVELS

ATT	HAEMOGLOBIN	
	MEAN	SD
BEFORE	11.53	1.29
AFTER	11.32	1.41
UNPAIRED T TEST		
P VALUE - 0.293		
NON SIGNIFICANT		

CHART – 27

HAEMOGLOBIN LEVELS

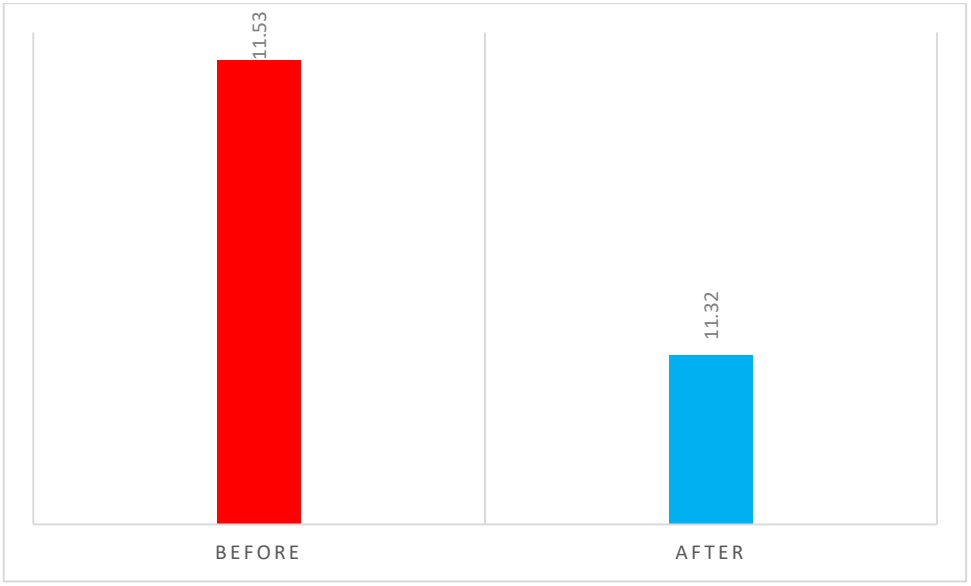


TABLE – 28

MEAN HEART RATE

ATT	HEART RATE	
	MEAN	SD
BEF0RE	98.05	15.49
AFTER	84.11	11.33
UNPAIRED T TEST		
P VALUE - 0.001		
SIGNIFICANT		

CHART – 28

MEAN HEART RATE

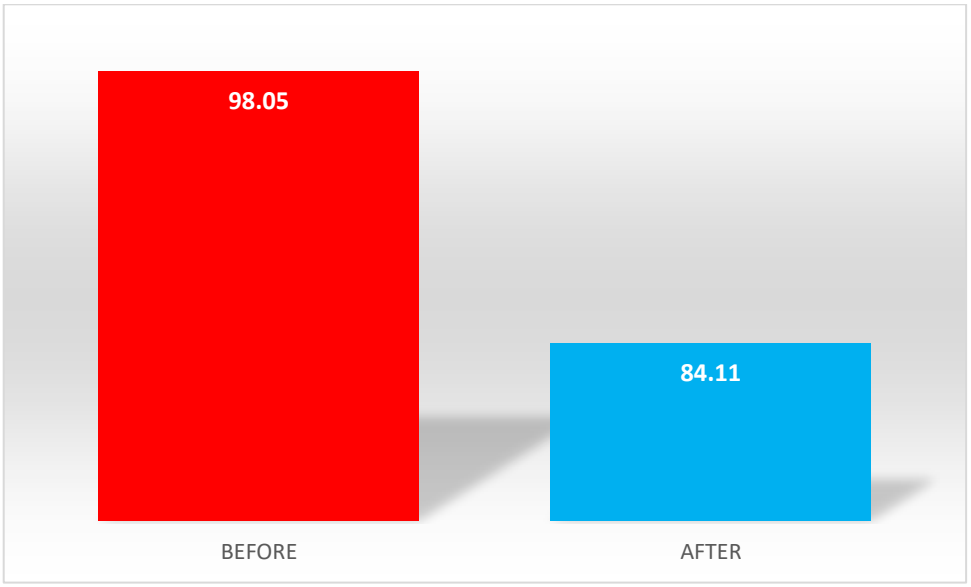


TABLE – 29

MEAN SP02 LEVELS

ATT	SP02	
	MEAN	SD
BEFORE	95.4	1.48
AFTER	97.24	0.91
UNPAIRED T TEST		
P VALUE - 0.001		
SIGNIFICANT		

CHART – 29

MEAN SP02 LEVELS

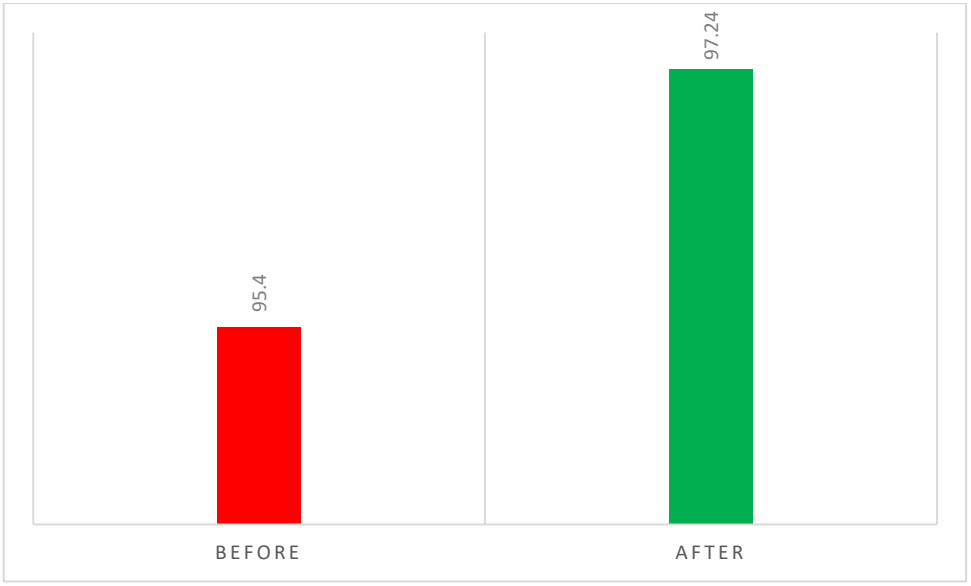


TABLE – 30

MEAN SERUM POTASSIUM

ATT	SERUM POTASSIUM	
	MEAN	SD
BEFORE	3.98	0.25
AFTER	3.97	0.26
UNPAIRED T TEST		
P VALUE - 0.568		
NON SIGNIFICANT		

CHART – 30

MEAN SERUM POTASSIUM

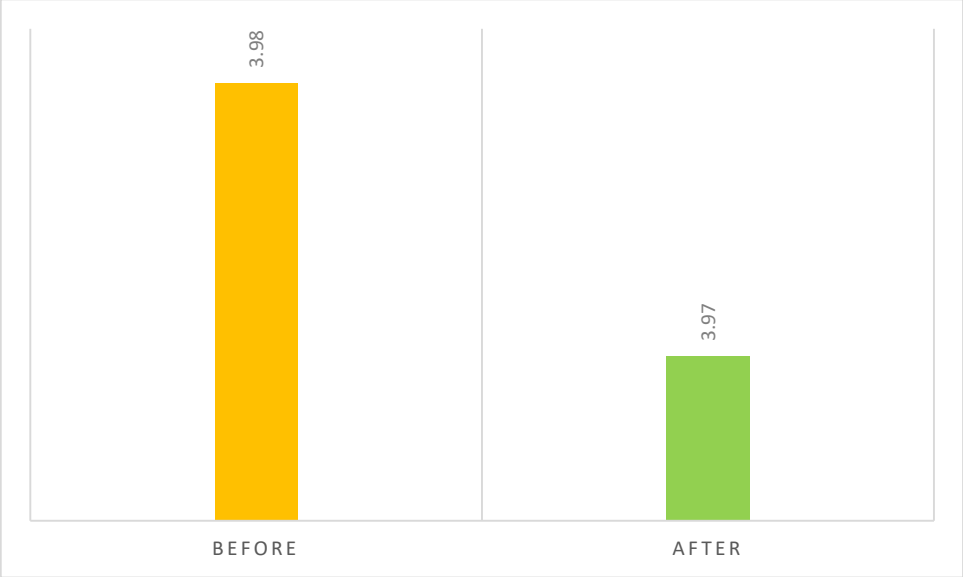


TABLE – 31

MEAN SERUM CALCIUM

ATT	SERUM CALCIUM	
	MEAN	SD
BEFORE	9.54	0.51
AFTER	9.53	0.43
UNPAIRED T TEST		
P VALUE - 0.067		
NON SIGNIFICANT		

CHART – 31

MEAN SERUM CALCIUM

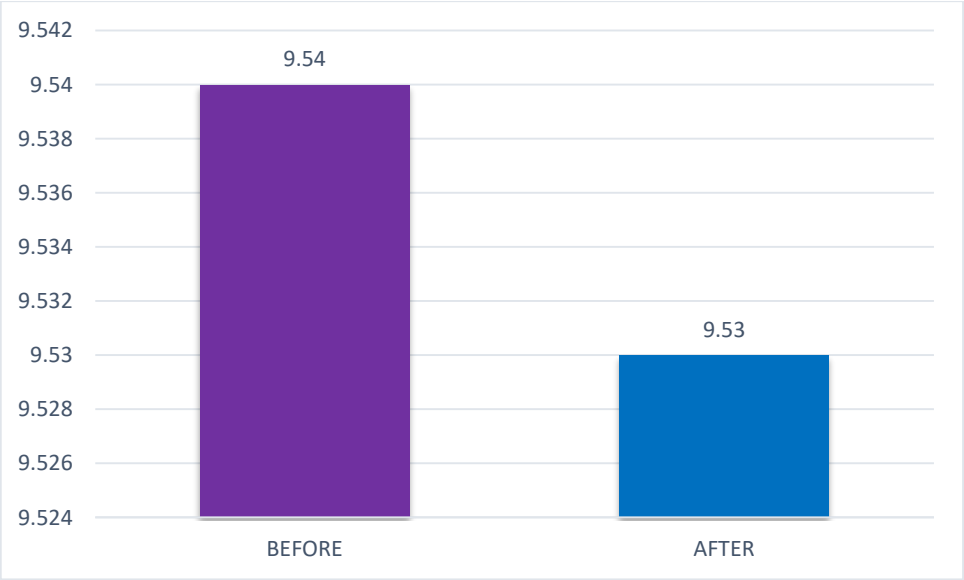


TABLE – 32

TEMPERATURE

ATT	**TEMPERATURE**	
	MEAN	**SD**
BEF0RE	100.24	0.55
AFTER	98.57	0.33
UNPAIRED T TEST		
P VALUE - 0.001		
SIGNIFICANT		

DISCUSSION

In our study 100 cases of new sputum positive pulmonary tuberculosis without any known heart disease were studied the ECG changes before and after anti tuberculous therapy was observed where ECG changes seen in 66 (66%) patients out of 100 and absent 34 patients, study conducted by DASTI MA et al showed ECG changes in 72 (72%) out of 100 in study conducted by S.N GAUR et al studied in 110 patents were 51 (46.4.%) showed ECG abnormalities. IN DASTI ET AL 67% were male and 33% were female. in our study 78% were male and female were 22%. In both of our studies male gender was predominant. Duration of symptoms in S.N GAUR ET AL was between 1 to 5 months. In our study duration less than 6 months was 59% and between 6 to 12 months was 41% mean duration of symptoms in patients with ECG changes 3.51 ± 0.52 (P Value 0.012) which was statistically significant common ECG findings identified are sinus tachycardia in 45% of patients with mean 98.05 ± 15.49 before ATT and mean 84.11 ±11.33 (p value 0.001) which is statistically significant compared to dasti ma et al was 22% and in S.N.Gaur et al study was 72%. P pulmonale was seen 5 % with mean 2.17 ± 0.66 before ATT and mean of 1.98±0.57 AFTER ATT (P Value 0.14) which was statistically significant compared to GAUR ET AL 7 patients had p pulmonale and in study by DASTI MA ET 9 (12.5%) patients had p pulmonale in our study 15 % had low voltage complexes before ATT and 8 patients AFTER ATT (p value 0.091) which was not statistically significant in our study p wave axis $+9o^{o}$ seen in 11 patients before ATT and in 4 patients after ATT

which was not statistically significant with p value o.602. in GAUR ET AL 14 patients had p wave axis + 90º before ATT and in 5 patents after ATT.

Sl. No.	ECG Changes	No. of abnormalities before ATT (n = 100)	No. of Abnormalities return to normal after ATT (n = 100)	Percentage of reversion after ATT
1	Sinus tachycardia	45	32	71%
2	Sinus bradycardia	2	1	50%
3	P pulmonale	5	0	0%
4	Low voltage complex	15	8	53%
5	P wave axis +90º	11	4	36.3%
6	Qrs axis + 90º	11	6	54.5%
7	Prolonged PR interval	2	0	0%
8	T inversion	5	4	80%
9	Sinus arrythmia	10	4	40%

Sl. No.	ECG Changes	No. of Abnormalities in current study	No. of abnormalities in DASTI MA et al	No. of abnormalities in GAUR et al
1	Sinus tachycardia	45	22	47
2	Sinus bradycardia	02	03	01
3	P pulmonale	05	09	07
4	Low voltage complex	15	04	20
5	P wave axis +90º	11	-	14
6	Qrs axis + 90º	11	05	10
7	Prolonged PR interval	02	07	-
8	T inversion	05	-	06
9	Sinus arrythmia	10	-	12

In current study ECG changes seen are sinus tachycardia (45%), low voltage complex (15%), p wave axis +90º (11%), Qrs axis+90º (11%), sinus arrhythmia (10%), p pulmonale (5%), pr prolongation (2%), t wave inversion (5%) where sinus tachycardia (71%), low voltage complexes (53%), t wave inversion (80%) reverted to normal after anti-tuberculous therapy.

CONCLUSION

Pulmonary tuberculosis is one of the common diseases in clinical practice which affects any system and cardiac involvement is one of its complications **,**it can affect any cardiac region but mainly affects pericardium and less frequently myocardium and endocardium. In our present study sinus tachycardia, low voltage complexes, Qrs axis+9oº, p pulmonale ,sinus arrhythmia are the findings observed and sinus tachycardia, low voltage complexes, t wave inversion reverted to normal after anti-tuberculous therapy, therefore we should be aware in patients presenting with patients presenting with abnormal features like chest pain and breathlessness and with abnormal ecg findings ,where such individuals should be further evaluated with echocardiography and ensure completing ATT since there is reversal of ECG changes after completing ATT, hence earlier development of irreversible cardiac complications can be prevented.

ANNEXURE - I

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ANNEXURE - II

PROFORMA

Name :

Age :

Sex :

OP No :

Department :

Hospital :

Address :

Contact No :

Occupation :

Chief complaints

H/O Cough with expectoration

H/O Fever

H/O LOW/LOA

H/O Breathing difficulty

H/O Fatigue

H/O Weight loss inspite of good apptite.

Examination

General Examination:

Built and nourishment, pallor, icteric, cyanosis, clubbing, lymphadenopathy, pedal edema.

Systemic Examination: CVS, ABDOMEN, CNS.

Examination of Respiratory system: Inspection, Palpation, Percussion, Auscultation.

Vitals : blood pressure, pulse rate, spo2

Investigations

ELECTROCARDIOGRAPHY- during presentation and follow up ECG after 6 months

CXR-PA View

Sputum AFB

Others:

Complete Hemogram,

Renal Function Test,

Serum electrolytes,

Liver Function Test,

Blood Sugar,

Lipid Profile.

Treatment

CAT-1 ATT Treatment

Follow up ECG at end of 6 months

ANNEXURE - III

CONSENT FORM

STUDY TITLE "PROSPECTIVE STUDY ON ELECTROCARDIOGRAPHIC CHANGES IN SPUTUM POSITIVE PULMONARY TUBERCULOSIS BEFORE AND AFTER ANTITUBERCULOUS THERAPY"

I……, hereby give consent to participate in the study conducted by Dr.J.BALASUBRAMANIYAM, Post graduate in general medicine, Coimbatore Medical College, Coimbatore and to use my personal clinical data and the result of investigations for the purpose of analysis and to study the nature of the disease, I also give consent to give my sample for further investigations. I also learn that there is no additional risk in this study. I also give my consent for my investigator to publish the data in any forum or journal

Signature/ Thumb impression of the patient:

Patient Name & Address: **Signature of the investigator:**

Place:

Date: **Signature of the guide:**

ANNEXURE - IV

ஒப்புதல் படிவம்

பெயர் :

வயது :

பாலினம் :

முகவரி:

கோவை அரசு மருத்துவக்கல்லூரி மருத்துவமனையில் மருத்துவர் **ஐ. பாலசுப்பிரமணியம்** தலைமையில் நடைபெறும் இந்த ஆய்வில் முழு சம்மதத்துடன் கலந்துகொள்ள சம்மதிக்கிறேன். இந்த ஆய்வில் என்னை பற்றி விவரங்களை பாதுகாப்புடன் இந்த ஆய்வில் வெளியிட ஆட்சேபணை இல்லை என்று தெரிவித்துக் கொள்கிறேன். எந்த நேரத்திலும் ஆய்வில் இருந்து எந்த நேரத்திலும் விலக்கிக்கொள்ளும் உரிமை உண்டு என்று அறிவேன்.

இடம் :

தேதி:

கைகெயாப்பம் / ரேகை

ANNEXURE - V

KEY TO MASTER CHART

NSR	-	Normal Sinus Rhythm
A	-	After ATT
B	-	Before ATT
PVC	-	Premature Ventricular Contraction
APC	-	Atrial Premature Contraction
RBBB	-	Right Bundle Branch Block

ANNEXURE - VI

MASTER CHART

S.NO	NAME	AGE	SEX	DURATION OF SYMPTOM	ATT	P WAVE DURATION	P WAVE AXIS	P WAVE AMPLITUDE	P-R INTERVAL	QRS DURATION	QRS AXIS	QRS AMPLITUDE	QT INTERVAL	ST SEGMENT	T WAVE	HEART RATE	RHYTHM	AXIS	HAEMOGLOBIN	BLOOD PRESSURE	SR.POTASSIUM	SR.CALCIUM	TEMPERATURE	SPO2	CONDUCTION BLOCK
1	DURAIRAJ	35	M	4	B	N	N	2	N	N	N	N	N	N	N	115	NSR	N	10.4	110/70	4	9	101.7	95	-
					A	N	N	1	N	N	N	N	N	N	N	90	NSR	N	10.2	100/60	3.7	8.9	98.6	97	-
2	ANANDAN	46	M	5	B	N	N	2.5	N	N	N	N	N	N	INVERTED	120	NSR	N	10	110/70	3.6	10	101	94	-
					A	N	N	2	N	N	N	N	N	N	INVERTED	108	NSR	N	10.1	110/60	4.1	9.7	99	96	-
3	DINAKARAN	28	M	9	B	N	(+)90	3	N	N	(+)90	LOW	N	N	N	110	NSR	RIGHT	10.2	120/70	3.8	9.8	100.6	93	-
					A	N	(+)90	3	N	N	N	N	N	N	N	87	NSR	N	9.7	110/70	3.7	9.5	99.2	94	-
4	KALAVATHI	45	F	6	B	N	(+)90	2	N	N	(+)90	N	N	N	N	90	NSR	RIGHT	11.6	100/80	3.9	10.2	99.7	93	-
					A	N	N	1	N	N	N	N	N	N	N	75	NSR	N	11.5	110/80	4	9.8	98.6	97	-
5	VEERATHAL	70	F	1	B	N	N	2	N	N	N	N	N	N	N	85	NSR	N	12	130/80	3.8	9.9	99.9	86	-
					A	N	N	2.5	N	N	N	N	N	N	N	80	NSR	N	12.2	90/60	3.6	10.2	98.5	98	-
6	SOMASUNTHARAM	55	M	1	B	N	N	2.5	N	N	N	N	N	N	N	90	NSR	N	13	120/80	3.6	9.7	99.8	97	-
					A	N	N	2	N	N	N	N	N	N	N	78	NSR	N	12.6	130/80	3.7	9.8	98.4	98	-
7	MANIKAM	52	M	4	B	N	N	2.5	N	N	N	N	N	N	N	87	NSR	RIGHT	11.7	110/70	4	9.5	100.2	94	-
					A	N	N	2.5	N	N	N	N	N	N	N	89	NSR	N	10.5	100/60	4.2	9.6	98.7	96	-
8	MAHALI	52	M	2	B	N	N	2.5	N	N	N	N	N	N	N	88	NSR	N	10.4	115/60	3.7	9.9	100.1	97	-
					A	N	N	2	N	N	N	N	N	N	N	80	NSR	N	10.6	125/70	4	9.3	98.6	97	-
9	THIRUMOORTHI	49	M	8	B	N	N	2	N	N	N	LOW	N	N	N	80	NSR	N	11	100/65	3.6	8.8	99.8	96	-
					A	N	N	2	N	N	N	LOW	N	N	N	75	NSR	N	10.8	110/75	4	8.9	98.5	98	-
10	VENKATESH	52	M	4	B	N	N	1	N	N	N	N	N	N	N	85	NSR	N	10.9	120/75	4	8.5	100	97	-
					A	N	N	2	N	N	N	N	N	N	N	70	NSR	N	10.8	110/80	4.2	8.9	98.1	98	-
11	SAKUNTHALA	60	F	1	B	N	N	1	N	N	N	N	N	N	N	86	NSR	N	11.5	110/70	3.6	10.1	99.5	97	-
					A	N	N	2.5	N	N	N	N	N	N	N	78	NSR	N	10.9	100/60	3.7	9.8	98.5	97	-
12	TAMILARASAN	21	M	1	B	N	N	2	N	N	N	N	N	N	N	80	NSR	N	13	110/70	3.8	10.5	99.9	96	-
					A	N	N	1.5	N	N	N	N	N	N	N	75	NSR	N	13	110/60	4.1	9.7	98.4	98	-
13	HASAN	50	M	3	B	N	N	2	N	N	N	N	N	N	N	120	NSR	N	10	120/70	4	9.8	100.4	93	-
					A	N	N	2	N	N	N	N	N	N	N	88	NSR	N	10.2	110/70	4.2	9.5	99	97	-
14	NATARAJ	56	M	4	B	N	N	1	PROLONGED	N	N	N	N	N	N	90	NSR	N	11.7	100/80	3.6	8.9	100.1	96	-
					A	N	N	2	PROLONGED	N	N	N	N	N	N	80	NSR	N	11.4	110/80	4	8.8	98.4	98	-
15	SHANMUGAM	63	M	4	B	N	N	2.5	N	N	N	N	N	N	INVERTED	115	NSR	N	13	130/80	4	9.8	100.4	94	-
					A	N	N	2.5	N	N	N	N	N	N	N	89	NSR	N	13.3	90/60	4	9.7	98.8	97	-
16	ARUMUGAM	57	M	5	B	N	N	3	N	N	N	LOW	N	N	N	116	NSR	N	10.7	120/80	4.1	9.9	100.6	95	-
					A	N	N	3	N	N	N	LOW	N	N	N	110	NSR	N	10.5	130/80	4.3	9.8	99	95	-
17	BABU	53	M	1	B	N	N	2	N	N	N	N	N	N	N	92	(PVC)	N	12	110/70	4.1	10.5	100.2	97	-
					A	N	N	2.5	N	N	N	N	N	N	N	80	(PVC)	N	12.7	100/60	3.6	10	98.5	97	-
18	SAMPATH	50	M	6	B	N	N	2	N	N	N	LOW	N	N	N	85	NSR	N	11.7	115/60	3.8	9.6	99	95	RBBB
					A	N	N	2	N	N	N	LOW	N	N	N	78	NSR	N	12	125/70	4	9.5	98.4	97	RBBB
19	CHINNASAMY	75	M	4	B	N	N	2.5	N	N	N	N	N	N	N	110	NSR	N	10.1	100/65	4.1	8.6	100.4	95	-
					A	N	N	1	N	N	N	N	N	N	N	83	NSR	N	10	110/75	3.8	8.7	98.7	97	-
20	SIVAKUMAR	32	M	2	B	N	N	2	N	N	N	N	N	N	N	117	SINUS ARRYTHMIA	N	10	120/75	3.4	8.8	101	93	-
					A	N	N	1	N	N	N	N	N	N	N	88	NSR	N	10.2	110/80	3.6	8.9	99.2	96	-
21	SENTHILKUMAR	45	M	9	B	N	(+)90	2.5	N	N	(+)90	N	N	N	N	90	NSR	RIGHT	11.2	110/70	3.9	9.7	100.1	97	RBBB
					A	N	(+)90	2	N	N	(+)90	N	N	N	N	80	NSR	RIGHT	11.4	100/60	4.1	9.5	98.6	97	RBBB
22	LAKSHMANAN	35	M	5	B	N	N	1.5	N	N	N	LOW	N	N	N	88	NSR	N	12.4	110/70	4	9	100	96	-
					A	N	N	2	N	N	N	LOW	N	N	N	75	NSR	N	12	110/60	4.2	9.2	98.8	98	-
23	SELVANATHAN	55	M	1	B	N	N	3	N	N	N	N	N	N	N	110	NSR	N	10.3	120/70	3.9	9.3	101.1	95	-
					A	N	N	3	N	N	N	N	N	N	N	88	NSR	N	10	110/70	4	9.5	99	97	-
24	PALANIYAMMAL	40	F	5	B	N	N	2	N	N	(+)90	LOW	N	N	N	106	NSR	RIGHT	10.4	100/80	4.2	9.7	100.1	94	-
					A	N	N	2	N	N	N	N	N	N	N	84	NSR	N	10	110/80	4.4	9.5	98.2	97	-

25	SANGILIYAMMAL	75	F	2	B	N	N	2.5	N	N	N	N	N	N	N	80	NSR	N	13.2	130/80	3.6	10	99	96	-
					A	N	N	2	N	N	N	N	N	N	N	75	NSR	N	10.5	90/60	3.9	9.3	98.4	98	-
26	KRISHNAN	65	M	2	B	N	N	3	N	N	N	N	N	N	INVERTED	117	NSR	N	10.4	120/80	4	9.8	100.3	93	-
					A	N	N	3	N	N	N	N	N	N	N	86	NSR	N	11.5	130/80	4.3	9.6	98.6	96	-
27	RAJENDRAN	51	M	1	B	N	N	2.5	N	N	N	N	N	N	N	86	NSR	N	13.9	110/70	3.6	9.5	100.1	96	-
					A	N	N	2	N	N	N	N	N	N	N	75	NSR	N	12	100/60	3.9	9.3	99	98	-
28	SRINIVASAN	36	M	7	B	N	(+)90	3.5	N	N	(+)90	N	N	N	N	109	NSR	RIGHT	11.1	115/60	4	9.2	100.4	93	RBBB
					A	N	(+)90	3.5	N	N	(+)90	N	N	N	N	86	NSR	RIGHT	10	125/70	4.1	9.7	98.2	97	RBBB
29	KANNAN	44	M	1	B	N	N	1	N	N	N	N	N	N	N	90	(APC)	N	10.4	100/65	4.2	9.9	100.4	96	-
					A	N	N	2	N	N	N	N	N	N	N	75	NSR	N	10.3	110/75	4.5	9.1	98.5	98	-
30	RAMACHANDRAN	34	M	5	B	N	90	1	N	N	N	LOW	N	N	N	112	NSR	N	10.5	120/75	4.2	10.2	100.3	96	-
					A	N	N	2.5	N	N	N	LOW	N	N	N	87	NSR	N	13.4	110/80	4.2	9.5	99	96	-
31	RANJITH	25	M	2	B	N	N	2	N	N	N	N	N	N	N	88	NSR	N	14	110/70	3.8	8.7	100.2	96	-
					A	N	N	1.5	N	N	N	N	N	N	N	79	NSR	N	14.5	100/60	3.7	8.9	98.5	98	-
32	LAKSHMI	40	F	1	B	N	N	2	N	N	N	N	N	N	N	118	NSR	N	14.1	110/70	4	9.1	100.6	95	-
					A	N	N	2	N	N	N	N	N	N	N	88	NSR	N	12	110/60	4.2	9.8	98.4	95	-
33	KARTHIK	35	M	2	B	N	N	1	N	N	N	N	N	N	N	90	NSR	N	12.6	120/70	3.9	10.4	99	96	-
					A	N	N	2	N	N	N	N	N	N	N	78	NSR	N	10.3	110/70	4	10.6	98.2	98	-
34	KALAVATHI	35	F	5	B	N	(+)90	2.5	N	N	N	N	N	N	N	116	NSR	N	10.2	100/80	4.2	9	100	94	-
					A	N	(+)90	2.5	N	N	N	N	N	N	N	108	NSR	N	10	110/80	3.6	9.3	98.6	96	-
35	KARTHIKA	21	F	4	B	N	N	2	N	N	N	N	N	N	N	110	NSR	N	10.2	130/80	3.8	9.7	101	95	-
					A	N	N	2	N	N	N	N	N	N	N	83	NSR	N	14	90/60	3.7	9.4	99	97	-
36	BALAN	29	M	2	B	N	N	2	N	N	N	N	N	N	N	85	NSR	N	14.2	120/80	4	9.8	99.8	96	-
					A	N	N	2.5	N	N	N	N	N	N	N	75	NSR	N	9.7	130/80	4.2	9.5	97.9	98	-
37	CHINNASAMY	47	M	6	B	N	(+)90	2	N	N	N	N	N	N	N	112	NSR	N	10.2	110/70	3.8	9	100.6	94	-
					A	N	N	2	N	N	N	N	N	N	N	88	NSR	N	12	100/60	3.7	9.3	98.7	96	-
38	CHANDRASEKAR	53	M	1	B	N	N	2.5	N	N	N	N	N	N	N	86	NSR	N	13.2	115/60	3.9	9.6	100	97	-
					A	N	N	1	N	N	N	N	N	N	N	78	NSR	N	10.1	125/70	4	9.8	98.6	98	-
39	AJMAL	36	M	8	B	N	N	2	N	N	(+)90	LOW	N	N	N	121	NSR	RIGHT	10	100/65	4.2	9.9	100.6	94	RBBB
					A	N	N	1	N	N	(+)90	LOW	N	N	N	109	NSR	RIGHT	13	110/75	4.4	9.7	99.1	98	RBBB
40	JEYASELAN	38	M	2	B	N	N	2.5	N	N	N	N	N	N	N	90	SINUS ARRYTHMIA	N	13.4	120/75	3.9	8.9	99.8	97	-
					A	N	N	2	N	N	N	N	N	N	N	85	SINUS ARRYTHMIA	N	12.4	110/70	4.1	8.8	98.1	97	-
41	PALANISAMY	48	M	1	B	N	N	1.5	N	N	N	N	N	N	N	53	NSR	N	12.7	100/60	4.3	8.3	99	96	-
					A	N	N	2	N	N	N	N	N	N	N	55	NSR	N	10	110/70	3.8	9.4	98.6	96	-
42	RAJENDRAN	45	M	5	B	N	N	2	N	N	N	N	N	N	N	111	NSR	N	10.4	110/60	3.6	9.3	100.6	92	-
					A	N	N	1	N	N	N	N	N	N	N	84	NSR	N	13	120/70	3.9	9.7	99.1	95	-
43	ARUMUGAM	60	M	3	B	N	N	2	N	N	N	LOW	N	N	N	88	NSR	N	10.6	110/70	4.1	9.2	100	97	-
					A	N	N	2.5	N	N	N	N	N	N	N	75	NSR	N	12.6	100/80	4.3	9.8	98.1	99	-
44	NAVINKUMAR	24	M	1	B	N	N	2.5	N	N	N	N	N	N	N	90	NSR	N	13	110/80	4	9.9	100.4	97	-
					A	N	N	2	N	N	N	N	N	N	N	80	NSR	N	14	130/80	4.1	9.3	98.7	97	-
45	STALIN	29	M	2	B	N	N	3	PROLONGED	N	N	N	N	N	N	85	NSR	N	13.1	90/60	4	8.9	99.7	97	-
					A	N	N	3	PROLONGED	N	N	N	N	N	N	78	NSR	N	13.8	120/80	3.8	8.8	98.4	98	-
46	LAKSHMANAN	39	M	1	B	N	N	2.5	N	N	N	N	N	N	N	90	NSR	N	13	130/80	3.7	10.3	100.4	96	-
					A	N	N	2	N	N	N	N	N	N	N	85	NSR	N	10.2	110/70	3.6	10	99.8	98	-
47	SERMAKANI	69	F	9	B	N	(+)90	2	N	N	(+)90	N	N	N	N	85	NSR	RIGHT	10.6	100/60	3.9	9.9	100.3	94	-
					A	N	(+)90	1.5	N	N	(+)90	N	N	N	N	75	NSR	RIGHT	10.5	115/60	4	9.8	98	98	-
48	KAVIARASAN	31	M	4	B	N	N	1	N	N	N	N	N	N	N	111	SINUS ARRYTHMIA	N	10.2	125/70	4.3	9.7	100.4	93	-
					A	N	N	2	N	N	N	N	N	N	N	85	NSR	N	11.6	100/85	3.6	9.7	98.6	96	-
49	RAJENDIRAN	55	M	1	B	N	N	1	N	N	N	N	N	N	N	80	NSR	N	11	110/75	3.8	9.6	100.4	97	-
					A	N	N	2.5	N	N	N	N	N	N	N	75	NSR	N	10.1	120/75	4	9.5	98.2	99	-
50	ANNAMMAL	62	F	4	B	N	N	2	N	N	N	N	N	N	N	117	NSR	N	10.3	110/80	4.2	10.5	101	96	-
					A	N	N	1.5	N	N	N	N	N	N	N	107	NSR	N	11.8	110/70	3.8	10.3	98.7	96	-
51	LIYO	36	F	1	B	N	N	2	N	N	N	N	N	N	N	90	NSR	N	12	100/60	3.9	9.8	99	96	-
					A	N	N	2.5	N	N	N	N	N	N	N	80	NSR	N	10.1	110/70	3.7	9.7	98.1	98	-
52	SANMUGAM	45	M	5	B	N	N	1	N	N	N	N	N	N	N	107	NSR	N	10.3	110/60	3.6	9.6	100.5	93	-
					A	N	N	2	N	N	N	N	N	N	N	88	NSR	N	11.9	120/70	4	9.5	99	97	-

53	PALANISAMY	54	M	2	B	N	N	2.5	N	N	N	N	N	N	INVERTED	85	NSR	N	12.7	110/70	4.2	9.4	100	97	-
					A	N	N	2.5	N	N	N	N	N	N	N	80	NSR	N	14	100/80	4.3	9.3	98.3	98	-
54	RESHMA	22	F	1	B	N	N	2	N	N	N	N	N	N	N	90	NSR	N	13.6	110/80	4.1	10	100.4	97	-
					A	N	N	2	N	N	N	N	N	N	N	75	NSR	N	10.4	130/80	4.2	10.3	98.3	98	-
55	SANJAYKUMAR	25	M	3	B	N	N	2	N	N	N	N	N	N	N	120	SINUS ARRYTHMIA	N	10.7	90/60	4.1	9.8	101	94	-
					A	N	N	2.5	N	N	N	N	N	N	N	90	SINUS ARRYTHMIA	N	13	120/80	3.8	9.5	99	97	-
56	SUBASH	24	M	1	B	N	N	2	N	N	N	N	N	N	N	85	NSR	N	12.7	130/80	3.9	9.8	100.1	97	-
					A	N	N	2	N	N	N	N	N	N	N	80	NSR	N	11	110/70	4	10	98.2	98	-
57	JUDITH PRIYA	27	F	2	B	N	N	2.5	N	N	N	N	N	N	INVERTED	90	NSR	N	11.7	100/60	4.1	9.5	99	97	-
					A	N	N	1	N	N	N	N	N	N	N	86	NSR	N	15	115/60	3.7	9.6	98.1	98	-
58	SEKAR	49	M	1	B	N	N	2	N	N	N	N	N	N	N	88	NSR	N	14.3	125/70	3.6	9.3	99.7	96	-
					A	N	N	1	N	N	N	N	N	N	N	75	NSR	N	10	100/65	3.7	9.2	98.6	98	-
59	SAIYMA	35	F	6	B	N	(+)90	3	N	N	(+)90	N	N	N	N	117	NSR	RIGHT	10.4	110/75	3.8	9.5	100.6	95	-
					A	N	(+)90	3	N	N	N	N	N	N	N	88	NSR	N	10.2	120/75	4	9.8	98.6	97	-
60	MEENAKSHI	21	F	3	B	N	N	1.5	N	N	N	N	N	N	N	107	NSR	N	10.4	110/80	4.1	9.6	100.3	94	-
					A	N	N	2	N	N	N	N	N	N	N	89	NSR	N	10	110/70	3.9	9.5	98.4	97	-
61	SIVASAMY	50	M	2	B	N	N	2	N	N	N	N	N	N	N	120	NSR	N	10.6	100/60	3.7	9.3	100.6	95	-
					A	N	N	1	N	N	N	N	N	N	N	110	NSR	N	13	110/70	4	9.2	99	96	-
62	RANGASAMY	65	M	5	B	N	N	3.5	N	N	N	LOW	N	N	N	75	NSR	N	13.7	110/60	4.3	9.4	100	98	-
					A	N	N	3.5	N	N	N	LOW	N	N	N	70	NSR	N	14	120/70	3.8	9	98.2	98	-
63	NATARAJ	60	M	4	B	N	N	2.5	N	N	(+)90	N	N	N	N	80	NSR	RIGHT	13.7	110/70	3.9	9.4	100.2	95	-
					A	N	N	2	N	N	N	N	N	N	N	70	NSR	N	10.2	100/80	4	9.8	98.4	98	-
64	PALANISAMY	62	M	5	B	N	N	2	N	N	N	N	N	N	N	116	NSR	N	10.7	110/80	4.4	9.7	100.5	95	-
					A	N	N	2	N	N	N	N	N	N	N	89	NSR	N	11	130/80	3.7	9.3	98.6	97	-
65	RAVI	52	M	1	B	N	N	2.5	N	N	N	N	N	N	N	115	NSR	N	10.5	90/60	3.9	10.6	101	93	-
					A	N	N	2	N	N	N	N	N	N	N	106	NSR	N	10.8	120/80	3.7	10.4	98.1	97	-
66	THASLIMA	22	F	4	B	N	N	2	N	N	N	N	N	N	N	90	NSR	N	11.4	130/80	4.6	10.2	99	96	-
					A	N	N	2.5	N	N	N	N	N	N	N	85	NSR	N	10.2	110/70	4.2	10.4	98	98	-
67	JASMIN	21	F	1	B	N	N	1	N	N	N	N	N	N	N	110	SINUS ARRYTHMIA	N	10	100/60	4.4	9.7	100.7	95	-
					A	N	N	2	N	N	N	N	N	N	N	84	NSR	N	11.7	115/60	4.5	9.4	98.6	98	-
68	RAJAMANIKAM	48	M	1	B	N	N	1	N	N	N	N	N	N	N	90	NSR	N	12	125/70	4.2	9.5	99.7	96	-
					A	N	N	2.5	N	N	N	N	N	N	N	75	NSR	N	13	100/65	4	9.6	98.4	98	-
69	PRIYADARSHINI	28	F	6	B	N	N	2	N	N	N	N	N	N	N	88	NSR	N	13.7	110/75	4.1	9.7	100	97	-
					A	N	N	1.5	N	N	N	N	N	N	N	75	NSR	N	10	120/75	3.7	9.8	98.1	98	-
70	KUMARASAMY	65	M	1	B	N	(+)90	3	N	N	N	LOW	N	N	N	111	NSR	N	10.3	110/80	3.8	8.9	100.7	94	-
					A	N	N	3	N	N	N	N	N	N	N	106	NSR	N	14	110/70	4.5	8.4	98.6	97	-
71	KOWSALYA	29	F	4	B	N	N	1	N	N	N	N	N	N	N	80	NSR	N	13.8	100/60	4.1	9	99.9	96	-
					A	N	N	2	N	N	N	N	N	N	N	75	NSR	N	10.2	110/70	3.6	9.3	97.9	98	-
72	PALANISAMY	64	M	3	B	N	N	2.5	N	N	N	N	N	N	N	112	NSR	N	10.7	110/60	3.8	9.5	101	95	-
					A	N	N	2.5	N	N	N	N	N	N	N	80	NSR	N	10	120/70	3.9	9.6	99	97	-
73	RANI	33	F	1	B	N	N	2	N	N	N	N	N	N	N	112	NSR	N	10.6	110/70	4	10	100.7	95	-
					A	N	N	2	N	N	N	N	N	N	N	108	NSR	N	14.7	100/80	4	10.1	98.8	97	-
74	SENTHILKUMAR	40	M	4	B	N	N	2	N	N	N	N	N	N	N	90	NSR	N	14.2	110/80	4.3	10.5	101.1	97	-
					A	N	N	2.5	N	N	N	N	N	N	N	88	NSR	N	10.7	130/80	3.8	9.9	99	97	-
75	KAALEESHWARI	29	F	1	B	N	N	2	N	N	N	N	N	N	N	120	NSR	N	10.4	90/60	3.9	9.8	100.4	92	-
					A	N	N	2	N	N	N	N	N	N	N	90	NSR	N	11.3	120/80	4.2	9.7	98.6	96	-
76	VIJAYAGANESH	42	M	2	B	N	N	2.5	N	N	N	N	N	N	N	85	(APC)	N	12	130/80	4.1	9.6	100	96	-
					A	N	N	1	N	N	N	N	N	N	N	80	(APC)	N	12.4	110/70	4	9.5	98.4	98	-
77	MARISELVAM	39	M	1	B	N	N	2	PROLONGED	N	N	N	N	N	N	116	NSR	N	11.6	100/60	3.8	9.3	101.2	93	-
					A	N	N	1	PROLONGED	N	N	N	N	N	N	110	NSR	N	11.8	115/60	3.9	9.2	98.8	97	-
78	MAHESHWARAN	45	M	4	B	N	N	2.5	N	N	N	N	N	N	N	85	NSR	N	11	125/70	3.8	8.9	99	98	-
					A	N	N	2	N	N	N	N	N	N	N	75	NSR	N	11.1	100/65	3.7	8.8	98.1	99	-
79	AJITHKUMAR	22	M	2	B	N	N	1.5	N	N	N	LOW	N	N	N	119	NSR	N	10	110/75	3.8	8.9	101	94	-
					A	N	N	2	N	N	N	N	N	N	N	110	NSR	N	10	120/75	4	9.2	98.7	97	-
80	KHALIF	34	M	1	B	N	N	2	N	N	N	N	N	N	N	116	NSR	N	9.9	110/80	4.3	10	100.7	95	-
					A	N	N	1	N	N	N	N	N	N	N	80	NSR	N	13.4	110/70	4.5	10.3	98.8	97	-

81	SELVARAJ	69	M	1 1/2	B	N	N	2	N	N	N	N	N	N	N	90	NSR	N	12.8	100/60	4.4	9.9	100.4	98	-
					A	N	N	2.5	N	N	N	N	N	N	N	85	NSR	N	11.9	110/70	3.7	9.7	98.6	99	-
82	SIVAKUMAR	40	M	1	B	N	N	2.5	N	N	N	N	N	N	N	56	NSR	N	11.4	110/60	3.8	9.6	100	97	-
					A	N	N	2	N	N	N	N	N	N	N	67	NSR	N	13.2	120/70	3.7	9.5	98.4	97	-
83	PAPATHI	65	F	1	B	N	N	2	N	N	N	N	N	N	N	88	NSR	N	12.8	110/70	3.8	9.3	100.3	97	-
					A	N	N	2	N	N	N	N	N	N	N	76	NSR	N	10.3	100/80	3.9	9.2	98.6	98	-
84	PERUMAL	50	M	2	B	N	N	2.5	N	N	N	LOW	N	N	N	116	NSR	N	10.6	110/80	4	9	101.1	93	-
					A	N	N	2	N	N	N	N	N	N	N	110	NSR	N	11.9	130/80	4.2	9.2	99	97	-
85	ARUNRAJ KUMAR	31	M	6	B	N	N	2	N	N	N	LOW	N	N	N	95	NSR	N	12.4	90/60	4	9.8	99.8	96	-
					A	N	N	2.5	N	N	N	N	N	N	N	85	NSR	N	10.4	120/80	3.9	9.7	98.3	97	-
86	GANESHAN	47	M	3	B	N	(+)90	1	N	N	(+)90	N	N	N	N	109	(APC)	RIGHT	10.6	130/80	4.5	9.6	100.5	94	-
					A	N	N	2	N	N	N	N	N	N	N	86	(APC)	N	11	110/70	4.2	9.3	98.6	97	-
87	KANNAPPAN	35	M	1	B	N	N	1	N	N	N	N	N	N	N	109	NSR	N	10.7	100/60	4.3	9	100.6	94	-
					A	N	N	2.5	N	N	N	N	N	N	N	81	NSR	N	14	115/60	4	8.8	98.7	96	-
88	SENTHILKUMAR	45	M	2	B	N	N	2	N	N	N	N	N	N	N	85	NSR	N	13.1	125/70	4.2	9.5	100.1	97	-
					A	N	N	1.5	N	N	N	N	N	N	N	75	NSR	N	12.1	100/65	3.6	9.8	98.6	97	-
89	SELVARAJ	35	M	3	B	N	N	2	N	N	N	LOW	N	N	N	90	NSR	N	11.4	110/75	4.2	9	99.9	96	-
					A	N	N	2	N	N	N	N	N	N	N	80	NSR	N	10.4	120/75	3.8	9.3	98.2	98	-
90	GOPAL	40	M	1	B	N	N	1	N	N	N	N	N	N	N	109	NSR	N	9.8	110/80	4.3	10.3	100.8	94	-
					A	N	N	2	N	N	N	N	N	N	N	82	NSR	N	12.6	110/70	4.5	10.4	98.9	97	-
91	KAVIN	27	M	2	B	N	N	2.5	N	N	N	N	N	N	N	90	NSR	N	11.9	100/60	3.9	9.2	100	97	-
					A	N	N	2.5	N	N	N	N	N	N	N	85	NSR	N	14.1	110/70	3.8	9.8	98.1	97	-
92	MOHAMED ALI	29	M	6	B	N	N	2	N	N	N	N	N	N	N	85	NSR	N	13.6	110/80	4.1	8.9	100.6	97	-
					A	N	N	2	N	N	N	N	N	N	N	70	NSR	N	10.7	100/70	4.2	9.4	98.5	98	-
93	MUTHURATHINAM	43	M	8	B	N	(+)90	3.5	N	N	(+)90	N	N	N	N	120	NSR	RIGHT	12.3	110/70	3.8	8.6	100.7	95	-
					A	N	(+)90	3.5	N	N	(+)90	N	N	N	N	110	NSR	RIGHT	11.5	100/80	4.3	10.2	99	98	-
94	VELLINGIRI	55	M	1	B	N	N	2	N	N	N	N	N	N	N	112	SINUS ARRYTHMIA	N	10.3	110/80	4.6	10.1	101	94	-
					A	N	N	2	N	N	N	N	N	N	N	89	SINUS ARRYTHMIA	N	10	130/80	4.7	10	98.6	97	-
95	GOKUL	28	M	2	B	N	N	2.5	N	N	N	N	N	N	N	75	NSR	N	13.4	90/60	3.8	9	99.8	97	-
					A	N	N	1	N	N	N	N	N	N	N	65	NSR	N	12.6	120/80	3.5	8.8	98.3	97	-
96	DHEENADAYALAN	50	M	4	B	N	N	2	N	N	N	N	N	N	N	85	NSR	N	10.4	130/80	4.1	8.9	100.6	97	-
					A	N	N	1	N	N	N	N	N	N	N	78	NSR	N	10.2	110/70	3.9	9.1	98.7	98	-
97	KATHIRVEL	37	M	1	B	N	N	2.5	N	N	N	N	N	N	N	117	NSR	N	10.7	100/60	4.7	9.6	100	95	-
					A	N	N	2	N	N	N	N	N	N	N	86	NSR	N	12.6	115/80	4.4	9.8	98.7	98	-
98	PREMKUMAR	26	M	4	B	N	N	1.5	N	N	N	N	N	N	N	90	NSR	N	12	125/70	3.8	10.4	100.2	97	-
					A	N	N	2	N	N	N	N	N	N	N	80	NSR	N	13	100/65	4.3	10.5	98.6	97	-
99	RAMESH	47	M	1	B	N	N	2	N	N	N	N	N	N	N	115	NSR	N	10.5	110/75	4.2	9.8	100.4	94	-
					A	N	N	1	N	N	N	N	N	N	N	80	NSR	N	9.9	120/75	3.8	9.9	99	97	-
100	KANTHASAMY	58	M	4	B	N	N	2	N	N	N	N	N	N	N	85	NSR	N	11.5	110/80	3.5	8.9	99.9	96	-
					A	N	N	2.5	N	N	N	N	N	N	N	75	NSR	N	10.8	110/70	3.4	9.3	98.3	97	-

S.NO	NAME	AGE	SEX	DURATION OF SYMPTOM	ATT	P WAVE DURATION	P WAVE AXIS	P WAVE AMPLITUDE	P-R INTERVAL	QRS DURATION	QRS AXIS	QRS AMPLITUDE	QT INTERVAL	ST SEGMENT	T WAVE	HEART RATE	RHYTHM	AXIS	HAEMOGLOBIN	BLOOD PRESSURE	SR.POTASSIUM	SR.CALCIUM	TEMPERATURE	SPO2	CONDUCTION BLOCK
					B	N	N	2	N	N	N	N	N	N	N	115	NSR	N	10.4	110/70	4	9	101.7	95	-
1	DURAIRAJ	35	M	4	A	N	N	1	N	N	N	N	N	N	N	90	NSR	N	10.2	100/60	3.7	8.9	98.6	97	-
					B	N	N	2.5	N	N	N	N	N	N	INVERTED	120	NSR	N	10	110/70	3.6	10	101	94	-
2	ANANDAN	46	M	5	A	N	N	2	N	N	N	N	N	N	INVERTED	108	NSR	N	10.1	110/60	4.1	9.7	99	96	-
					B	N	(+)90	3	N	N	(+)90	LOW	N	N	N	110	NSR	RIGHT	10.2	120/70	3.8	9.8	100.6	93	-
3	DINAKARAN	28	M	9	A	N	(+)90	3	N	N	N	N	N	N	N	87	NSR	N	9.7	110/70	3.7	9.5	99.2	94	-
					B	N	(+)90	2	N	N	(+)90	N	N	N	N	90	NSR	RIGHT	11.6	100/80	3.9	10.2	99.7	93	-
4	KALAVATHI	45	F	6	A	N	N	1	N	N	N	N	N	N	N	75	NSR	N	11.5	110/80	4	9.8	98.6	97	-
					B	N	N	2	N	N	N	N	N	N	N	85	NSR	N	12	130/80	3.8	9.9	99.9	96	-
5	VEERATHAL	70	F	1	A	N	N	2.5	N	N	N	N	N	N	N	80	NSR	N	12.2	90/60	3.6	10.2	98.5	98	-
					B	N	N	2.5	N	N	N	N	N	N	N	90	NSR	N	13	120/80	3.6	9.7	99.8	97	-
6	SOMASUNTHARAM	55	M	1	A	N	N	2	N	N	N	N	N	N	N	78	NSR	N	12.6	130/80	3.7	9.8	98.4	98	-
					B	N	N	2.5	N	N	N	N	N	N	N	87	NSR	RIGHT	11.7	110/70	4	9.5	100.2	94	-
7	MANIKAM	52	M	4	A	N	N	2.5	N	N	N	N	N	N	N	89	NSR	N	10.5	100/60	4.2	9.6	98.7	96	-
					B	N	N	2.5	N	N	N	N	N	N	N	88	NSR	N	10.4	115/60	3.7	9.9	100.1	97	-
8	MAHALI	52	M	2	A	N	N	2	N	N	N	N	N	N	N	80	NSR	N	10.6	125/70	4	9.3	98.6	97	-
					B	N	N	2	N	N	N	LOW	N	N	N	80	NSR	N	11	100/65	3.6	8.8	99.8	96	-
9	THIRUMOORTHI	49	M	8	A	N	N	2	N	N	N	LOW	N	N	N	75	NSR	N	10.8	110/75	4	8.9	98.5	98	-
					B	N	N	1	N	N	N	N	N	N	N	85	NSR	N	10.9	120/75	4	8.5	100	97	-
10	VENKATESH	52	M	4	A	N	N	2	N	N	N	N	N	N	N	70	NSR	N	10.8	110/80	4.2	8.9	98.1	98	-
					B	N	N	1	N	N	N	N	N	N	N	86	NSR	N	11.5	110/70	3.6	10.1	99.5	97	-
11	SAKUNTHALA	60	F	1	A	N	N	2.5	N	N	N	N	N	N	N	78	NSR	N	10.9	100/60	3.7	9.8	98.5	97	-
					B	N	N	2	N	N	N	N	N	N	N	80	NSR	N	13	110/70	3.8	10.5	99.9	96	-
12	TAMILARASAN	21	M	1	A	N	N	1.5	N	N	N	N	N	N	N	75	NSR	N	13	110/60	4.1	9.7	98.4	98	-
					B	N	N	2	N	N	N	N	N	N	N	120	NSR	N	10	120/70	4	9.8	100.4	93	-
13	HASAN	50	M	3	A	N	N	2	N	N	N	N	N	N	N	88	NSR	N	10.2	110/70	4.2	9.5	99	97	-
					B	N	N	1	PROLONGED	N	N	N	N	N	N	90	NSR	N	11.7	100/80	3.6	8.9	100.1	96	-
14	NATARAJ	56	M	4	A	N	N	2	PROLONGED	N	N	N	N	N	N	80	NSR	N	11.4	110/80	4	8.8	98.4	98	-
					B	N	N	2.5	N	N	N	N	N	N	INVERTED	115	NSR	N	13	130/80	4	9.8	100.4	94	-
15	SHANMUGAM	63	M	4	A	N	N	2.5	N	N	N	N	N	N	N	89	NSR	N	13.3	90/60	4	9.7	98.8	97	-
					B	N	N	3	N	N	N	LOW	N	N	N	116	NSR	N	10.7	120/80	4.1	9.9	100.6	95	-
16	ARUMUGAM	57	M	5	A	N	N	3	N	N	N	LOW	N	N	N	110	NSR	N	10.5	130/80	4.3	9.8	99	95	-
					B	N	N	2	N	N	N	N	N	N	N	92	(PVC)	N	12	110/70	4.1	10.5	100.2	97	-
17	BABU	53	M	1	A	N	N	2.5	N	N	N	N	N	N	N	80	(PVC)	N	12.7	100/60	3.6	10	98.5	97	-
					B	N	N	2	N	N	N	LOW	N	N	N	85	NSR	N	11.7	115/60	3.8	9.6	99	95	RBBB
18	SAMPATH	50	M	6	A	N	N	2	N	N	N	LOW	N	N	N	78	NSR	N	12	125/70	4	9.5	98.4	97	RBBB
					B	N	N	2.5	N	N	N	N	N	N	N	110	NSR	N	10.1	100/65	4.1	8.6	100.4	95	-
19	CHINNASAMY	75	M	4	A	N	N	1	N	N	N	N	N	N	N	81	NSR	N	10	110/75	3.8	8.7	98.7	97	-
					B	N	N	2	N	N	N	N	N	N	N	117	SINUS ARRYTHMIA	N	10	120/75	3.4	8.8	101	93	-
20	SIVAKUMAR	32	M	2	A	N	N	1	N	N	N	N	N	N	N	88	NSR	N	10.2	110/80	3.6	8.9	99.2	96	-
					B	N	(+)90	2.5	N	N	(+)90	N	N	N	N	90	NSR	RIGHT	11.2	110/70	3.9	9.7	100.1	97	RBBB
21	SENTHILKUMAR	45	M	9	A	N	(+)90	2	N	N	(+)90	N	N	N	N	80	NSR	RIGHT	11.4	100/60	4.1	9.5	98.6	97	RBBB
					B	N	N	1.5	N	N	N	LOW	N	N	N	88	NSR	N	12.4	110/70	4	9	100	96	-
22	LAKSHMANAN	35	M	5	A	N	N	2	N	N	N	LOW	N	N	N	75	NSR	N	12	110/60	4.2	9.2	98.8	98	-
					B	N	N	3	N	N	N	N	N	N	N	110	NSR	N	10.3	120/70	3.9	9.3	101.1	95	-
23	SELVANATHAN	55	M	1	A	N	N	3	N	N	N	N	N	N	N	86	NSR	N	10	110/70	4	9.5	99	97	-
					B	N	N	2	N	N	(+)90	LOW	N	N	N	106	NSR	RIGHT	10.4	100/80	4.2	9.7	100.1	94	-
24	PALANIYAMMAL	40	F	5	A	N	N	2	N	N	N	N	N	N	N	84	NSR	N	10	110/80	4.4	9.5	98.2	97	-

					B	N	N	2.5	N	N	N	N	N	N	N	80	NSR	N	13.2	130/80	3.6	10	99	96	-
25	SANGILIYAMMAL	75	F	2	A	N	N	2	N	N	N	N	N	N	N	75	NSR	N	10.5	90/60	3.9	9.3	98.4	98	-
					B	N	N	3	N	N	N	N	N	N	INVERTED	117	NSR	N	10.4	120/80	4	9.8	100.3	93	-
26	KRISHNAN	65	M	2	A	N	N	3	N	N	N	N	N	N	N	86	NSR	N	11.5	130/80	4.3	9.6	98.6	96	-
					B	N	N	2.5	N	N	N	N	N	N	N	86	NSR	N	11.9	110/70	3.6	9.5	100.1	96	-
27	RAJENDRAN	51	M	1	A	N	N	2	N	N	N	N	N	N	N	75	NSR	N	12	100/60	3.9	9.3	99	98	-
					B	N	(+)90	3.5	N	N	(+)90	N	N	N	N	109	NSR	RIGHT	11.1	115/60	4	9.2	100.4	93	RBBB
28	SRINIVASAN	36	M	7	A	N	(+)90	3.5	N	N	(+)90	N	N	N	N	86	NSR	RIGHT	10	125/70	4.1	9.7	98.2	97	RBBB
					B	N	N	1	N	N	N	N	N	N	N	90	(APC)	N	10.4	100/65	4.2	9.9	100.4	96	-
29	KANNAN	44	M	1	A	N	N	2	N	N	N	N	N	N	N	75	NSR	N	10.3	110/75	4.5	9.1	98.5	98	-
					B	N	90	1	N	N	N	LOW	N	N	N	112	NSR	N	10.5	120/75	4.2	10.2	100.3	96	-
30	RAMACHANDRAN	34	M	5	A	N	N	2.5	N	N	N	LOW	N	N	N	87	NSR	N	13.4	110/80	4.2	9.5	99	96	-
					B	N	N	2	N	N	N	N	N	N	N	88	NSR	N	14	110/70	3.8	8.7	100.2	96	-
31	RANJITH	25	M	2	A	N	N	1.5	N	N	N	N	N	N	N	79	NSR	N	14.5	100/60	3.7	8.9	98.5	98	-
					B	N	N	2	N	N	N	N	N	N	N	118	NSR	N	14.1	110/70	4	9.1	100.6	95	-
32	LAKSHMI	40	F	1	A	N	N	2	N	N	N	N	N	N	N	88	NSR	N	12	110/60	4.2	9.8	98.4	95	-
					B	N	N	1	N	N	N	N	N	N	N	90	NSR	N	12.6	120/70	3.9	10.4	99	96	-
33	KARTHIK	35	M	2	A	N	N	2	N	N	N	N	N	N	N	78	NSR	N	10.3	110/70	4	10.6	98.2	98	-
					B	N	(+)90	2.5	N	N	N	N	N	N	N	116	NSR	N	10.2	100/80	4.2	9	100	94	-
34	KALAVATHI	35	F	5	A	N	(+)90	2.5	N	N	N	N	N	N	N	108	NSR	N	10	110/80	3.6	9.3	98.6	96	-
					B	N	N	2	N	N	N	N	N	N	N	110	NSR	N	10.2	130/80	3.8	9.7	101	95	-
35	KARTHIKA	21	F	4	A	N	N	2	N	N	N	N	N	N	N	83	NSR	N	14	90/60	3.7	9.4	99	97	-
					B	N	N	2	N	N	N	N	N	N	N	85	NSR	N	14.2	120/80	4	9.8	99.8	96	-
36	BALAN	29	M	2	A	N	N	2.5	N	N	N	N	N	N	N	75	NSR	N	9.7	130/80	4.2	9.5	97.9	98	-
					B	N	(+)90	2	N	N	N	N	N	N	N	112	NSR	N	10.2	110/70	3.8	9	100.6	94	-
37	CHINNASAMY	47	M	6	A	N	N	2	N	N	N	N	N	N	N	88	NSR	N	12	100/60	3.7	9.3	98.7	96	-
					B	N	N	2.5	N	N	N	N	N	N	N	86	NSR	N	11.2	115/60	3.9	9.6	100	97	-
38	CHANDRASEKAR	53	M	1	A	N	N	1	N	N	N	N	N	N	N	78	NSR	N	10.1	125/70	4	9.8	98.6	98	-
					B	N	N	2	N	N	(+)90	LOW	N	N	N	121	NSR	RIGHT	10	100/65	4.2	9.9	100.6	94	RBBB
39	AJMAL	36	M	8	A	N	N	1	N	N	(+)90	LOW	N	N	N	109	NSR	RIGHT	13	110/75	4.4	9.7	99.1	98	RBBB
					B	N	N	2.5	N	N	N	N	N	N	N	90	SINUS ARRYTHMIA	N	13.4	120/75	3.9	8.9	99.8	97	-
40	JEYASELAN	38	M	2	A	N	N	2	N	N	N	N	N	N	N	85	SINUS ARRYTHMIA	N	12.4	110/70	4.1	8.8	98.1	97	-
					B	N	N	1.5	N	N	N	N	N	N	N	53	NSR	N	12.7	100/60	4.3	8.3	99	96	-
41	PALANISAMY	48	M	1	A	N	N	2	N	N	N	N	N	N	N	55	NSR	N	10	110/70	3.8	9.4	98.6	96	-
					B	N	N	2	N	N	N	N	N	N	N	111	NSR	N	10.4	110/60	3.6	9.3	100.6	92	-
42	RAJENDRAN	45	M	5	A	N	N	1	N	N	N	N	N	N	N	84	NSR	N	13	120/70	3.9	9.7	99.1	95	-
					B	N	N	2	N	N	N	LOW	N	N	N	88	NSR	N	10.6	110/70	4.1	9.2	100	97	-
43	ARUMUGAM	60	M	1	A	N	N	2.5	N	N	N	N	N	N	N	75	NSR	N	12.6	100/80	4.3	9.8	98.1	99	-
					B	N	N	2.5	N	N	N	N	N	N	N	90	NSR	N	13	110/80	4	9.9	100.4	97	-
44	NAVINKUMAR	24	M	1	A	N	N	2	N	N	N	N	N	N	N	80	NSR	N	14	130/80	4.1	9.3	98.7	97	-
					B	N	N	3	PROLONGED	N	N	N	N	N	N	85	NSR	N	13.1	90/60	4	8.9	99.7	97	-
45	STALIN	29	M	2	A	N	N	3	PROLONGED	N	N	N	N	N	N	78	NSR	N	13.8	120/80	3.8	8.8	98.4	98	-
					B	N	N	2.5	N	N	N	N	N	N	N	90	NSR	N	13	130/80	3.7	10.3	100.4	96	-
46	LAKSHMANAN	39	M	1	A	N	N	2	N	N	N	N	N	N	N	85	NSR	N	10.2	110/70	3.6	10	99.8	98	-
					B	N	(+)90	2	N	N	(+)90	N	N	N	N	85	NSR	RIGHT	10.6	100/60	3.9	9.9	100.3	94	-
47	SERMAKANI	69	F	9	A	N	(+)90	1.5	N	N	(+)90	N	N	N	N	75	NSR	RIGHT	10.5	115/60	4	9.8	98	98	-
					B	N	N	1	N	N	N	N	N	N	N	111	SINUS ARRYTHMIA	N	10.2	125/70	4.3	9.7	100.4	93	-
48	KAVIARASAN	31	M	4	A	N	N	2	N	N	N	N	N	N	N	85	NSR	N	11.6	100/65	3.6	9.7	98.6	96	-
					B	N	N	1	N	N	N	N	N	N	N	80	NSR	N	11	110/75	3.8	9.6	100.4	97	-
49	RAJENDIRAN	55	M	1	A	N	N	2.5	N	N	N	N	N	N	N	75	NSR	N	10.1	120/75	4	9.5	98.2	99	-
					B	N	N	2	N	N	N	N	N	N	N	117	NSR	N	10.3	110/80	4.2	10.5	101	96	-
50	ANNAMMAL	62	F	4	A	N	N	1.5	N	N	N	N	N	N	N	107	NSR	N	11.8	110/70	3.8	10.3	98.7	96	-
					B	N	N	2	N	N	N	N	N	N	N	90	NSR	N	12	100/60	3.9	9.8	99	96	-
51	LIYO	36	F	1	A	N	N	2.5	N	N	N	N	N	N	N	80	NSR	N	10.1	110/70	3.7	9.7	98.1	98	-
					B	N	N	1	N	N	N	N	N	N	N	107	NSR	N	10.3	110/60	3.6	9.6	100.5	93	-
52	SANMUGAM	45	M	5	A	N	N	2	N	N	N	N	N	N	N	88	NSR	N	11.9	120/70	4	9.5	99	97	-

					B	N	N	2.5	N	N	N	N	N	N	INVERTED	85	NSR	N	12.7	110/70	4.2	9.4	100	97	-
53	PALANISAMY	54	M	2	A	N	N	2.5	N	N	N	N	N	N	N	80	NSR	N	14	100/80	4.3	9.3	98.3	98	-
					B	N	N	2	N	N	N	N	N	N	N	90	NSR	N	13.6	110/80	4.1	10	100.4	97	-
54	RESHMA	22	F	1	A	N	N	2	N	N	N	N	N	N	N	75	NSR	N	10.4	130/80	4.2	10.3	98.3	98	-
					B	N	N	2	N	N	N	N	N	N	N	120	SINUS ARRYTHMIA	N	10.7	90/60	4.1	9.8	101	94	-
55	SANJAYKUMAR	25	M	3	A	N	N	2.5	N	N	N	N	N	N	N	90	SINUS ARRYTHMIA	N	13	120/80	3.8	9.5	99	97	-
					B	N	N	2	N	N	N	N	N	N	N	85	NSR	N	12.7	130/80	3.9	9.8	100.1	97	-
56	SUBASH	24	M	1	A	N	N	2	N	N	N	N	N	N	N	80	NSR	N	11	110/70	4	10	98.2	98	-
					B	N	N	2.5	N	N	N	N	N	N	INVERTED	90	NSR	N	11.7	100/60	4.1	9.5	99	97	-
57	JUDITH PRIYA	27	F	2	A	N	N	1	N	N	N	N	N	N	N	86	NSR	N	15	115/60	3.7	9.6	98.1	98	-
					B	N	N	2	N	N	N	N	N	N	N	88	NSR	N	14.3	125/70	3.6	9.3	99.7	96	-
58	SEKAR	49	M	1	A	N	N	1	N	N	N	N	N	N	N	75	NSR	N	10	100/65	3.7	9.2	98.6	98	-
					B	N	(+)90	3	N	N	(+)90	N	N	N	N	117	NSR	RIGHT	10.4	110/75	3.8	9.5	100.6	95	-
59	SAIYMA	35	F	6	A	N	(+)90	3	N	N	N	N	N	N	N	88	NSR	N	10.2	120/75	4	9.8	98.6	97	-
					B	N	N	1.5	N	N	N	N	N	N	N	107	NSR	N	10.4	110/80	4.1	9.6	100.3	94	-
60	MEENAKSHI	21	F	3	A	N	N	2	N	N	N	N	N	N	N	89	NSR	N	10	110/70	3.9	9.5	98.4	97	-
					B	N	N	2	N	N	N	N	N	N	N	120	NSR	N	10.6	100/60	3.7	9.3	100.6	95	-
61	SIVASAMY	50	M	2	A	N	N	1	N	N	N	N	N	N	N	110	NSR	N	13	110/70	4	9.2	99	96	-
					B	N	N	3.5	N	N	N	LOW	N	N	N	75	NSR	N	13.7	110/60	4.3	9.4	100	98	-
62	RANGASAMY	65	M	5	A	N	N	3.5	N	N	N	LOW	N	N	N	70	NSR	N	14	120/70	3.8	9	98.2	98	-
					B	N	N	2.5	N	N	(+)90	N	N	N	N	80	NSR	RIGHT	13.7	110/70	3.9	9.4	100.2	95	-
63	NATARAJ	60	M	4	A	N	N	2	N	N	N	N	N	N	N	70	NSR	N	10.2	100/80	4	9.8	98.4	98	-
					B	N	N	2	N	N	N	N	N	N	N	116	NSR	N	10.7	110/80	4.4	9.7	100.5	95	-
64	PALANISAMY	62	M	5	A	N	N	2	N	N	N	N	N	N	N	89	NSR	N	11	130/80	3.7	9.3	98.6	97	-
					B	N	N	2.5	N	N	N	N	N	N	N	115	NSR	N	10.5	90/60	3.9	10.6	101	93	-
65	RAVI	52	M	1	A	N	N	2	N	N	N	N	N	N	N	106	NSR	N	10.8	120/80	3.7	10.4	98.1	97	-
					B	N	N	2	N	N	N	N	N	N	N	90	NSR	N	11.4	130/80	4.6	10.2	99	96	-
66	THASLIMA	22	F	4	A	N	N	2.5	N	N	N	N	N	N	N	85	NSR	N	10.2	110/70	4.2	10.4	98	98	-
					B	N	N	1	N	N	N	N	N	N	N	110	SINUS ARRYTHMIA	N	10	100/60	4.4	9.7	100.7	95	-
67	JASMIN	21	F	1	A	N	N	2	N	N	N	N	N	N	N	84	NSR	N	11.7	115/60	4.5	9.4	98.6	98	-
					B	N	N	1	N	N	N	N	N	N	N	90	NSR	N	12	125/70	4.2	9.5	99.7	96	-
68	RAJAMANIKAM	48	M	1	A	N	N	2.5	N	N	N	N	N	N	N	75	NSR	N	13	100/65	4	9.6	98.4	98	-
					B	N	N	2	N	N	N	N	N	N	N	88	NSR	N	13.7	110/75	4.1	9.7	100	97	-
69	PRIYADARSHINI	28	F	6	A	N	N	1.5	N	N	N	N	N	N	N	75	NSR	N	10	120/75	3.7	9.8	98.1	98	-
					B	N	(+)90	3	N	N	N	LOW	N	N	N	111	NSR	N	10.3	110/80	3.8	8.9	100.7	94	-
70	KUMARASAMY	65	M	1	A	N	N	3	N	N	N	N	N	N	N	106	NSR	N	14	110/70	4.5	8.4	98.6	97	-
					B	N	N	1	N	N	N	N	N	N	N	80	NSR	N	13.8	100/60	4.1	9	99.9	96	-
71	KOWSALYA	29	F	4	A	N	N	2	N	N	N	N	N	N	N	75	NSR	N	10.2	110/70	3.6	9.3	97.9	98	-
					B	N	N	2.5	N	N	N	N	N	N	N	112	NSR	N	10.7	110/60	3.8	9.5	101	95	-
72	PALANISAMY	64	M	3	A	N	N	2.5	N	N	N	N	N	N	N	80	NSR	N	10	120/70	3.9	9.6	99	97	-
					B	N	N	2	N	N	N	N	N	N	N	112	NSR	N	10.6	110/70	4	10	100.7	95	-
73	RANI	33	F	1	A	N	N	2	N	N	N	N	N	N	N	108	NSR	N	14.7	100/80	4	10.1	98.8	97	-
					B	N	N	2	N	N	N	N	N	N	N	90	NSR	N	14.2	110/80	4.3	10.5	101.1	97	-
74	SENTHILKUMAR	40	M	4	A	N	N	2.5	N	N	N	N	N	N	N	88	NSR	N	10.7	130/80	3.8	9.9	99	97	-
					B	N	N	2	N	N	N	N	N	N	N	120	NSR	N	10.4	90/60	3.9	9.8	100.4	92	-
75	KAALEESHWARI	29	F	1	A	N	N	2	N	N	N	N	N	N	N	90	NSR	N	11.3	120/80	4.2	9.7	98.6	96	-
					B	N	N	2.5	N	N	N	N	N	N	N	85	(APC)	N	12	130/80	4.1	9.6	100	96	-
76	VIJAYAGANESH	42	M	2	A	N	N	1	N	N	N	N	N	N	N	80	(APC)	N	12.4	110/70	4	9.5	98.4	98	-
					B	N	N	2	PROLONGED	N	N	N	N	N	N	116	NSR	N	11.6	100/60	3.8	9.3	101.2	93	-
77	MARISELVAM	39	M	1	A	N	N	1	PROLONGED	N	N	N	N	N	N	110	NSR	N	11.8	115/60	3.9	9.2	98.8	97	-
					B	N	N	2.5	N	N	N	N	N	N	N	85	NSR	N	11	125/70	3.8	8.9	99	98	-
78	MAHESHWARAN	45	M	4	A	N	N	2	N	N	N	N	N	N	N	75	NSR	N	11.1	100/65	3.7	8.8	98.1	99	-
					B	N	N	1.5	N	N	N	LOW	N	N	N	119	NSR	N	10	110/75	3.8	8.9	101	94	-
79	AJITHKUMAR	22	M	2	A	N	N	2	N	N	N	N	N	N	N	110	NSR	N	10	120/75	4	9.2	98.7	97	-
					B	N	N	2	N	N	N	N	N	N	N	116	NSR	N	9.9	110/80	4.3	10	100.7	95	-
80	KHALIF	34	M	1	A	N	N	1	N	N	N	N	N	N	N	80	NSR	N	13.4	110/70	4.5	10.3	98.8	97	-

					B	N	N	2	N	N	N	N	N	N	N	90	NSR	N	12.8	100/60	4.4	9.9	100.4	98	-
81	SELVARAJ	69	M	1 1/2	A	N	N	2.5	N	N	N	N	N	N	N	85	NSR	N	11.9	110/70	3.7	9.7	98.6	99	-
					B	N	N	2.5	N	N	N	N	N	N	N	56	NSR	N	11.4	110/60	3.8	9.6	100	97	-
82	SIVAKUMAR	40	M	1	A	N	N	2	N	N	N	N	N	N	N	67	NSR	N	13.2	120/70	3.7	9.5	98.4	97	-
					B	N	N	2	N	N	N	N	N	N	N	88	NSR	N	12.8	110/70	3.8	9.3	100.3	97	-
83	PAPATHI	65	F	3	A	N	N	2	N	N	N	N	N	N	N	76	NSR	N	10.3	100/80	3.9	9.2	98.6	98	-
					B	N	N	2.5	N	N	N	LOW	N	N	N	116	NSR	N	10.6	110/80	4	9	101.1	93	-
84	PERUMAL	50	M	2	A	N	N	2	N	N	N	N	N	N	N	110	NSR	N	11.9	130/80	4.2	9.2	99	97	-
					B	N	N	2	N	N	N	LOW	N	N	N	95	NSR	N	12.4	90/60	4	9.8	99.8	96	-
85	ARUNRAJ KUMAR	31	M	6	A	N	N	2.5	N	N	N	N	N	N	N	85	NSR	N	10.4	120/80	3.9	9.7	98.3	97	-
					B	N	(+)90	1	N	N	(+)90	N	N	N	N	109	(APC)	RIGHT	10.6	130/80	4.5	9.6	100.5	94	-
86	GANESHAN	47	M	3	A	N	N	2	N	N	N	N	N	N	N	86	(APC)	N	11	110/70	4.2	9.3	98.6	97	-
					B	N	N	1	N	N	N	N	N	N	N	109	NSR	N	10.7	100/60	4.3	9	100.6	94	-
87	KANNAPPAN	35	M	1	A	N	N	2.5	N	N	N	N	N	N	N	81	NSR	N	14	115/60	4	8.8	98.7	96	-
					B	N	N	2	N	N	N	N	N	N	N	85	NSR	N	13.1	125/70	4.2	9.5	100.1	97	-
88	SENTHILKUMAR	45	M	2	A	N	N	1.5	N	N	N	N	N	N	N	75	NSR	N	12.1	100/65	3.6	9.8	98.6	97	-
					B	N	N	2	N	N	N	LOW	N	N	N	90	NSR	N	11.4	110/75	4.2	9	99.9	96	-
89	SELVARAJ	35	M	3	A	N	N	2	N	N	N	N	N	N	N	80	NSR	N	10.4	120/75	3.8	9.3	98.2	98	-
					B	N	N	1	N	N	N	N	N	N	N	109	NSR	N	9.8	110/80	4.3	10.3	100.8	94	-
90	GOPAL	40	M	1	A	N	N	2	N	N	N	N	N	N	N	82	NSR	N	12.6	110/70	4.5	10.4	98.9	97	-
					B	N	N	2.5	N	N	N	N	N	N	N	90	NSR	N	11.9	100/60	3.9	9.2	100	97	-
91	KAVIN	27	M	2	A	N	N	2.5	N	N	N	N	N	N	N	85	NSR	N	14.1	110/70	3.8	9.8	98.1	97	-
					B	N	N	2	N	N	N	N	N	N	N	85	NSR	N	13.6	110/60	4.1	8.9	100.6	97	-
92	MOHAMED ALI	29	M	6	A	N	N	2	N	N	N	N	N	N	N	70	NSR	N	10.7	100/70	4.2	9.4	98.5	98	-
					B	N	(+)90	3.5	N	N	(+)90	N	N	N	N	120	NSR	RIGHT	12.3	110/70	3.8	8.6	100.7	95	-
93	MUTHURATHINAM	43	M	8	A	N	(+)90	3.5	N	N	(+)90	N	N	N	N	110	NSR	RIGHT	11.5	100/80	4.3	10.2	99	98	-
					B	N	N	2	N	N	N	N	N	N	N	112	SINUS ARRYTHMIA	N	10.3	110/80	4.6	10.1	101	94	-
94	VELLINGIRI	55	M	1	A	N	N	2	N	N	N	N	N	N	N	89	SINUS ARRYTHMIA	N	10	130/80	4.7	10	98.6	97	-
					B	N	N	2.5	N	N	N	N	N	N	N	75	NSR	N	13.4	90/60	3.8	9	99.8	97	-
95	GOKUL	28	M	2	A	N	N	1	N	N	N	N	N	N	N	65	NSR	N	12.6	120/80	3.5	8.8	98.3	97	-
					B	N	N	2	N	N	N	N	N	N	N	85	NSR	N	10.4	130/80	4.1	8.9	100.6	97	-
96	DHEENADAYALAN	50	M	4	A	N	N	1	N	N	N	N	N	N	N	78	NSR	N	10.2	110/70	3.9	9.1	98.7	98	-
					B	N	N	2.5	N	N	N	N	N	N	N	117	NSR	N	10.7	100/60	4.7	9.6	100	95	-
97	KATHIRVEL	37	M	1	A	N	N	2	N	N	N	N	N	N	N	86	NSR	N	12.6	115/60	4.4	9.8	98.7	98	-
					B	N	N	1.5	N	N	N	N	N	N	N	90	NSR	N	12	125/70	3.8	10.4	100.2	97	-
98	PREMKUMAR	26	M	4	A	N	N	2	N	N	N	N	N	N	N	80	NSR	N	13	100/65	4.3	10.5	98.6	97	-
					B	N	N	2	N	N	N	N	N	N	N	115	NSR	N	10.5	110/75	4.2	9.8	100.4	94	-
99	RAMESH	47	M	1	A	N	N	1	N	N	N	N	N	N	N	80	NSR	N	9.9	120/75	3.8	9.9	99	97	-
					B	N	N	2	N	N	N	N	N	N	N	85	NSR	N	11.5	110/80	3.5	8.9	99.9	96	-
100	KANTHASAMY	58	M	4	A	N	N	2.5	N	N	N	N	N	N	N	75	NSR	N	10.8	110/70	3.4	9.3	98.3	97	-