

**“EARLY AND EFFECTIVE DRAINAGE OF
TUBERCULAR PLEURAL EFFUSION VIA
PIGTAIL CATHETER-
A DESCRIPTIVE STUDY”**

By

Dr. SUNIL KUMAR REDDY KARRI

**Dissertation Submitted to the
KLE UNIVERSITY BELGAUM, Karnataka.**

**In Partial Fulfillment of the requirements for the
degree of**

**MASTER OF SURGERY (M.S.)
IN
GENERAL SURGERY**

Under the Guidance of

**Dr. M.S. SANGOLLI MS
Professor**

**DEPARTMENT OF GENERAL SURGERY,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
BELGAUM-590010, KARNATAKA
MAY – 2011**

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Place: **Belgaum.**

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ABSTRACT

Background and Objectives.

Tube thoracostomy is used for therapeutic removal of fluid or air from pleural space. Placement of a large bore chest tube is an invasive procedure with potential morbidity and complications include hemothorax, perforation of intra-abdominal or intra-thoracic organs, diaphragmatic laceration, empyema, pulmonary edema and Horner's syndrome and therefore the use of pigtail catheter may be desirable. Tuberculous pleurisy can result in obvious clinical symptoms, pleural fibrosis, and pleural thickening. Some studies of tuberculous pleurisy have suggested that symptomatic improvement and minimisation of sequelae can be achieved by completely draining the effusion during treatment, although the results have not been conclusive and the objective was to evaluate the early and effective drainage of tuberculous pleural effusion via pigtail catheter and to evaluate the radiological response in the improvement of pleural effusion after pigtail catheter insertion

Methodology.

The present study was conducted in the Department of Surgery K.L.E.S Dr. Prabhakar Kore Hospital and Medical Research Centre , Belgaum on 40 patients diagnosed as tubercular pleural effusion between Nov 2008 to Nov 2009. Pre and post insertion chest radiographs are reviewed to determine efficacy of drainage. The patients were treated as per hospital protocols. They were followed up for 6 months. The outcome variables like amount of fluid drained, total duration of hospitalization, level of radiological resolution, RPT as assessed by chest x-ray, and complications were noted

Results:

There were 27 males & 13 females in the study. 60% of patients were above age group of 35, the mean amount of pleural fluid drained was 2575 ml, the mean duration of pigtail catheter was 4.5 days, the mean duration of hospitalization was 6 days, radiological resolution was clear in 75 % patients (during discharge) and minimal complications like chest pain(7.5%), pneumothorax (5%), tube blockade(7.5%) were seen and at the six months of ATT residual pleural thickening was seen in 50 % of cases.

Conclusion.

Early effective drainage and complete ATT with pigtail catheter hasten clearance of pleural effusion and is safe, efficacious, comfortable, decreased hospital stay, cost-effective, minimal complications and improves patient quality of life and does not reduce residual pleural thickening occurrence.

Key words: Tubercular pleural effusion; Pigtail catheter; Drainage.

Abbreviations : RPT= residual pleural thickening,ATT= anti tubercular treatment.

INTRODUCTION

Pleural effusion is the presence of excessive quantity of fluid in the pleural space¹. Though it produces minimal clinical manifestations, it should be considered as an ominous sign of a serious disorder. So no effort should be left in arriving at specific diagnosis to give the most rational treatment. Effusion may be transudative due to abnormalities of hydrostatic, or osmotic pressures, and exudative resulting from increased permeability or trauma. Aetiological diagnosis is often difficult. In developing countries tuberculosis is the most common cause of effusion, even then other causes should be excluded before concluding as tuberculosis.

Investigation of a pleural effusion demands a pleural aspiration and biopsy. The pleural fluid is sent for measurement of proteins and glucose content, cytological and microbiological examination. Cytology and microbiology benefit from testing as large as quantity of fluid as possible. A "diagnostic tap" of 10-20 ml of pleural fluid without a pleural biopsy is inadequate. In as many as 20% of all pleural effusions basic testing does not establish the diagnosis and even thoracotomy or thoracoscopy may not reveal the cause of the effusion².

The diagnosis of tuberculous pleural effusion is important because tuberculosis is normally a treatable cause of exudative lymphocytic pleural effusion. Other differential diagnoses of exudative lymphocytic pleural effusions are malignancy, fungal infection, melioidosis, sarcoidosis, and connective tissue diseases. The primary difficulty in getting a diagnostic confirmation of tuberculous pleural effusion is the identification of mycobacterium in the pleural fluid. Pleural biopsy is usually the main diagnostic support, but its invasive nature and the difficult technique that limits its practice.

Tuberculous (TB) pleurisy can cause clinical symptoms and pleural fibrosis

with resultant residual pleural thickening (RPT). Therapeutic thoracentesis or initial complete drainage in addition to anti-TB drugs has been tried in order to rapidly relieve dyspnoea caused by effusions and decrease the occurrence of RPT. Delayed diagnosis and or treatment of TB pleurisy may cause disordered fibrin turnover in the pleural cavity with subsequent fibrin deposition and loculation of pleural fluid, and may impair uneventful resolution of pleural effusion³.

The present study is being undertaken to investigate the usefulness of early effective drainage of pleural effusion in the treatment of TB pleurisy using a pigtail catheter which is relatively atraumatic and is associated with less number of complications as compared to the routine drainage using large bore tube thoracostomy.

AIMS OF THE STUDY

1. To evaluate the early and effective drainage of tubercular pleural effusions via pigtail catheter.
2. To evaluate the radiological response in the improvement of pleural effusion after pig tail catheter insertion.

REVIEW OF LITERATURE

HISTORICAL REVIEW:

Hippocrates first described Pleural effusion in fifth century B.C. in a case of pneumonia. In the fifth century B.C., A.D. Alexander gave an account of classical picture of pleurisy. The disease associated with cough, fever, blood spitting and emaciation was recognized as Laoping in China and Rajayakshma (king among diseases, Kshaya tissues getting withered) in India, was called phthisis by Hippocrates {460-377 BC}.^{4,5}

Sylvius (1614-1672) saw small rounded, whitish gray bodies called "tubercles" and considered them enlarged glands in the lungs due to accumulation of viscid humors. A clear transition in the perception of tuberculosis occurred with the contributions of Sylvius, Willis and Morton.⁵

Through experimental procedure Robert Koch (1882) established that tuberculous affections of man and animals are caused by tiny slender rod shaped bacilli and called them "tubercle bacilli".

X-rays discovered by Roentgen in 1895 revolutionized the method of diagnosis, detection of tuberculosis even at its early stages of development.^{4,5}

Calmette and Guerin accidentally found the cultures of bovine tubercle bacilli growing in glycerol potato medium altering their character on addition of ox bile.⁴

Defrancis and associates (1955) used Vim Silverman needle for first time for obtaining biopsies of parietal pleura. Abrams (1958) used Harrified Biopsy Needle, first designed by Leon D. Abram a surgical registrar in Harrified hospital therefore called as Harriefied needle.

No effective chemotherapeutic agent was available for treating tuberculosis till

as late as 1940.⁶ The introduction of major anti-tubercular drugs like para-aminosalicylic acid (1943) Streptomycin (1944) Iso nicotinic acid hydrazide (1952) have revolutionized the grim outlook of tuberculous patients. Many new anti-tubercular drugs have been introduced since then. Ethambutol and Rifampicin introduced initially for resistant cases of tuberculosis have found a prime place as first line drugs in the management of tuberculosis.^{5,6}

Emerson (1966) described pleural effusion due to lymphatic obstruction Yellow nail Syndrome. Gaensler and Kaplan 1971 described benign pleural effusion due to exposure to asbestos. Light and Ball in 1973 described the measurement of pleural fluid lactic dehydrogenase in discriminating transudates and exudates.^{4,7}

ANATOMY OF THE PLEURA:

Each lung is covered by a serous membrane arranged in the form of a closed invaginated sac termed pleura. It consists of two layers. A part of pleura adheres to the pulmonary surface and its inter lobar fissures as visceral or pulmonary pleura. Its continuation lines the corresponding half of the thoracic wall covers much of the diaphragm and structures occupying middle region of thorax, called as parietal pleura. The potential space between them being the pleural cavity. The left pleural cavity is smaller of the two because heart extends further left.^{7,8}

DEVELOPMENT:

During its separation from the foregut respiratory primordium forms a mid line structure the trachea and two lateral out pocketing, the lung buds. With subsequent growth in caudal and lateral directions the lung buds penetrate into the coelomic cavity. The mesoderm, which covers the outside of the lung, develops into the visceral pleura. The somatic mesoderm layers covering the body wall from inside

becomes the parietal pleura. The space between the parietal and visceral pleura is the pleural cavity.^{8,9,10,11.}

VISCERAL PLEURA (PULMONARY PLEURA)

It is inseparably connected with the lung. It covers the surface and fissures of the lung except at the hilum and along the attachment of pulmonary ligament, where it is continuous with the parietal pleura.⁸

PARIETAL PLEURA:

The different regions of parietal pleura have distinct names. The part internal to thoracic wall and vertebral bodies is the costo-vertebral pleura, that lining thoracic surface of diaphragm is Diaphragmatic Pleura, the part over the pulmonary apices (in the neck) is the cervical pleura, and that applied to the structures occupying interpulmonary region is mediastinal pleura.^{8,11}

MICROSCOPIC STRUCTURE OF THE PLEURA :

The pleura is a layer of connective tissue covered by a simple squamous epithelium. Pleural surface is smooth, moistened by serous fluid and consists of a single layer of flat mesothelial cells on a basal lamina beneath which networks of several prominent layers of elastic fibers, collagen tissue containing some fibroblasts and macrophages and other cells typical of areolar tissue are present.¹⁰

NERVE - SUPPLY:

Parietal pleura is supplied by somatic (body wall) nerves, inter costal and phrenic, making it pain sensitive. The visceral pleura develops from the splanchnopleural layer of the lateral plate mesoderm, and is supplied by the

sympathetic nerves (Autonomic T2-5) and vagus, accompanying the bronchial vessels, this part of pleura is pain insensitive.^{8,11,12}

BLOOD SUPPLY AND LYMPHATIC DRAINAGE:

Pleura is supplied by intercostal, internal mammary and musculo-phrenic arteries. The veins drain mostly into the azygous and internal thoracic veins.

The lymphatics drain into intercostal, internal mammary, diaphragmatic, inner deep cervical and into the upper most group of axillary glands.

The visceral pleura being an integral part of the lung, derives its blood supply mainly from bronchial and partly from pulmonary vessels. The veins follow the corresponding arteries. The lymphatics drain into the broncho pulmonary lymph nodes.^{8,11,12}

PHYSIOLOGY OF PLEURAL SPACE:13

Pleural pressure is the pressure of the fluid in the narrow space between the parietal pleura and the visceral pleura. The normal pleural pressure at the beginning of inspiration is about 5-cm water.¹³

Fluid can enter the pleural space from the capillaries in the parietal or visceral pleura or from the interstitial spaces or lymphatics or either pleural surface. The passage of protein free liquid across the pleural membrane is dependent on the hydrostatic and oncotic pressure's across them. When the capillaries in the parietal pleura are considered, it can be seen that the net hydrostatic pressure favoring the movement of fluid from these capillaries to the pleural space is the systemic capillary pressure (28 cm HbO) minus the negative pleural pressure (-5 cm FbO) or 33 cm HsO. Opposing this is the oncotic

pressure in the blood (30 cm H₂O) minus the oncotic pressure in the pleural fluid (4cm hbO), or 26 cm I-bO. The resulting net pressure difference of 7 cm H₂O (33-26) favors the movement of fluid from parietal pleura into the pleural space. Capillaries of the visceral pleura have a slightly lower hydrostatic pressure because they drain into the low-pressure pulmonary veins. The net force across the visceral pleura is 2 cm H₂O, again favoring the formation of pleural fluid.^{13,14}

Parietal pleura	pleural space	Visceral pleura
Hydrostatic pressure (cm H₂O)		
+28 _____	-5	^ ____ + 23
33		28
NET" —>7	2 *—	"NET
^ 26		26 ^
+30	+4	+30
Oncotic Pressure		

In recent years it has become apparent that the origin of much pleural fluid is the interstitial spaces of the lung. The pleural membranes are leaky to both liquid and protein. The pleural pressure is lower than the interstitial and this pressure difference produces a gradient for the fluid to move from interstitium to the pleural space.

The rate of pleural fluid formation in normal animals with thick pleura is approximately 0.01 ml/kg/hour or 15 ml/per 24 hours for a 60-kg individual. There is a small amount of protein in this fluid. Normally, the pleural space is maintained nearly fluid free because the filtered fluid is removed from the pleural space by the pleural lymphatics which can remove over 0.20 ml/kg/hour. Pleural space is called a potential space because it normally is so narrow that it is not obviously a physical space. In this

space pleural fluid will accumulate producing a pleural effusion when the rate of pleural fluid formation exceeds to capacity of the lymphatics in the parietal pleura to remove the fluid. The rate of turnover of pleural fluid in human is rapid and may exceed 1L per day.^{10,11,13,14}

PATHOPHYSIOLOGY OF PLEURAL EFFUSION :

Pleural fluid will accumulate when the rate of pleural fluid formation is greater than the rate of pleural fluid removal by the lymphatics. Pleural fluid will continue to accumulate until another equilibrium is reached. Pleural effusions have classically been divided into transudative and exudative pleural effusions. A transudative pleural effusion occurs when alteration in the systemic factors that influence pleural fluid movement result in a pleural effusion. Examples are increased pulmonary interstitial fluid and elevated visceral pleural capillary pressure with left heart failure, elevated parietal pleural capillary pressure with right heart failure, and decreased serum oncotic pressure with the nephrotic syndrome. In contrast, exudative pleural effusion occur when local factors are altered in such a way that pleural fluid accumulates. Inflammation of the lung or the pleura leading to increased flux of fluid from the capillaries of the lung or the pleura into the pleural space is the most common cause of exudative pleural effusion. However, exudative effusion can also occur with decreased lymphatic flow or with a more negative pleural pressure as with atelectasis.^{7,10,13,14}

Composition of pleural fluid: ^{5,11,15}(Table-1)

	Normal	Tubercular
Appearance	Clear	straw coloured
Volume	0.1-0.2ml/kg	min-massive
Cells/cumm	1000-5000	1000-6000
% monocytes	30-75%	few
% lymphocytes	2-30%	50-90%
% mesothelial cells	3-70%	Few
% granulocytes	10%	<10%
Proteins	1-2gm/dl	> 2.5gm/dl
Glucose	= plasma	usually < serum
LDH	< 50% plasma	> 2/3 plasma
pH	> plasma	7.3 -7.4
		rarely <7.3
		never > 7.4

AETIOLOGY OF PLEURAL EFFUSION :

The collection of appreciable amount of fluid in the pleural space is due to a number of factors. They are¹¹

1. Diseases primarily involving the pleura.
2. Diseases of the lung involving the visceral pleura secondarily.
3. Diseases of the chest-wall, mediastinum or diaphragm, involving the parietal pleura secondarily.
4. Due to factors causing generalized oedema.

ORIGIN OF PLEURAL EXUDATES AND TRANSUDATES

When a patient is found to have a pleural effusion, an effort should be made to determine the cause. The first step is to determine whether the effusion is a transudate or an exudate.¹

The initial step in analyzing the pleural fluid is to ascertain whether effusion is a transudate or exudate.

A transudative pleural effusion occurs when systemic factors that influence the formation and absorption of pleural fluid are altered. An exudative pleural effusion occurs when local factors that influence the formation and absorption of pleural fluid are altered.^{1,6,12}

CLASSIFICATION (Table-2)

Transudates differ from exudates in physical and biochemical nature.^{10,11,13}

Transudates	Exudates
1. Clear fluid and thin.	1. Opalescent or turbid and thick Sometimes purulent or mucopurulent
2. Often bilateral	2. Usually unilateral
3. Specific Gravity is low (Less than 1015)	3. Sp.gravity is high (More than 1016)
4. Proteins below 2 gms%	4. Proteins above 3gms%
5. Cells only a few lymphocytes	5. Increased number of cells lymphocytes or polymorphs.
6. Culture is sterile	6. Culture may yield organism
7. Does not clot on standing.	7. Often clots on standing.

The current criteria for differentiating between exudate and' transudative effusions rely on the fact that elevation of pleural fluid to serum ratios for proteins and lactate dehydrogenase reflects pleural inflammation.

Light's criteria were published by which an exudative is defined by fulfilling one or more of the following.^{1,7,11,13}

1. Pleural fluid/Serum protein ratio of more than 0.5.
2. Pleural fluid LDH of greater than 2/3rd the upper normal limits for blood LDH levels.
3. Pleural fluid/Serum ratio of LDH of greater than 0.6.
4. Pleural fluid LDH greater than 200 IU.

5. A pleural fluid protein greater than 3 gms/dl.

When these criteria are not met, effusion is classified as transudate. An albumin gradient of 1.2 gms/dl indicates presence of exudative effusions and greater than 1.2 gms/dl, a transudative effusion.

In view of the fact that treatment of patients with some exudative effusions in particular empyema often requires surgical intervention, Light has further subdivided exudate into simple and complicated exudative (paranpneumonic) effusion.

A complicated exudative effusion is an exudate also having one of the following additional criteria indicating the likely need for a procedure more invasive than a thoracentesis (in increasing order of importance) ^{1,6,10}

1. Loculated leural
2. PH-below-7.2
3. A glucose level below 60 mg/dl.
4. A positive gram stain or culture of the pleural fluid
5. Presence of gross pus in the pleural space.

The above criteria miss-identify approximately 25% of transudates as exudates. If one or more of the exudative criteria are met and the patient is clinically thought to have a condition producing transudative effusion, the difference between the albumin levels in the serum and the pleural fluid should be measured. If this gradient is greater than 12 g/l (1.2 mg/dl), the exudative categorization by the above criteria can be ignored because almost all such patients have a transudative pleural effusions.¹⁶

(Table 3)

CAUSES OF PLEURAL EFFUSIONS:^{1,7,11}

<p>A) PLEURAL TRANSUDATES :</p> <p>1. INCREASED HYDROSTATIC PRESSURE</p> <p>Congestive cardiac ailure Constrictive pericarditis Pericardial effusion Restrictive ardiomyopathy Massive pulmonary embolism.</p>	<p>B) PLEURAL EXUDATES:</p> <p>1. INFECTIONS</p> <p>Pneumonias, Abscess, Tuberculosis, Hepatic Amoebiasis Fungal and Actinomycotic Disease.</p> <p>Viral - HIV viral infection Subphrenic abscess Parasitic</p>
<p>2. DECREASED CAPILLARY OSMOTIC PRESSURE</p> <p>Cirrhosis with ascitis Nephrotic syndrome Malnutrition Protein losing enteritis Small bowel disease</p>	<p>2. NEOPLASMS:</p> <p>Bronchogenic carcinoma Pulmonary secondary Mesothelioma of pleura Lymphoma Leukemia Chest wall tumors Pleural sarcoma</p>
<p>3. TRANSMISSION FROM PERITONEUM</p> <p>Any cause of Ascites Peritoneal Dialysis Liver transplantation</p>	<p>3. IMMUNE DISORDERS</p> <p>Post, myocardial infarct Rheumatoid Disease Systemic Lupus Erythematosis Wegener's granulmatosis Rheumatic Fever</p>
<p>4. INCREASED CAPILLARY PERMEABILITY</p> <p>Small pulmonary emboli Myxoedema.</p>	<p>4. INTRA - ABDOMINAL DISEASES</p> <p>Acute Pancreatitis Uraemia Malignant peritonitis as in ca. liver, stomach, colon. Sub- diaphragmatic Primary mesothelioma of peritoneum Intra hepatic abscess - Amoebic Pseudo rnyxoma peritoni</p>

<p>5. MISCELLANEOUS</p> <p>Urinothorax Acute Atelectasis Wet Beriberi Idiopathic Subclavian catheter misplacement Lung transplantation Superior venacaval obstruction</p>	<p>5. PULMONARY EMBOLISM & INFARCTION</p>
	<p>6 DRUG INDUCED PLEURAL DISEASE Nitro furantoin, Methysergide, Practolol, bromocriptine, procarbazine, amiodarone</p>
	<p>7. HAEMOTHORAX</p>
	<p>8. CHYLOTHORAX</p>
	<p>9. MISCELLANEOUS</p> <p>Esophageal rupture Sarcoidosis Radiation Therapy Asbestos exposure Congenital Yellow Nail Syndrome</p>
	<p>10.Familial Paroxysmal Polyserosites, idiopathic</p>

CLINICAL FEATURES OF PLEURAL EFFUSION

SYMPTOMATOLOGY: ^{1,7,11,17,18}

The effects of accumulation of fluid in the pleural space depend on the cause and amount of fluid. The symptoms due to pleural effusions depends on how fast the fluid is collecting. Small effusions are often symptomless, and even very large effusions if they accumulate slowly they cause little or no symptoms, for the patients.^{7,10}

Symptoms indicating a pleural effusion include a feeling of

heaviness or tightness in the chest, pleuritic chest pain, shortness of breath due to diminished lung volume or underlying lung parenchymal disease, fever, which indicates an acute focus existing in the lung or exudate may be an empyema, and cough.^{7,11}

Pleuritic chest **pain** is one of the earliest symptoms in pleural effusions. Pain is perceived by the parietal pleura because only this surface is innervated by the pain fibres. Pain is more severe early in the development of effusion, when two pleural surfaces still rub against each other, and once the fluid starts accumulating, pain disappears. Pleuritic pain is catching in type and is aggravated on deep inspiration, coughing and movements of chest wall. When it is severe the patient breaths in short grunts and may attempt to splint the appropriate part of the chest by holding it or lying on it. The site of the pain is usually be localised quite closely and most frequently in the lower postero-lateral parts of the chest. Primitive pain may be referred to shoulder tip via phrenic nerve when the peripheral diaphragm is affected.^{1,7,11,17}

Dyspnoea is a usual symptom of a large effusion, which is often accompanied by dull ache on the affected side, this is especially likely if the effusion is due to malignant disease of the pleura. Even very large effusion if they accumulate slowly may cause little or no discomfort to the patient. The symptoms of cough in pleural effusion is not well understood, may be due to underlying parenchymal disease or compression of bronchial walls by the effusion, recurrent dry cough is frequently present, especially if the fluid has accumulated quickly.^{1,7}

Fever, if the degree of fever is out of proportion to exudate present indicates that there is an active focus existing in the lung or exudate may be empyema. Evening rise of temperature associated with sweating favors tuberculosis. Hectic temperature and constitutional symptoms are in favour of empyema, pneumonia and infarction. Fever may be absent in malignant effusion. There may be associated rigors. In about 82% of patients with pleural effusion there will be loss of weight. Some patients may complain of anorexia, night sweats, and lassitude for sometime before the onset of pleural effusion.^{1,11}

PHYSICAL SIGNS IN PLEURAL - EFFUSION:

If the amount of fluid is less than 100ml it cannot be detected by any means. 100-300 ml fluid can be detected by x-ray chest. To detect pleural effusion clinically there must be at least 500 ml of fluid in the pleural cavity.¹⁰

INSPECTION: 6,11,17,18

1. With large effusions preference is for lying on the same side, as well during fibrinous pleurisy
2. Fullness or prominence of the chest wall on the affected side.
3. Respiratory movements of the affected side may be diminished or absent.
4. In massive pleural effusion patient is dyspnoeic.
5. Mediastinal displacement to the opposite side.
 - a) Apex beat may be displaced to the opposite side in severe pleural effusion, unless there is an underlying collapse.

- b) Sternocleidomastoid sign (Trail Sign) Sternocleidomastoid muscle on the side of mediastinal displacement may be prominent.^{6,10,17,18}

PALPATION:^{17,18}

1. With large effusions, Trachea will be shifted to the opposite side, the Apex beat displaced to the opposite side, unless there is an underlying collapse. In such cases trachea may be pulled to same side, central or to opposite side depends on collapse or pleural effusion, which is dominant, this is referred to as collapse effusion sign.
2. Diminished mobility and expansion of the chest on the side of effusion.
3. Vocal fremitus is diminished or absent; in pleural effusion. It is not due to intervening fluid, but due to collapsed lung.
4. Tenderness over the intercostal space may suggest acute pleurisy or empyema.
5. A friction fremitus may be found occasionally during recovery stage.

PERCUSSION:^{6,7,17,18}

- 1) The stony dullness over an area of effusion is very characteristic. The highest point of dullness is situated in the axilla.
- 2) ELLIS-S-SHAPED CURVE:
It is also called Dumiseou's line or S shaped curve of Ellis. In

moderate sized effusions within the pleural sac, as one percusses in sitting positions, the upper boarder of dullness which is highest in the axilla and lowest at the spine (back) and reaches its summit in the midaxilla and then slopes downward as it passes anteriorly towards the sternum tends to assume the shape of letter S, and hence refereed as 'S' shaped curve.

3) SKODIAC RESONANCE:

A hyper resonant note with a boxy quality heard just above the level of pleural - effusions. It is heard in moderate pleural effusions. The characteristic note over such relaxed lung tissue (partially filled with air) is described as Skodiac resonance. This resonance becomes dull as the quantity of fluid increases.¹⁸

4) SHIFTING DULLNESS:

The upper level of fluid may vary with change of posture of patient in infrapulmonary effusions. If the effusion is massive or loculated this may not be demonstrable. It is of value in hydropneumothorax and of limited value in pleural - effusions. In most effusions fluid shifts on changing position, but not immediately except infrapulmonary effusions.

5) OBLITERATION OF TRAUBE'S SPACE :

It is a semilunar space overlying the stomach anteriorly. It is bounded on the right by left part of inferior margin of liver, above by the

left lung and on the left by spleen and below by the left costal margin.

Normally the percussion of this area gives a tympanic note due to presence of air in the stomach. This space will be obliterated in case of left sided pleural effusions.

6) GARLAND'S TRIANGLE:

In cases of moderate sized or large pleural effusions, the lung on the side of effusions floats upwards and backwards, its lower part being relaxed. The roughly triangular area with a slight tympanic note, that may be elicited in such a case by percussion over the relaxed area of lung is referred to as Garland's triangle. It is an inconstant sign.¹⁷

3. GROCCO'S TRIANGLE:

An area of relative dullness or impairment of note, roughly triangle in shape can some times be mapped out by percussion in large or medium sized pleural effusion over the back of the chest on the contralateral side.

The triangle is bounded medially by spinal line from above the level of effusion to the level of 10th thoracic vertebra, below by a horizontal line extending outwards from 10th dorsal vertebra along the lower limit of lung resonance for a distance of about 3.7 cms and laterally by a curved line connecting these two lines. The presence of Grocco's line is as a rule evidence of pleural effusion. Exact explanation for the sign is not known.

AUSCULTATION:^{7,11,17,18}

- 1) Diminished or absent breath sounds
- 2) Diminished or absent vocal resonance
- 3) Aegophony - It is the term applied to the spoken voice heard with nasal quality above level of pleural effusions. It is due to relaxed lung in upper part of effusions.
- 4) If there is collapse of the lung associated with patent ronchi, bronchial breathing is heard.
- 5) Pleural Rub -It is a friction rub heard above the level of effusion. It is due to roughened surfaces rubbing against one another.
- 6) Adventitious sounds may be heard above 'the level of effusions.

INVESTIGATIONS

RADIOLOGICAL FEATURE:^{6,10}

X-ray chest :

Free fluid in small amounts (about 100ml) may be seen as blunting of costophrenic angle in PA or lateral films. Before appearance of radiological shadow atleast 300ml of fluid must be present in the pleural space. However as little as 150 ml can be seen in lateral decubitus film. As the fluid rides up around the edges of the lung the attenuation of x-rays decreases the radiographic appearances suggest that the effusion is higher in axilla than anteriorly or posteriorly. If there is doubt about the presence of fluid radiographically, a lateral decubitus film may be taken to show the shift of effusion to the mediastinum or lateral chest wall.

This may help particularly in differentiating a small effusion from pleural thickening. Massive effusions may obscure the whole hemithorax and displace trachea and heart to the opposite side.^{7,11}

In a case of hydropneumothorax, a horizontal fluid level that extends unto periphery is seen with increased translucency of air above the fluid level. In the case of a sub pulmonic effusion (fluid situated in between base of the lung and diaphragm), it looks like elevated hemidiaphragm on the affected side. A radiograph taken in the lateral decubitus position confirms the diagnosis, and shows definite shift of fluid.^{1,7,11}

ENCYSTED EFFUSIONS:

Sometimes because of associated pleurisy and frequent adhesions the fluid may be trapped confining to different anatomical portions inside the lung giving rise encysted effusions causing diagnostic difficulties.^{7,11}

Anatomical types of Encysted Effusions are:

1. Parietal Effusions
 2. Interlobar effusions
 3. Mediastinal effusions
 4. Mediastino-inter lobar effusions
 5. Sub-pulmonic effusion or Diaphragmatic effusion
- ULTRASOUND, CT SCAN AND MRI are indicated in difficult cases if clinical diagnosis of plural effusion is doubtful or any complications are suspected .

II THORACOCENTESIS :^{6,10}

Usually a diagnostic aspiration is done, and should be preferred when cause of effusion is uncertain. Indications for diagnostic thoracocentesis are

- 1) Large pleural effusions up to clavicle
- 2) Cardiac or respiratory embarrassment
- 3) Bilateral effusions
- 4) Secondary infection of effusion
- 5) If effusion does not tend to get absorbed spontaneously even when anti tubercular treatment is being given .
- 6) Synpneumonic effusion: The effusion associated with Bacterial pneumonia is initially sterile. However it may be frequently invaded by the Bacteria leading to empyema or eventual healing by fibrosis. For this reason aspiration is as far as possible to dryness is necessary. At the time of presentation, it is unwise to wait in hope of resolution of effusion with antibiotic treatment.

Therapeutic thoracocentesis usually involves large amount of fluid. However not more than 1000 - 1500 ml of fluid should be removed at one time, since expansion pulmonary oedema may occur as fluid compressing the underlying lung is removed.

III ANALYSIS OF PLEURAL FLUID : 4,7,11,13,19

A PHYSICAL EXAMINATION

1 COLOR AND ODOUR:

The fluid of a transudate is clear and pale. Exudate tend to be more amber colored and turbid if cell count is high; They are odorless.

Uniform blood staining of a red or brown color frequently means pleural tumor, pulmonary infarction. Tumor may be hemorrhagic Turbid yellow suggests infection including tuberculosis. Turbid green suggests rheumatoid pleuritis. Milky white fluid which is cloudy is usually due to chyle. A pleural effusion is designated as chylothorax when triglyceride level exceeds 1.2 mmol (110mg/dl).^{4,7,11,19}

2. SPECIFIC GRAVITY: 7,11

Pleural fluid transudates generally have specific gravity values less than 1.016, and exudates have specific gravity values more than 1.016, Specific gravity depends upon the protein content. It showed a sensitivity of 78% and specificity of 89%.

B. BIOCHEMICAL ANALYSIS OF PLEURAL FLUID:

1. TOTAL PROTEIN CONTENT

Pleural fluid is classically divided into transudate and exudate on the basis of protein content of pleural fluid.¹

2. GLUCOSE AND pH: 7,11

The concentration of Glucose in pleural fluid transudates is

normally more than 60 mg %. The pleural fluid to serum glucose ratio is more than 0.5 Low levels of PH and glucose are found in ernpyema, malignancy, tuberculosis, rheumatoid arthritis, SLE. and esophageal rupture. Thickening of pleural membrane appears to be most important factor in low PH and glucose in Rheumatic pleural effusion One third of patients with malignant pleural effusion has pleural fluid PH below 7.30(range 6.90-7.29).

3. AMYLASE

Amylase levels may be very high (over 100 units) in effusions due to Pancreatitis and esophageal rupture.

4. LIPIDS:^{7,11,19}

A pleural effusion that contains chyle is chylothorax, and the milky or opalescent fluid indicates presence of increased lipid. Hamm et al ¹⁹ measured cholesterol levels in pleural effusions in 62 patients with pleural effusions of known etiology. They found that using a value of 60mg/dl as a cut off (> 60mg/dl indicates an exudate, < 60mg/dl a transudate). Test was 100% sensitive and 95% specific (Versus 100% and 70% for light's criteria) all transudate effusion had cholesterol values below 60mg/dl (Mean 30±12mg/dl) and all malignant effusion had cholesterol values greater than 60mg/dl (mean 94±25 mg/dl), therefore the test could separate definitely these two categories of effusions. Patients with exudative pleural effusions had values between

these two groups (Mean 76 mg/dl).

5. LDH : In all exudative pleural effusion, there will be increased LDH (Lights criteria)

6. COMPLEMENT

Complement levels have been reduced in effusions associated with SLE and Rheumatoid diseases when compared to other inflammatory and malignant effusions. Low pleural fluid total hemolytic complement C1q binding, C3 and C4 have been found in patients with lupus pleural effusions. In Rheumatoid pleurisy, total hemolytic complement and complement compounds have reported to be low and immune complexes found to be increased.^{11,19}

7. VALUE OF ADENOSINE DEAMINASE, LYSOZYME, WITH INTERFERON AND SOLUBLE INTERLEUKIN-2 RECEPTOR LEVELS IN PLEURAL EFFUSION

A frequent diagnostic problem is that of exudative pleural effusions with Negative cytology and fluid Lymphocytosis. Possible diagnosis include Tuberculosis, collagen vascular disease, malignancies including lymphoma. ADA levels is found markedly elevated in Tubercular pleural effusion (Mean 92 U/L) as compared to malignant effusions (Mean 13 U/L). Levels greater than 45 U/L are highly sensitive and specific tubercular pleural effusions, although Rheumatoid effusions have

similarly elevated levels.

Increased pleural fluid lysozyme (muramidase) levels and pleural fluid/serum lysozyme ratios (> 1.2) also differentiate between malignant and tubercular effusions. (Sensitivity 100% specificity 94.9%) lysozyme levels are also increased in empyema and rheumatoid arthritis.

Combined use of lysozyme in pleural fluid/serum and ADA showed elevated ADA (>33 U/L) and elevated pleural fluid/serum lysozyme ratio (>1.2) yields a sensitivity and specificity of 100% for tubercular effusions if empyema is excluded.

With interferon is increased in Tubercular effusions in comparison with effusions due to other causes. ($91.2\mu\text{/ml}$ Vs $2 \mu\text{/ml}$). Soluble interleukin 2 receptor levels have also been found to be higher in Tubercular effusions as compared to malignant and transudative effusions. In contrast interleukin do not clearly differentiate malignant from tuberculous effusions.^{11,19}

C. CYTOLOGICAL ANALYSIS OF PLEURAL FLUID :

1. CYTOLOGY

Pleural fluid analysis should always include a differential cell count and careful cytological examination to identify malignant cells. Normal fluid contains 1,500 cells/ μL with predominance of monocytic cells, some lymphocytes, macrophages and mesothelial cells with rare-polymorphs. When fluid is a transudate (eg : CCF), mesothelial cells and lymphocytes accounts majority of cells ($<1,000/\mu\text{L}$), and polymorphonuclear leucocytes (PMN) does not exceed 10% of total cell

count-. Cell counts $> 50,000/\mu\text{l}$ are seen in parapneumonic effusions, with a predominant PMNs. Tuberculosis and malignancy have cell counts $< 5,000\mu/\text{ml}$, a predominance of lymphocytes is suggestive of tuberculous pleurisy. Pleural fluid eosinophilia ($> 10\%$) is usually associated with benign diseases especially post haemothorax pneumothorax, previous thoracocentesis and asbestoses associated effusions.^{7,11,13}

The examination of pleural fluid for malignant cells may lead to diagnosis. At least 25-30 ml of fluid should be submitted for cytological evaluation. The commonest cause of malignant pleural effusion is primary' Adenocarcinoma of lung followed by breast ovary and pancreases. In young patients lymphoreticular malignancies are the commonest cause of malignant effusion. In the cases of malignant effusion cytology alone is diagnosing in 33.72% of cases.^{7,11,13}

D. BACTERIOLOGICAL ANALYSIS OF PLEURAL FLUID:

1. DIRECT SMEAR AND CULTURE:

It is useless to do a direct smear for tubercle bacilli, since 60-80% of tuberculous effusions it is negative.⁵

Since pleural space is normally sterile, isolation of microorganisms on anaerobic, tuberculosis or fungal cultures of pleural fluid provides definitive evidence of infections.

2. PATHOGENICITY TESTS

The Guinea pig is the most suitable animal for diagnostic inoculation, owing to the high susceptibility of tuberculosis' Pleural fluid of 300-500cc is centrifuged and sediment of

200cc is taken for Inoculation.^{7,11}

IV. PLEURAL BIOPSY:

To prove the exact pathological process and aetiology of the pleural effusion. The different needles used for pleural biopsy are :

- 1 Abram's punch biopsy needle
2. Cope's Needle

Vim Silvermann Needle.

Abram's needle is superior to all needles and is now widely used everywhere. Presence of predominantly small lymphocytes (less than 50% of cells in an undiagnosed pleural fluid exudate) has been suggested an indication for pleural fluid biopsy an indication for malignancy and tuberculosis. In tuberculous pleuritis, needle biopsy of pleura is positive for granulomata or mycobacteria in upto 80% of patients.

The diagnostic yield of pleural biopsy for malignancy is generally less than the yield of pleural fluid cytology & histopathological confirmation being attained in 40-60% cases. Closed pleural biopsy is indicated in patients with an undiagnosed pleural effusion in whom malignancy or tuberculosis is suspected. The combination of thoracocentesis and closed biopsy increases diagnostic yield to 86% for tuberculosis and 79% for malignancy. Repeat thoracocentesis was positive in initial false negative tests, and therefore recommend a negative test should be repeated. Closed biopsy is having a

complication rate of 11 % commonest being pneumothorax, although vascular trauma has been reported.^{6,11}

V. THORACOSCOPY IN PLEURAL EFFUSIONS :^{20,21}

Following a complete diagnostic evaluation including closed pleural biopsy 20% of effusions remains undiagnosed. In these patients diagnostic rigid thoracoscope is advocated and has repeatedly been shown to be highly sensitive (93-97%) and specific (100%) for the diagnosis of tuberculous and malignant pleural effusions.²⁰ When thoracoscopy accurately identifies benign pleural effusions as non-malignant, seldom helps in identifying the cause of effusion.

VI. BRONCHOSCOPY:²¹

In cases of undiagnosed pleural effusions bronchoscopy is usually not helpful, unless there is other pathology on chest X-ray or the patients also has haemoptysis.

VII. SPUTUM EXAMINATION

Sputum examination for evidence of AFB in tuberculous effusion may not be of much importance. In primary effusion Sputum may not be available. But in effusions with primary parenchymal tuberculosis, AFB may be positive as demonstrated by Ziehl Neelson stains.^{4,7,11}

VIII. LYMPH-NODE BIOPSY :

Tuberculous lymphadenitis, malignancies like bronchogenic carcinoma, Hodgkin's disease and breast cancer with effusion can be diagnosed by Lymph node biopsy. Axillary node biopsy or scalene node biopsy indicated, if there is secondaries in the Nodes.

IX. TUBERCULIN TEST (MANTOUX TEST).

Mantoux test is helpful in the diagnosis of tuberculous pleural effusion when other tests are inconclusive.

X. ROUTINE INVESTIGATIONS:

1. HAEMOGLOBIN:

Normocytic hypochromic anemia may be noted. Iron deficiency anaemia exists in many patients. Recurrent haemoptysis may lead to iron' deficiency. Malignant effusion patients become anemic early.

2. WBC COUNT: Total count with polymorphonuclear neutrophilia suggest parapneumonic effusions, lymphocyte predominance suggests tubercular effusion.

3. EOSINOPHIL COUNT: Eosinophilic leucocytosis Eosinophilic effusion.

4. ERYTHROCYTE - SEDIMENTATION RATE :

ESR is raised in extensive disease. Raised ESR is of prognostic significance, rather than of diagnostic significance. An ESR may remain elevated in inactive cases and normal in active cases. However its importance has remained in assessing the activity of the disease.

XI. OTHER NEWER DIAGNOSTIC MODALITIES :

- a) Rapid methods for culture of tubercle bacilli.
- b) Semi-Direct tests
- c) Detection of specific DNA sequence by PCR (Polymerase chain Reaction).

a) Rapid method of culture for tubercle bacilli : ^{5,22}

During the last two decades, several methods for achieving early growths of *M.tuberculosis* have been developed.^{22,23,24}

- i) **BACTEC system:** This assay system developed by Becton Dickinson, is based on generation of radioactive carbon dioxide from substrate palmitic acid.²⁵ To a 4ml of middle brook 7H12 broth containing 1 IC-labelled palmitic acid (BACTEC 12 B vial) 0.5ml of processed sample is added along with a mixture of antibiotic. In this manner growth can be ascertained by liberation of ¹⁴C as fatty acids are metabolized by bacteria and detect by BACTEC 460 instrument, with this system average time o recovery of *M.tuberculosis* for smear positive specimen is 8 days and in smear negative it is 14 days.

- i) MGIT (Mycobacterial growth indicative tube) : This method has also been developed by Becton Dickinson and growth is detected by a radioactive detection system using fluorochromes.²⁶ This system consist of round bottom tubes containing 4 ml of modified middle Brook 7H9 broth which has oxygen sensitive fluroscent sensor on the bottom. When micobacteria grow they deplete the dissolved oxygen in the broth and allow the indicator to fluorescent brightly in 365 nm UV light. Positive signals are obtained in 7-12 days.^{23,24}
- ii) Septicheck MB system¹⁵ ; It is a biphasic medium consisting of 30ml Middle Brook 7H9 broth under enhanced CO2 to which a paddle is attached after inoculation of bottle or paddle part of the unit. Overall cultures can be detected in the broth or paddle part of the unit. Overall recovery in Septicheck system has been 80 to 95% compared to BACTEC at 71 to 95% and conventional solid media at 54 to 93%. With regard to time, the detection septicheck appears to be slightly better (about 20 days) than solid media but inferior to BACTEC.
- iv) Reporter phages : Mycobacterial specific phages and reportive genes like luciferace have been successfully used for detection of growth.^{5,15}

SEMI-DIRECT TESTS :

These include tests demonstrating the mycobacterial antigen either free or complex in mycobacterial immune complexes and revealing

mycobacterial DNA.

Presence of tuberculosis specific antigen indicates active infection, these antigens can be detected in many body fluids.

- i) Using Monoclonal antibodies : hybrid cell clones developed from hybridization technique, produce specific antibodies against M.tuberculosis. The major advantages of monoclonal antibodies are high specificity and absence of cross reactivity. The use of monoclonal antibodies to detect LAM antigen is a specific mode. A reverse passive haemagglutination test utilizing red blood cells coated with ML34 monoclonal antibody can neatly detect LAM antigen.²⁷
- ii) ELISA : It detects mycobacterial antigens and the test is highly sensitive (85%) and specific (97%). Antigens used are Ag-5, A60 and 38Kda antigen.⁵
- iii) Latex agglutination and biotin-avidin radioimmunoassay: are useful in detecting M.tuberculosis antigen in CSF.

Tuberculostearic acid :

(R-10-Methyloctadecanoic acid) is a structural component of mycobacterium and other members of actinomycetes such as nocardia and actinomyces and is not normally present in human tissues. It can be detected by using highly sensitive technique of gas chromatography/mass spectrometry (GC-MS) with selected ion monitoring (SIM) in various biological fluids. The main drawback of Gas-chromatography technique is the need for costly and complex analytical equipment.

Rapid identification of mycobacterial isolates:

Analysis of lipid profile

DNA probes

Microsomal RNA based probes

Gene amplification methods.

Polymerase chain reactio (PCR):

This is the new and most powerful technique of molecular biology used to assay body fluids for mycobacterial DNA. The various PCR assays used for identifying mycobacterial DNA includes:

- a) Amplification of genes encoding mycobacterial antigens such as 65Kda, MPB 64 protein or antigen b.
- b) Amplification of repetitive sequences including IS6110.
- c) Amplification of ribosomal RNA.

PCR is highly sensitive that it can detect as low as 10 bacilli in the sample. PCR has great diagnostic potential, its only disadvantage being, DNA from dead bacilli can be amplified. This could be a problem if PCR was used to monitor drug therapy or reactivation of disease.^{5,27}

Indirect tests :

Indirect tests measure the host response to infection by mycobacterium. They include –

- i) Elevated adenosine deaminase
- ii) Estimation of interferon

DIAGNOSIS:

The diagnosis of pleural effusion is two fold, viz. the anatomical diagnosis of the presence of effusion and its etiological diagnosis. The signs we elicit make the anatomical diagnosis of the effusion in the clinical examination. In a severe pleural effusion they are made by the presence of triad of signs - Mediastinal shift, stony dullness and diminished or absent air entry, and confirmed by thoracocentesis. The etiological diagnosis can be only be done by history, Radiographic features, Biochemical analysis, cytological analysis, Bacteriological analysis, later confirmed by a pleural biopsy.

TUBERCULOUS PLEURAL EFFUSION:

- a. It usually occurs in young persons between 15-25 years.
- b. H/O contact with tuberculous infection
- c. Strong positive Tuberculin test.
- d. Presence of constitutional symptoms
- e. Fluid is straw colored, clear and it is an exudate.
- f. Predominance of lymphocytes, absence of mesothelial cells, high blood ESR.
- g. Radiological evidence of pleural effusion.
- h. Biochemical analysis of ADA, lysozyme, and with interferon shows high value suggesting tuberculosis.
- i. Pleural biopsy shows characteristic tubercles.

TUBERCULOUS PLEURAL EFFUSION:^{1,7,10,11,13}

Epidemiology

The commonest cause of unilateral exudative pleural effusion in

our country is tuberculosis. Pleural effusion is uncommon below the age of 5. Pleural effusion has a fairly constant time relationship to the primary infection. Most cases occur 3-6 months or almost 1 year after the primary infection. Usually effusion is small to moderate in size, rarely they occupy an entire hemithorax. In about 2/3rd of patients there is no evidence of active parenchymal disease. Occasionally the initial primary infection goes unrecognized and by the time, effusion is detected, lung fields are clear like primary infection. Effusion is more common on the right side. Classical tuberculous pleurisy occurs in young in the absence of radiological and bacteriological evidence of pulmonary tuberculosis. However in current North American experience and also in India, tubercular effusion presents in many individuals with pulmonary Tuberculosis past age of 40.^{1,7}

Pleural effusion may occur as a complication of tuberculosis in three situations. In all cases disease is due to actual infection of pleura by tubercle bacilli through tuberculin hypersensitivity probably plays a part in potentiating the reaction.¹¹

First effusion may occur as a part of primary tuberculosis in children, when peripheral focus or caseating lymph nodes ruptures into the pleura. The disease is now relatively uncommon in developed countries, but typically present between ages of 5 and puberty and occurred in about 7% of patients with primary tuberculosis.

Secondly Pleural effusion present in adolescents or young adults often after a few weeks of malaise with acute pleuritic pain and fever.

The illness may initially manifest itself with recurrent dry pleurisy and all evidence of disease may disappear without treatment over a few months. However upto 2/3rd of patients will develop pulmonary tuberculosis with the ensuing five years. Moreover a proportion of patients in whom treatment is started late will have pulmonary fibrosis which may cause serious restriction in improvement of lung functions and require surgical pleurectomy.^{11,13}

The third type of effusion also seen relatively rare in the west, occurs when a tuberculous cavity in a patient with extensive post primary disease ruptures into the pleura. This usually causes a tuberculous pyopneumothorax. Patients become breathless and complain of pleuritic pain malaise and fever. Bronchopleural fistula may result and may cause considerable management problems. A fatal outcome is not infrequent in these individuals. When resolution takes place, Chronic fibrothorax is almost always the result, with extensive cavitation. This was also often the outcome of pleural effusions complicating artificial pneumothorax, treatment for tuberculosis in the prechemotherapy era.^{11,13}

Occasionally a paravertebral abscess associated with tuberculosis of thoracic vertebrae abuts on the parietal pleura results in pleural effusion which is sometimes bilateral and may progress to empyema. Tuberculous osteitis of ribs may be complicated by pleural effusion. Pleural effusion now occurs most often in older patients who may not have scarred lung lesion. Bilateral effusions may be due to of miliary

tuberculosis that is now seen more commonly in elderly people. Effusion nearly always occurs on the same side of the pulmonary focus and enlarged tracheo bronchial lymph nodes. In these cases effusion is fairly certain due to direct extension to the pleura from a sub pleural primary focus or from caseous lymphnode.^{7, 11}

MANAGEMENT OF TUBERCULOUS PLEURAL EFFUSION:

The treatment of tuberculous pleuritis has three goals :

- i) To prevent subsequent development of active TB
- ii) To relieve patient symptoms
- iii) To prevent development of fibrothorax

PREVENTION OF SUBSEQUENT ACTIVE TB :^{4,7,13}

The condition needs active treatment with antitubercular drugs even in the absence of pulmonary lesions as there is always a chance of later appearance of pulmonary tuberculosis. Patiala²⁸ followed up for atleast 7 years all 2816 members of the finnish armed forces who developed pleural effusions between 1939 and 1945. They reported that 43% of this large group of young men developed TB during the follow up period.

Confirmatory evidence for this large series comes from the series of Roper and Waring²⁹ in United States, who followed up 141 military personnel first seen from 1940 to 1944 with pleural effusion and a positive PPD test. In most patients, the effusions resolved and all other

symptoms disappeared within 2 to 4 months. Nevertheless, 92 of the 141 individuals (65%) subsequently developed some form of active TB. The incidence of subsequent TB was 60% in those with initially negative pleural fluid cultures for TB and 65% in those with initially positive pleural fluid cultures.

Because the administration of antituberculous chemotherapy reduces the incidence of subsequent TB^{30,31}, it is important to establish the diagnosis of tuberculous pleuritis and initiate proper treatment

CHEMOTHERAPY:^{32,33}

The initial phase of a 6-month regimen should consist of a two month period of isoniazid (INH), rifampicin and pyrazinamide. Ethambutol should be included in the initial regimen until the results of drug susceptibility studies are available, unless there is little possibility of drug resistance. The second phase of the treatment should be INH and rifampicin given for 4 months. DOT is recommended. Nine month regimen using INH and rifampicin are also effective when the organisms are fully susceptible to the drug.

The patient with isolated tuberculous pleuritis appears to have small bacterial burden because the symptoms are mainly due to delayed type of hypersensitivity. In the series by Patiala and Mattila³¹, the administration of chemotherapy decreased the incidence of subsequent active TB from 28% to 9%. Falk and Stead³⁴ reported that ATT reduced the incidence of subsequent TB from 19% to 4%.

The patients symptoms and radiologic abnormalities gradually disappear with treatment. The average patient becomes afebrile within 2 weeks, but temperature elevations may persist for as long as 2 months³⁵. If a therapeutic thoracocentesis is performed at the same time that antituberculous therapy is initiated, most patients become afebrile within 5 days.^{36,37}

2. TO RELIEVE SYMPTOMS:

- a) Pain: Can be relieved by simple analgesics. If the pain is still persisting, then local blockage of intercostal nerve supplying the painful area may be tried.
- b) Dyspnoea: Which may be very severe enough to cause respiratory distress, which can be relieved by thoracocentesis.
- c) Night Sweats: Severe night sweats can cause electrolytic imbalance and electrolytes have to be supplemented.
- d) Pyrexia: Anti pyretics can be used.
- e) Cough : Severe cough can be treated with antitussive drugs.

3. PREVENTION OF RESIDUAL FIBROTHORAX:

The incidence of pleural thickening at 6 to 12 months after beginning the treatment is approximately 50%³⁸. In a study done by Wong³⁹, 8 of 81 patients (10%) had a forced vital capacity (FVC) less than 80% predicted at the end of follow up period for their TB treatment. In this study there was weak correlation between the degree of pleural thickening and the reduction in the FVC.

The incidence of residual pleural thickening is not closely related to the initial pleural fluid findings; patients with a low glucose, high LDH and high cytokine levels are only slightly more likely to have residual pleural thickening.^{38,40}

Large et al⁴¹ studied the effect of aspiration in primary tuberculous pleural effusion. Out of 52 patients included in the trial, 33 were treated by repeated aspirations and 19 by single diagnostic aspiration along with ATT (Inj.streptomycin 1g daily with isoniazid 100 mg twice daily till 100 gm of streptomycin was given and then 1g was given on alternate days till a total of 140gm was reached). The results were compared in terms of duration of pyrexia, rate of clearance of fluid and residual opacity after 6 months. Although the repeated aspiration group tended to clear rapidly in the early stages, there was no significant difference between the two groups in the amount of pleural opacity remaining at 6 months.

Lai et al⁴² randomized 61 patients to receive pigtail drainage until the drainage was less than 50mL/day or no drainage. The degree of residual pleural thickening was basically identical in both groups. This shows that complete removal of pleural fluid alone does not reduce the amount of residual pleural thickening.

One randomized controlled study⁴³ of 52 patients demonstrated that intrapleural administration of 2.5mL of a hyaluronate-based gel resulted in significantly faster fluid absorption and significantly less pleural thickening at 3 months (0.57 vs 1.14 cm)^{cc}. Residual pleural thickening is more common if pleural effusion is initially loculated.

Role of corticosteroids:

The role of oral corticosteroids in the treatment of tuberculous pleurisy is

controversial. Galarza et al ³⁶ conducted a randomised trial for the role of corticosteroids in the treatment of tuberculous pleurisy. Patients received isoniazid (5mg/kg) and rifampicin (10mg/kg) for 6 months. Additionally they were randomly assigned to a double blind treatment with either prednisolone (1mg/kg/day for 15 days and then tapering off) or placebo during the first month of treatment. Out of 117 patients 57 received prednisolone and 60 placebo. They studied the clinical outcome, rate of reabsorption of the pleural fluid and pleural sequelae at the end of the treatment and concluded that the corticosteroids do not influence the clinical outcome or the development of long term pleural sequelae in tuberculous pleurisy.

In another study done by Lee CH ⁴⁴ it was shown that the corticosteroids decreased the duration of fever and the time required for fluid resorption, but no influence on the degree of residual pleural thickening was noted after 6 to 12 months after initiation of therapy.

ROLE OF INTERCOASTAL TUBE DRAINAGE:

A chest tube (chest drain or tube thorostomy in British medicine or intercostal drain) is a flexible plastic tube that is inserted through the side of the chest into the pleural space.. It is also known as a Bülow drain or an intercostal catheter.

INDICATIONS:

- 1) Pleural effusion
- 2) Pneumothorax
- 3) Chylothorax
- 4) Empyema

5) Hemothorax

6) Hydrothorax

CONTRAINDICATIONS:

Contraindications to chest tube placement include refractory coagulopathy, lack of cooperation by the patient, and diaphragmatic hernia. Additional contraindications include scarring in the pleural space (adhesion)

CHEST TUBE INSERTION

Chest tubes are inserted in to pleural space by four methods; tube thoracostomy with a guide wire and dilators, tube thoracostomy with a trocar, operative tube thoracostomy, and tube thoracoscopy through a single port thoracoscope. There are no controlled studies comparing the efficacy of the different methods of placing chest tubes.⁴⁵ If the chest tube is inserted to drain blood, pus, or another fluid from pleural space, the patient should be seated when the tube is inserted to ensure that the diaphragm is in the most dependent position and the fluid is collected in the lower part of the chest. When a chest tube is placed for pneumothorax, the patient should be recumbent if an anterior chest tube is placed, and should be in decubitus position if an axillary tube is placed.

Verification of chest tube placement

After the chest tube has been inserted and connected to a drainage system, a chest radiograph should be obtained to verify the correctness of its position. Ideally, both a posteroanterior (PA) and a lateral view should be obtained, because certain

ectopic locations may not be apparent on the PA view alone⁴⁶. A CT scan should be obtained when the chest tube does not drain adequately and the chest radiograph is not contributory⁴⁷ with CT, the tube can be visualized over its entire course with accurate location at its tip.⁴⁷

If there are undrained locules of fluid, additional chest tubes can be inserted. Interestingly, chest tubes frequently end up in fissures, even with operative tube thoracostomy. Curtin et al⁴⁸ reviewed the PA and lateral chest radiograph in 50 patients who had 66 chest tubes placed in the emergency room for trauma. They reported that 38 of 66 tubes (58%) were within a fissure decreased its functional effectiveness⁴⁸. It is possible that some of the chest tubes were not in the fissures originally but became positioned in the fissures after the fluid was drained.

PLEURAL DRAINAGE SYSTEMS

Chest tubes are inserted into pleural space to evacuate air or fluid. Because the pleural pressure is usually negative, at least during part of respiratory cycle, various methods have been developed to prevent air from entering the pleural space when the pleural pressure is negative but to permit air or fluid, or both, to drain from pleural space continuously when managing patient with chest tubes, one must understand how these various drainage systems operate. In the past, the bottle system was used for pleural drainage. Commercially manufactured collecting systems have subsequently replaced the bottle system.

AMOUNT AND TYPE OF DRAINAGE.

The amount and the character of the drainage from the chest tube should be recorded for each 24-hour period. The amount of drainage is most easily quantitated

by marking the level of liquid in the collecting chamber each day. This record-keeping is important because many therapeutic decisions are based on quantity of drainage. The character of drainage is best described by quantifying the percentage of solid drainage material. This quantification is easily done by marking the level of the sediment in the collection chamber each day. If the increase in volume of the entire collection system is known and if the increase in solid sediment is known, it is simple to calculate what percentage of the daily drainage is solid.

COMPLICATIONS OF TUBE THORACOSTOMY

There are numerous complications of tube thoracostomy. One of the most common complications is misplacement of the chest tube. The incidence of this complication has varied markedly. In one study, the incidence of malpositioned chest tubes was 26% with the emergency insertion of 77 tubes.⁴⁹ In contrast, the incidence of chest tube malposition was only 1% in one series of 449 patients in whom the chest tubes are inserted through blunt dissection. A PA and lateral chest radiograph should always be obtained after a chest tube is inserted. It should be noted, however, that frequently the malposition is not diagnosed by these routine films. Accordingly, if the patient has an air or fluid collection that is not being drained adequately, a chest CT scan should be obtained to assess the position of chest tube. In one series, malpositioned chest tubes are diagnosed by supine chest radiograph in only 7 of 20 patients (35%) whereas the CT scan was necessary for the diagnosis in the remaining 13 patients.⁴⁹

Many life-threatening complications occur when the tube is first inserted and include insertion of the chest tube into lung, stomach, spleen, liver, or heart.⁵⁰ These complications occur very rarely and are more likely when a trocar chest tube is used

or when the tube is inserted with out digital exploration of the insertion site. There is one case report in which a patient developed reversible cardiogenic shock owing to chest tube compression of the right ventricle.⁵¹ There is another report of two patients who developed tension pneumothoraces because the flutter valves were hooked up backward to the chest tube.⁵²

Pleural infection is another complication of tube thoracostomy. The administration of antibiotics to patients who have chest tubes for thoracic trauma may decrease the prevalence of empyema. Brunner et al.⁵³ randomly allocated 90 such patients to receive cefazolin or nothing immediately before and then every 6 hours until tube removal. They reported that there were six empyema cases and three of pneumonia in the control but only one case of pneumonia and no empyema in the antibiotic group.⁵³ Two subsequent studies, which also evaluated trauma patients, reported similar results^{54,55}. In view of the three studies, prophylactic antibiotics are recommended for all trauma patients who receive a chest tube. It should be noted, however, that in recent multicenter randomized double-blind study with a total of 224 patients, antibiotics had no significant effect on incidence of empyema.⁵⁶ However, the only two empyemas occurred in the placebo group⁵⁶.

The antibiotic chosen should have activity against staphylococcus aureus this is the organism that causes most infections⁵⁵ utility of prophylactic antibiotics in other situations such as in postoperative patients, patients with spontaneous pneumothorax, and those with malignant effusions undergoing pleurodesis, is yet to be evaluated

Another occasional complication of tube thoracostomy is the development of subcutaneous emphysema, which usually presents as a soft tissue crepitus around the

drain site but may rapidly spread to virtually any place in the body. The presence of subcutaneous emphysema in patients with tube thoracostomy indicates one of three possibilities:⁵⁷

- a) a side hole on the chest tube is lying outside the pleural space within the chest wall, allowing air to enter the tissue planes, b) the chest tube is blocked, or c) the drainage system cannot cope with air leak

The insertion of chest tube creates inflammation pleural space. Carvalho et al .⁵⁸

Studied the pleural fluid characteristics of sheep with an experimental pleural effusion and an Argyle 32-Fr tube in the pleural space. The white blood cell count in the pleural effusion increased from 125 to more than 6,000/mm³ within 6 hours. In this model, the pleural fluid protein level increased from 44 to 638 IU/L within 24 hours. There is one report of three patients (all with quadriplegia) who had developed lung entrapment as a result of prolonged chest tube drainage. They could not be weaned from the ventilator after a decortication was performed.⁵⁹

Minor complications include a subcutaneous hematoma or seroma, anxiety, shortness of breath (dyspnea), and cough (after removing large volume of fluid).

Subcutaneous emphysema indicates backpressure created by a clogged drain or insufficient negative pressure.

Overview of clinical trials of pigtail catheters:

Liang et al ⁶³ conducted a retrospective review of adult patients (≥ 18 years) who underwent ultrasound-guided pigtail catheter drainage of pleural effusions in the

ICUs from January 2005 to July 2007 among the 133 enrolled patients, there were 93 (70%) males and 40 (30%) females, with a mean age of 63.7 +/- 15.4 years old. The reasons for pigtail drainage were as follows: thoracic empyema (n = 59, 44%), massive transudative pleural effusions (n = 33, 25%), postoperative pleural effusion (n = 29, 15%), malignant pleural effusion (n = 18, 14%) and traumatic hemothorax (n = 3, 2%). In comparing the total amount of fluids drained, the duration of drainage, success rate and complication rate among these different causes of pleural effusion, pigtail drainage for massive transudative pleural effusion yielded the largest amount of pleural fluids (5,382 +/- 4,844 ml), provided the longest duration of drain (9 +/- 7 days), and had the highest complication rate (18%). The success rate was highest when used to treat traumatic hemothorax (100%) and postoperative pleural effusions (85%); drains inserted for empyema were more likely to fail (overall success rate, 42%). No significant insertion complications, such as hollow organ perforation, were caused by this procedure and they concluded that the ultrasound-guided pigtail catheter inserted by intensivists is a well-tolerated and effective method of draining all kinds of pleural effusions in critically ill patients.

Gammie et al ⁷³ studied pigtail catheter for pleural drainage and retrospectively reviewed 109 pigtail catheter placements without radiographic guidance and fifty one of 109 patients (46%) were mechanically ventilated and 26 patients (24%) had a coagulopathy and there were no complications related to pig tail catheter insertion seventy seven pigtail catheters were placed for pleural effusion and 32 for pneumothorax and mean effusion volume decreased from 43 to 9 percent and drained averaged 2889 ml over 97 hours and clinical success rates in effusion and pneumothorax groups were 86 and 81 percent and concluded pigtail catheters offers reliable treatment of pneumothoraces and simple effusion and is safe and less invasive

alternative to tube thoracostomy.

Sachin et al^{60,61,62,64,68,69,70,71} studied the efficacy and complications of percutaneous small bore pigtail catheters for tube thoracostomy and 50 cases of pleural effusion requiring drainage were enrolled in the study. Pigtail catheters of the size 9 to 12 F were used and found that the procedure was successful in 46/50 (92%) cases. Fibrinolytic therapy and pleurodesis was successful through these tubes. Complications included blockade in 4 (8%), small pneumothorax in 10 (20%) and chest pain at tube thoracostomy site requiring analgesics in 30 (60%) and they concluded Small bore pigtail catheters are safe, comfortable, cost effective and have few complications especially in loculated pleural effusions.

Lai et al⁴² studied Pigtail drainage in the treatment of tuberculous pleural effusions(a randomised study) and Sixty one patients with tuberculous pleurisy were divided into two groups; 30 patients received pigtail drainage combined with antituberculosis (TB) drug treatment and 31 received only anti-TB drugs. Outcomes were assessed for a period of 24 weeks after treatment and included symptom scores and the incidence of residual pleural thickening (RPT)and found the duration of dyspnoea was significantly shortened by the use of pigtail drainage(median 4 days (IQR 4–5) v 8 days (IQR 7–16), $p < 0.001$), a comparison of combined mean (SD)visual analogue scale (VAS) scores showed no significant difference between the groups after one week of treatment (57.1 (33.2) v 68.5 (44.7) or at any time during the follow up period and the incidence of RPT of more than 10 mm in the group treated with pigtail drainage and anti-TB drugs was 26% compared with 28% in the group receiving drug treatment only. The incidence of RPT levels of more than 2 mm in the two groups was 50% and 51%, respectively. No statistical difference between the twogroups in terms of forced vital capacity was found at the end of treatment (median

(IQR) 85.5% (69–94) of predicted v 88% (78–96) of predicted) and they concluded that addition of pigtail drainage to an effective anti-TB regimen is not clinically relevant and does not reduce the level of RPT.

Saffran et al ⁶⁵ studied outpatient Pleurodesis of Malignant Pleural Effusions Using a Small-Bore Pigtail Catheter and 14F pigtail catheter was percutaneously inserted into the pleural space and connected to a closed gravity-drainage bag system. The patients were instructed in the use of the drainage system and discharged to return for sclerosis with 4 g of talc after the drainage was < 100 mL/24 h. Patients were graded for dyspnea and performances status and baseline and transitional dyspnea index score (BDI-TDI) before tube placement and again at 30 days and found Ten ambulatory women, ages 41 to 79 years, were enrolled. The chest tube was left in place from 1 to 10 days, draining a mean of 2,956 mL (1,685 to 6,050 mL). Only two patients were unable to undergo sclerosis owing to catheter dislodgment and minimal drainage. Six reported symptomatic improvement at 30 days confirmed by TDI and ECOG scores in four of six. One with a prior history of a lobectomy was found to have a chylous pleural effusion and experienced a hydropneumothorax, for which sclerosis was unsuccessful. One died in hospital on day 26 after sclerosis despite radiographic resolution. Of the four patients who had improved dyspnea and functional status by TDI and EGOG scores, radiographic response was complete in three and partial in one. Two of the six were not able to return for follow-up because of weakness but reported improvement by telephone inquiry and concluded that ambulatory sclerosis of malignant effusion using a small-bore catheter is a feasible alternative to inpatient sclerosis with a large-bore chest tube, especially in patients with strong preferences for outpatient care.

Chung et al ⁶⁶ studied the role of early effective drainage in loculated

tuberculous (TB) pleurisy and consecutive patients with TB pleurisy subjected to anti-TB treatment and pigtail drainage (n = 64) were divided into 3 groups: 1) patients with free-flowing effusions irrigated with saline (free-flowing group; n = 20); 2) patients with loculated effusions irrigated with streptokinase (streptokinase group; n = 22); and 3) patients with loculated effusions irrigated with saline (saline group; n = 22). Pleural irrigation was performed for 3 days consecutively and the effusion drained as completely as possible.

Outcomes were assessed for 12 months by clinical symptoms, effusion removed, radiological scores for effusion amount, lung function and occurrence of residual pleural thickening and total effusion volumes removed were significantly greater in the free-flowing (2.36+/-1.62 L) and streptokinase groups (2.59+/-1.77 L) than in the saline group (1.28+/-1.21 L). Compared with the saline group, the free-flowing and streptokinase groups showed significant improvement in radiological scores and forced vital capacity at different time-points during follow-up, and a significantly lower occurrence of residual pleural thickening.. In summary, early effective drainage and complete anti-tuberculosis treatment may hasten clearance of pleural effusion, RPT occurrence and accelerate pulmonary function recovery in patients with symptomatic loculated tuberculous pleurisy.

Park et al ⁶⁷ studied the efficacy of pigtail Catheter drainage in patients with thoracic empyema or complicated parapneumonic effusion and 45 patients with complicated parapneumonic effusions or empyema were treated at Gil medical center from January 1998 to June 1999 and male gender was more frequent (42 men vs. 3 women), the mean age of the study population was 52(range:21~74) years. Empyema was found in 23 patients, complicated parapneumonic effusion in 22 patients. Four patients(three, parapneumonic effusion and one, with empyema) with

PCD only treated, were cast off and 36(80%) patients were treated with PCD only or PCD with urokinase. Among the 23 patients with empyema, surgical approach was required in five patients(27.1%, one required decortication, four open thoracostomy), one patient, treated with surgical procedure, died of sepsis and no significant difference of the duration of catheter insertion, the duration of hospital admission and the mean amount of effusion drained for initial 24 hours between the patients with only PCD treated and the patients treated with PCD and urokinase. The duration of catheter insertion and the duration of hospital admission after catheter insertion of the patients with only PCD treated were more longer than those of the patients treated with surgical procedure after PCD. They were same between the patients treated with urokinase after PCD and the patients treated with surgical procedure after PCD(11.1 ± 7.35 days vs. 19.2 ± 9.42 days, $p<0.05$, 17.5 ± 9.17 days vs. 38.6 ± 11.46 days, $p<0.01$). In 16 patients(44.4%) with only PCD treated or PCD and urokinase treated, the amount of effusion at the time of catheter removal was decreased more than 75% and in 17 patients(47.2%) effusion decreased 50~75%. .In one patient effusion decreased 25~50%, in two patients effusion decreased less than 25%. One month after catheter removal, in 35 patients(97.2%, four patients were cast off), the amount of pleural effusion was successfully decreased more than 50%. There were no complications related to pigtail catheter insertion.

Westcott et al ⁶⁸ studied-guided percutaneous catheter drainage (PCD) was used in 38 patients to diagnose and/or treat pleural effusions (21), hemothorax (one), symptomatic malignant effusions (four), and empyemas (12). Fluoroscopic guidance combined with the use of a small needle, J-tip guide wire, and pigtail catheter made it possible to safely drain both free and loculated fluid collections, including areas with difficult access. In 11 of the 12 patients with empyema, the pus (or purulent fluid) was

successfully drained using PCD. Two or more catheters were used in all but one patient in four of the empyemas, PCD was used successfully after incomplete or unsuccessful chest tube drainage. In five, PCD was used as the sole means of drainage. In three patients, chest-tube drainage was used to drain residual (one) or recurrent (two) empyemas after PCD. The safety and relative ease of PCD makes it an attractive alternative method for closed drainage of pleural fluid collections.(68)

Roberts et al ⁶⁹ described the efficacy of percutaneous pigtail catheters in evacuating pleural air or fluid in pediatric patients and designed a case series of children with percutaneous pigtail catheters placed in the pediatric ICU between January 1996 and August 1997 and Ninety-one children required 133 chest catheters. One hundred thirteen of the catheters (85%) were placed for pleural effusion, with 20 tubes (15%) placed for pneumothorax. Efficacy of drainage of pleural fluid was significantly greater in serous (96%) and chylous (100%) effusions compared with empyema (0%) or hemothorax (81%). Evacuation of pneumothorax was achieved by a pigtail catheter in 75% of patients. Complications due to placement of the pigtail catheters included hemothorax (n=3, 2%), pneumothorax (n=3, 2%), and hepatic perforation (n=1, 1%). There were also complications arising from the use of the catheters, including failure to drain, dislodgment, kinking, loss of liquid ventilation fluid, empyema, and disconnection in 27 of 133 catheters (20%) and concluded that Percutaneous pigtail catheters are highly effective in drainage of pleural serous and chylous effusions, somewhat less efficacious in drainage of hemothorax or pneumothorax, and least efficacious in drainage of empyema.

Reviewing the above literature small bore pigtail catheters are safe, efficacious, comfortable, cost effective and have minor complications.

Large bore thoracostomy tubes are commonly used for draining of pleural

fluid and is introduced by blunt dissection or by trocar assistance and have significant morbidity and complications.

Pig tail catheters are less traumatic and more comfortable with few complications and efficacy as good as large bore tubes.

METHODOLOGY

The study was done at KLES Dr Prabhakar Kore Hospital, which is a tertiary care hospital.

Source of data

Proved cases of tubercular pleural effusion at K.L.E'S Prabhakar Kore Hospital and MRC, Belgaum.

INCLUSION CRITERIA:

Patients above 18 yrs diagnosed as a case of tubercular pleural effusion at KLES Prabhakar Kore Hospital, Belgaum

EXCLUSION CRITERIA:

- 1) Patients with haemothorax, empyema.
- 2) Patient with para pneumonic effusion.
- 3) Bleeding and clotting disorders.
- 4) Thrombocytopenia.
- 5) Patients with pleural effusion secondary to ccf.

The following investigations were carried in all patients

1. COMPLETE BLOOD COUNT, BT, CT, PT.
2. CHEST X-RAY
3. BLOOD UREA
4. SERUM CREATININE

Detailed history was taken, respiratory system was examined. All the basic investigations were done in all patients as per the protocol.

Chest radiograph of posterior-anterior view were done in all the cases. CT scan of thorax was done wherever required.

The diagnosis of tubercular pleural effusion was done by the combination of various diagnostic modalities like sputum for AFB and

Pleural fluid analysis for:

Proteins;

Sugars;

Total count;

Differential count;

ADA level estimation;

PCR for MTB DNA;

Pleural fluid for AFB staining;

Procedure of pigtail catheter insertion:

Prior to commencing pigtail catheter insertion the procedure is explained fully to the patient and consent recorded. The best site to insert the catheter is the "safe triangle" . This is the triangle bordered by the anterior border of the latissimus dorsi, the lateral border of the pectoralis major muscle, a line superior to the horizontal level of the nipple, and an apex below the axilla.after identifying the site of maximum

dullness the part is infiltrated with a local anesthetic like 2% lignocaine under aseptic precautions. In contrast to large bore catheters, the pigtail catheter is inserted with the aid of a guidewire by a **Seldinger technique**. Blunt dissection is unnecessary as dilators are used in the insertion process. After infiltration with local anaesthesia, a needle and syringe are used to localise the position for insertion of pigtail catheter (point of maximum dullness). A guidewire is then passed down the hub of the needle, the needle is removed, and the tract enlarged using a dilator. A small bore pigtail catheter is then passed into the thoracic cavity along the wire. It is held securely to the chest wall with the help of purse string sutures. A three way stop cork is then connected to the free end of the pigtail catheter and the three way is further attached to an ICD bottle or a Romodrain bag with fluid level to create a closed system which prevents the development of iatrogenic pneumothorax.

All patients received anti tubercular treatment (2HRZE, 4HR).

- Pigtail catheters of size 7-9 F are used
- Pleural fluid will be aspirated thrice daily with a 20 ml syringe. The catheter will be removed as soon as the drainage fluid will be less than 50 ml for 3 consecutive days
- Post removal of tube, chest X-ray had been taken and opacity of lung fluids are conformed
- As all the patients are not affordable for ultrasound imaging and is the need for selecting chest x-ray for quantification of pleural effusion

RADIOLOGICAL QUANTIFICATION OF PLEURAL EFFUSION:

- 1) MILD- obliteration of costophrenic angle.
- 2) MODERATE- pleural effusion up to anterior end of 4th rib
- 3) MASSIVE- more than moderate effusion.

RADIOLOGICAL CRITERIA TO SAY WHETHER OPACITY IS DUE TO LUNG OR PLEURAL EFFUSION:

- free fluid in pleural cavity presents as a homogenous opacity with a concave upper border higher laterally than medially-so called PLEURAL MENISCUS.
- meniscus sign is absent in opacities arising from lung such as consolidation and mass

The following outcome variables were studied:

1. Total amount of pleural fluid drained.
2. Total duration of hospitalization.
3. Level of radiological resolution
4. Residual pleural thickening as assessed by chest radiography.
5. Complications like pneumothorax, chest pain, hydropneumothorax, pyothorax, tube blockage, infection of wound were recorded.

RESULTS

A total of 40 patients in tertiary care hospital over a period 1 year between November 2008 and November 2009 were included in the study and all patients are followed up over a period of six months.

GENDER WISE DISTRIBUTION

TABLE 4:

GENDER	TOTAL NO. OF PATIENTS (n=40)	PERCENTAGE
MALE	27	67.5
FEMALE	13	32.5

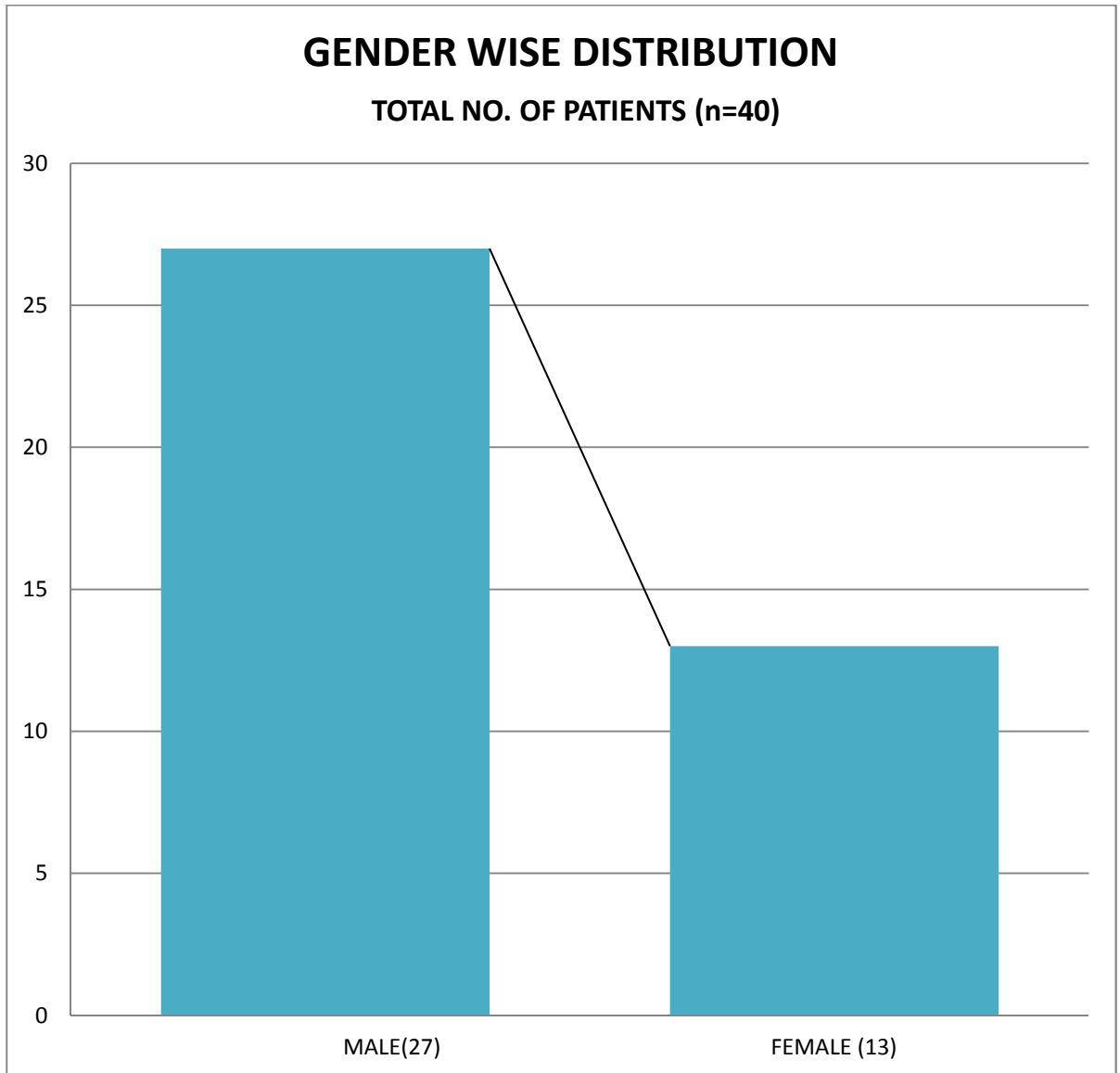
Total number of male patients were - 27 (67.5%)

Total number of female patients were - 13 (32.5%)

Male to female ratio - 2:1

GENDER WISE DISTRIBUTION

GRAPH 1:



AGE WISE DISTRIBUTION OF PATIENTS
TABLE 5:

AGE GROUP	TOTAL (n=40)	PERCENTAGE
< 20 YEARS	3	7.5
21-30 YEARS	10	25
31-40 YEARS	4	10
41-50 YEARS	11	27.5
>51 YEARS	12	30

Patients less than 20 years - 3 (7.5%)

Patients between 21-30 years - 10 (25%)

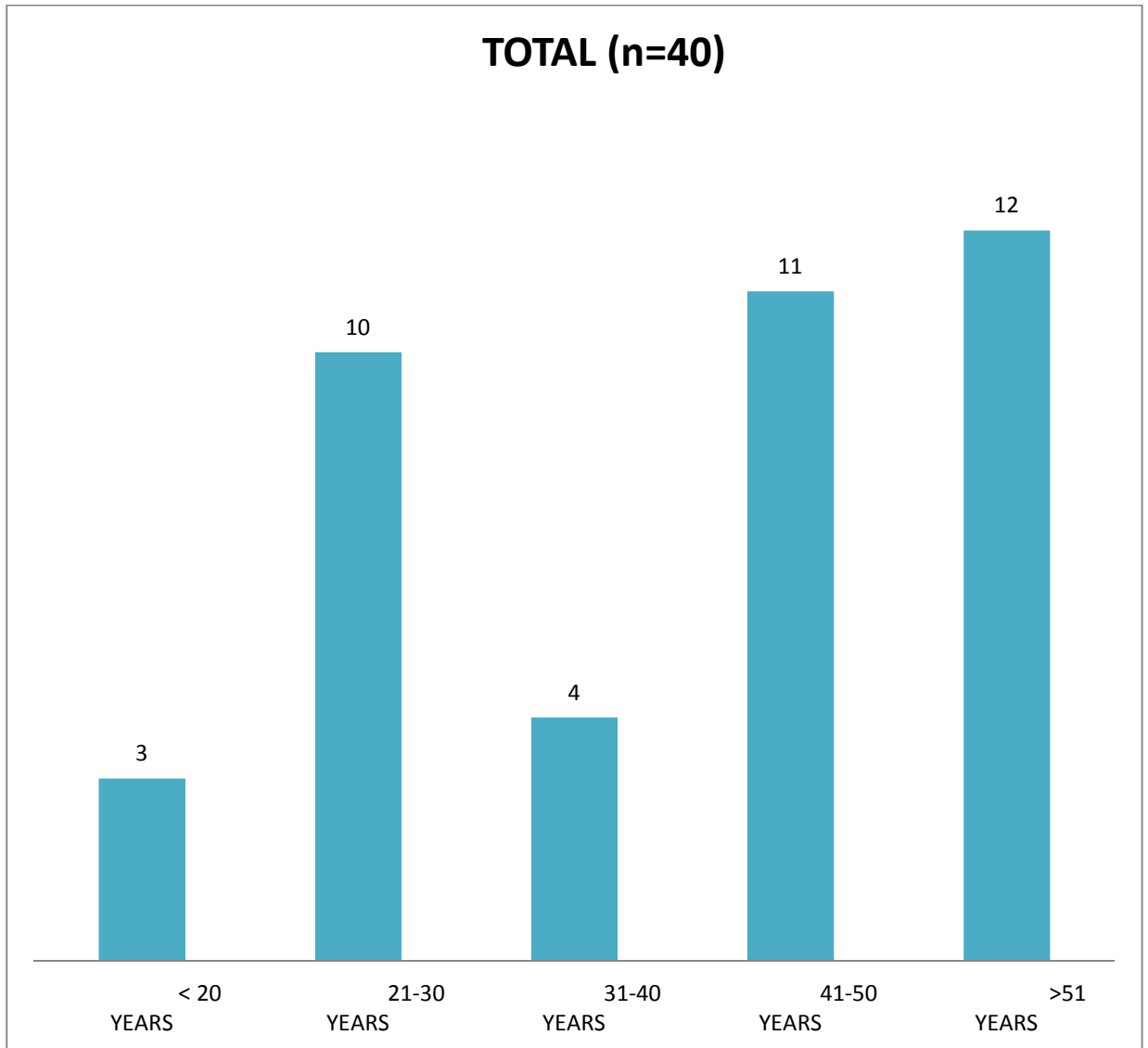
Patients between 31-40 years - 4 (10%)

Patients between 41-50 years - 11 (27.5%)

Patients more than 51 years - 12 (30%)

AGE WISE DISTRIBUTION OF PATIENTS

GRAPH 2:



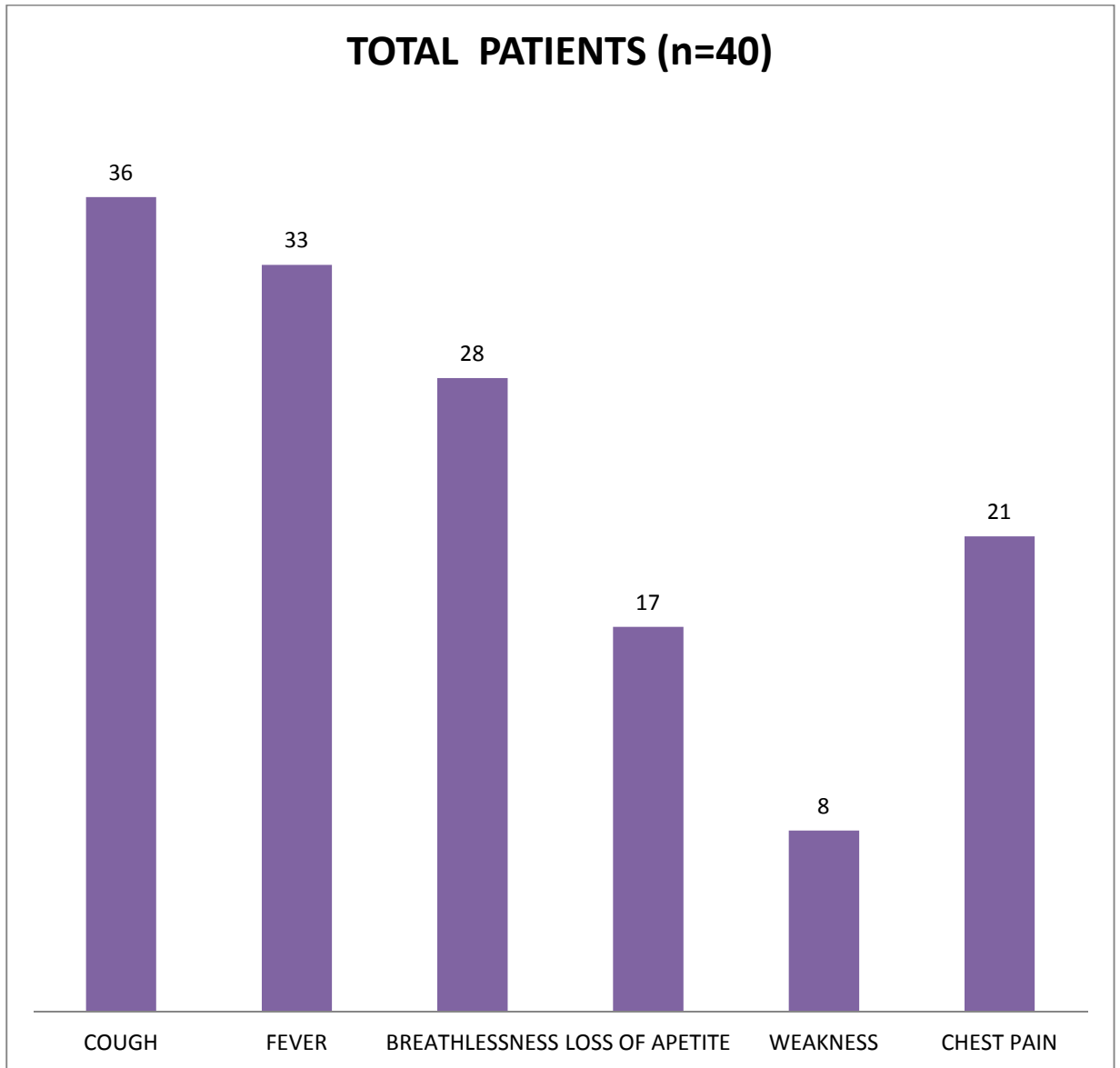
MAJOR SYMPTOMS**TABLE 6:**

SYMPTOMS	TOTAL PATIENTS (n=40)	PERCENTAGE
COUGH	36	90
FEVER	33	82
BREATHLESSNESS	28	70
LOSS OF APETITE	17	42
WEAKNESS	8	20
CHEST PAIN	21	52

The major symptoms of the patients were cough(90%), fever (82%), breathlessness (70%), chest pain (52%).and loss of appetite(42%)

MAJOR SYMPTOMS

GRAPH 3:



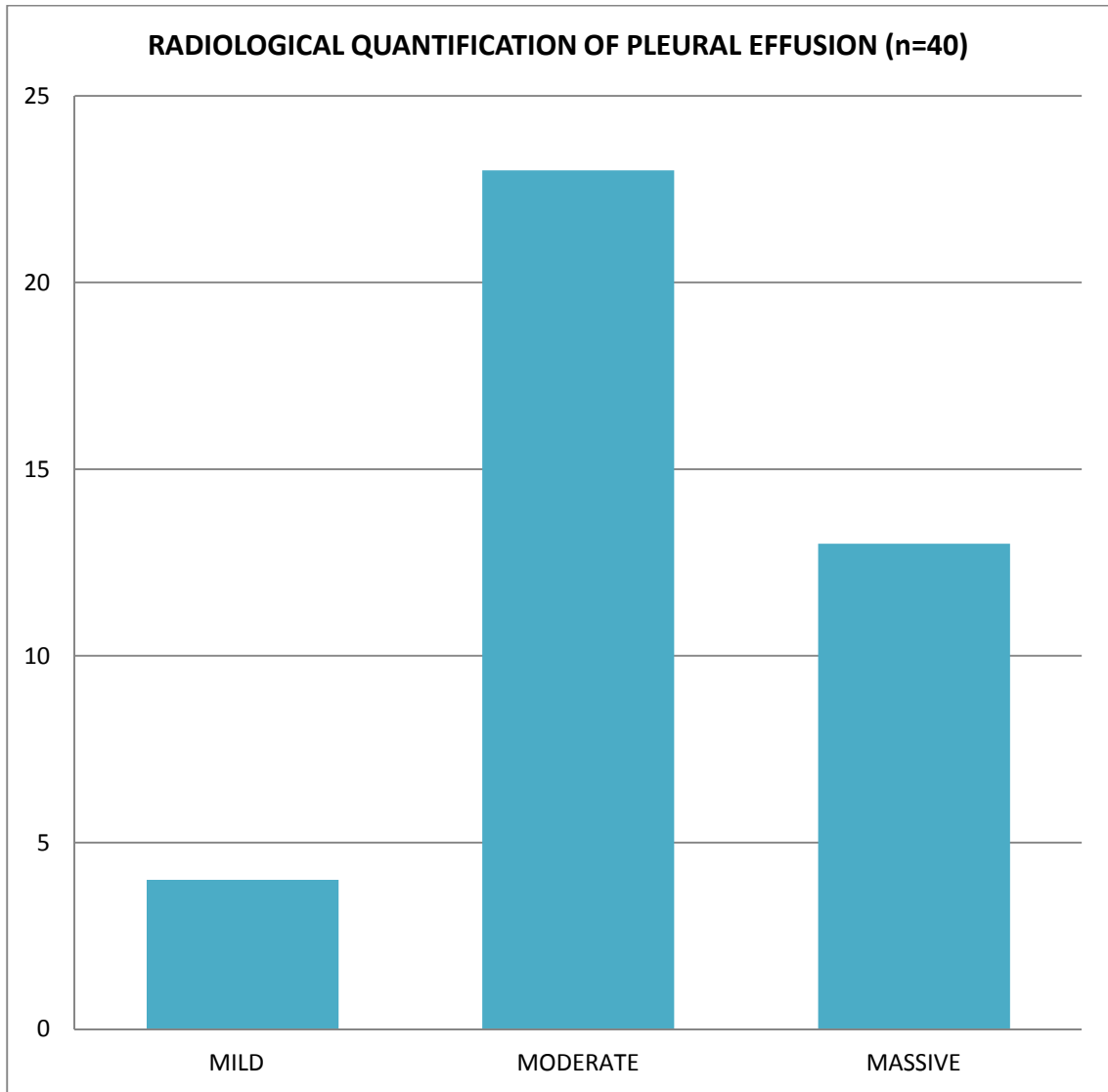
RADIOLOGICAL QUANTIFICATION OF PLEURAL EFFUSION**TABLE 7:**

QUANTIFICATION	TOTAL PATIENTS (n=40)	PERCENTAGE
MILD	4	10
MODERATE	23	57.5
MASSIVE	13	32.5

Most of the patients were with moderate (57.5%) and massive (32.5%) degree pleural effusion and only 10 % of patients were with mild degree pleural effusion.

RADIOLOGICAL QUANTIFICATION OF PLEURAL EFFUSION

GRAPH 4:



PLEURAL FLUID ANALYSIS OF ADA LEVELS (IU)

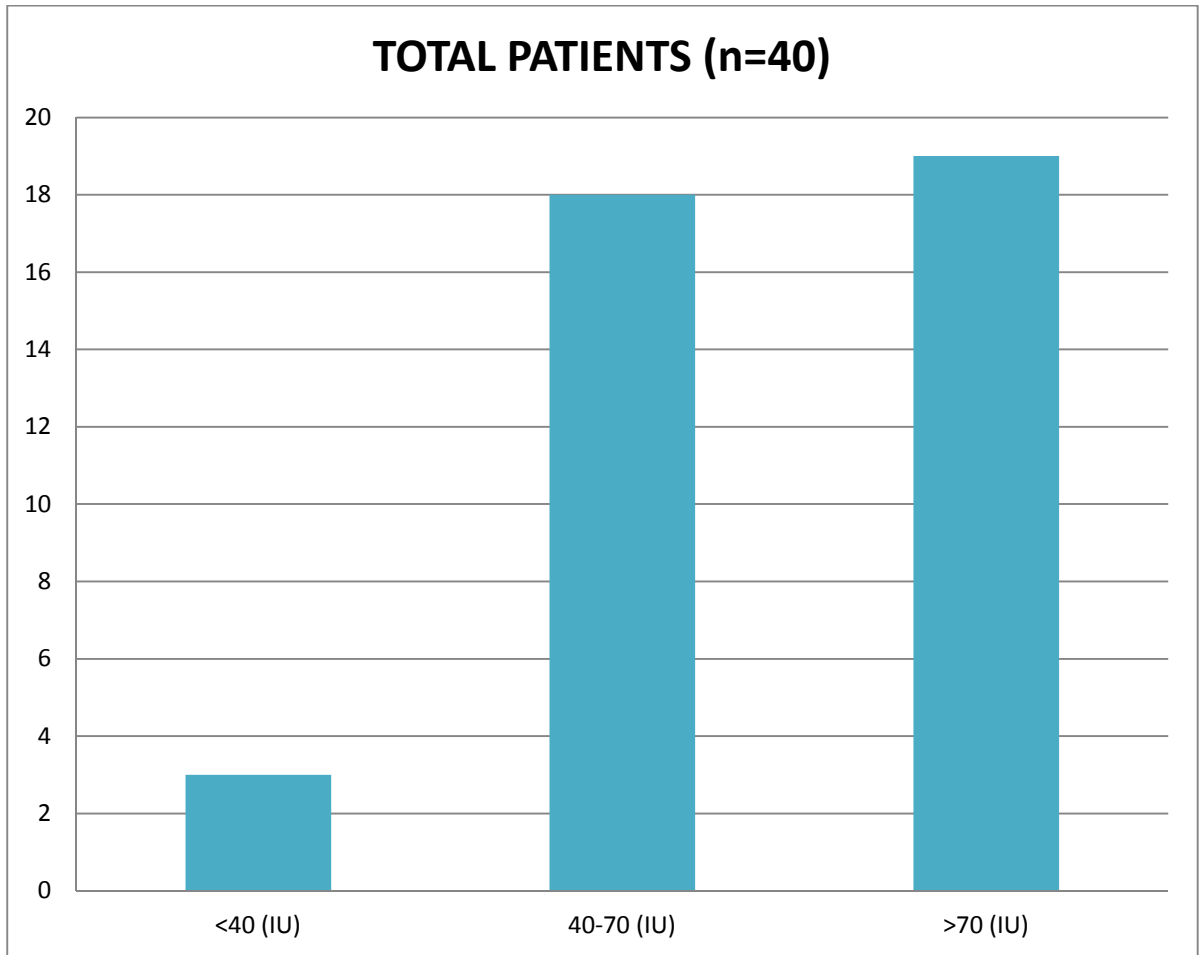
TABLE 8:

ADA levels	TOTAL PATIENTS (n=40)
< 40 IU	3
40-70 IU	18
>70 IU	19

Out of 40 patients,19 patients are having ADA levels >70 IU,18 patients are between 40-70 IU and 3 patients have < 40 IU.

PLEURAL FLUID ANALYSIS OF ADA LEVELS (IU)

GRAPH 5:



TOTAL AMOUNT OF DRAINAGE**TABLE : 9**

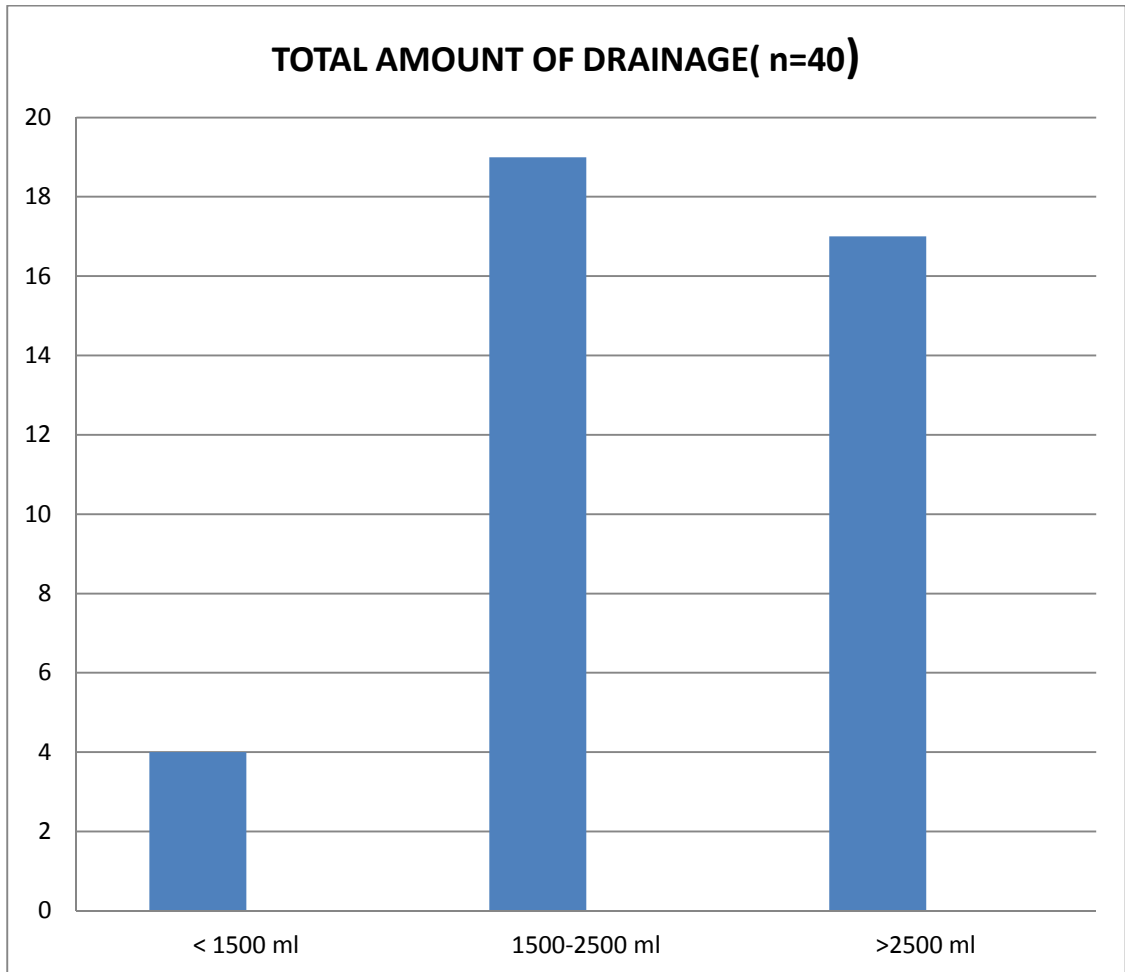
AMOUNT OF DRAIN (ml)	TOTAL PATIENTS (n=40)	PERCENTAGE
<1500	4	10
1500-2500	19	47.5
>2500	17	42.5

Out of 40 patients, < 1500 ml tubercular pleural fluid was drained in 4 patients (10%), 1500-2500ml in 19 patients (47.5%), > 2500ml in 17 patients (42.5%).

Mean amount of pleural fluid - 2575 ml

TOTAL AMOUNT OF DRAINAGE

GRAPH : 6



TOTAL DURATION OF PIG TAIL CATHETER**TABLE : 10**

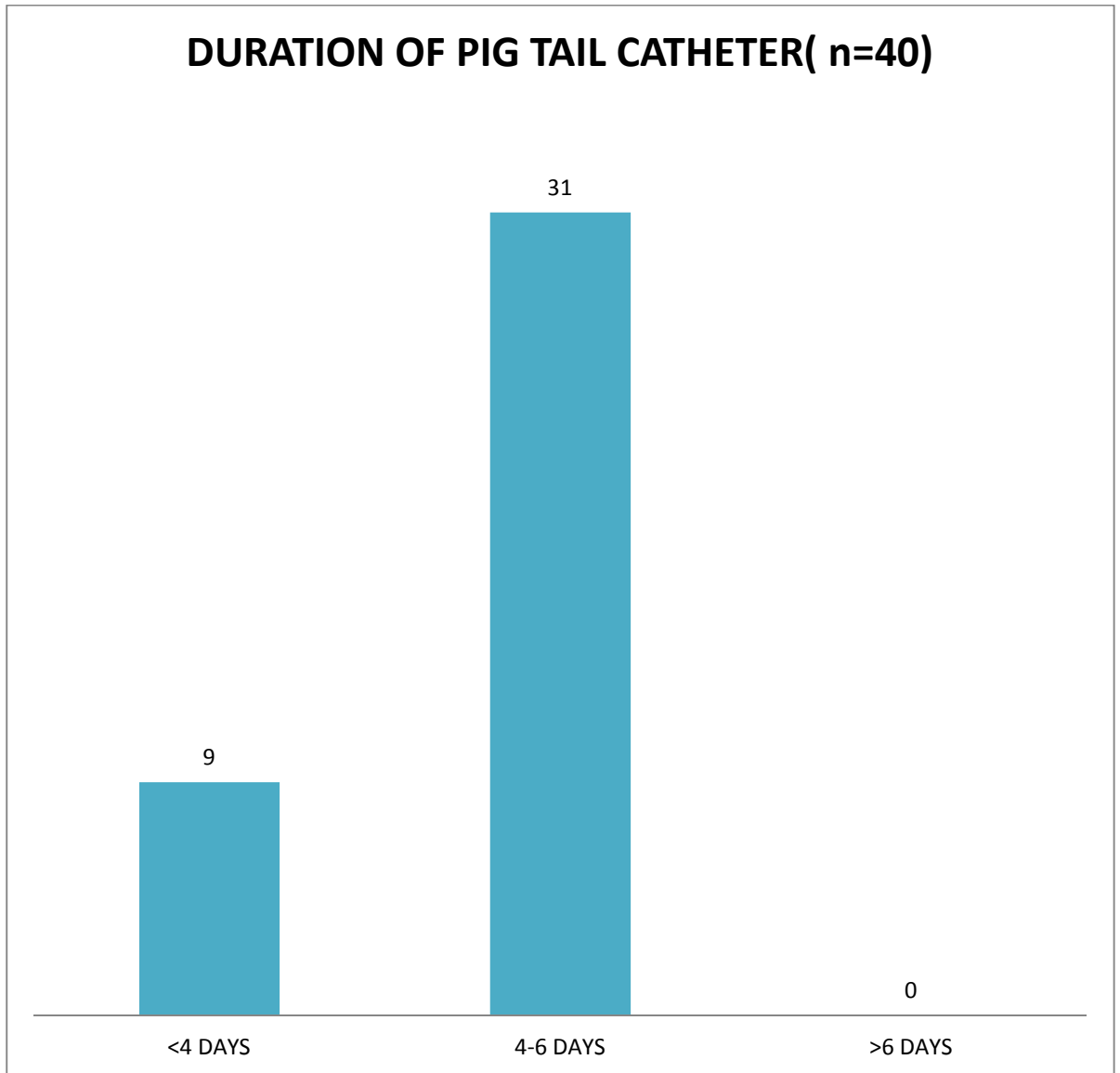
DURATION	TOTAL PATIENTS (n=40)	PERCENTAGE
<4 DAYS	9	22.5
4-6 DAYS	31	77.5
>6DAYS	0	0

Out of 40 patients,pig tail catheter was removed in 9 patients with a duration of < 4 days,31 patients with a duration of 4-6 days and no patients with the catheter for more than 6 days.

Mean duration of pigtail catheter - 4.5 days

TOTAL DURATION OF PIG TAIL CATHETER

GRAPH : 7



TOTAL DURATION OF HOSPITALIZATION
TABLE : 11

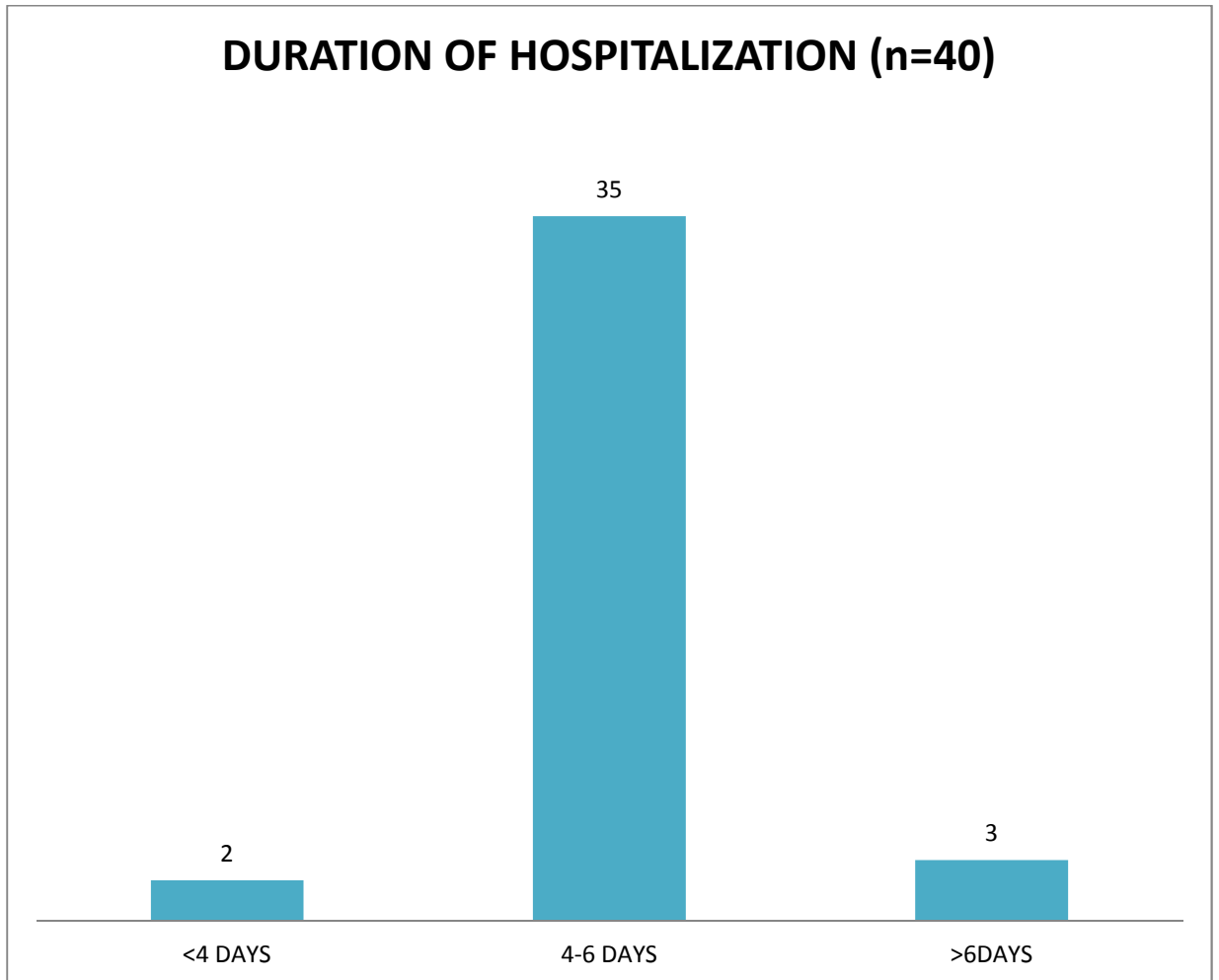
DURATION	TOTAL PATIENTS (n=40)	PERCENTAGE
<4 DAYS	2	5
4-6 DAYS	35	87.5
>6 DAYS	3	7.5

Out of 40 patients, 2 (5%) patients were discharged with a duration of < 4 days, 35 (87.5 %) between 4-6 days and 3 (7.5%) patients > 6 days. The associated symptoms like generalized weakness, intolerance of ATT in the form of gastritis, hepatitis and hypersensitivity which delayed the discharge of the patient.

Mean duration of hospitalization- 6 days

TOTAL DURATION OF HOSPITALIZATION

GRAPH : 8



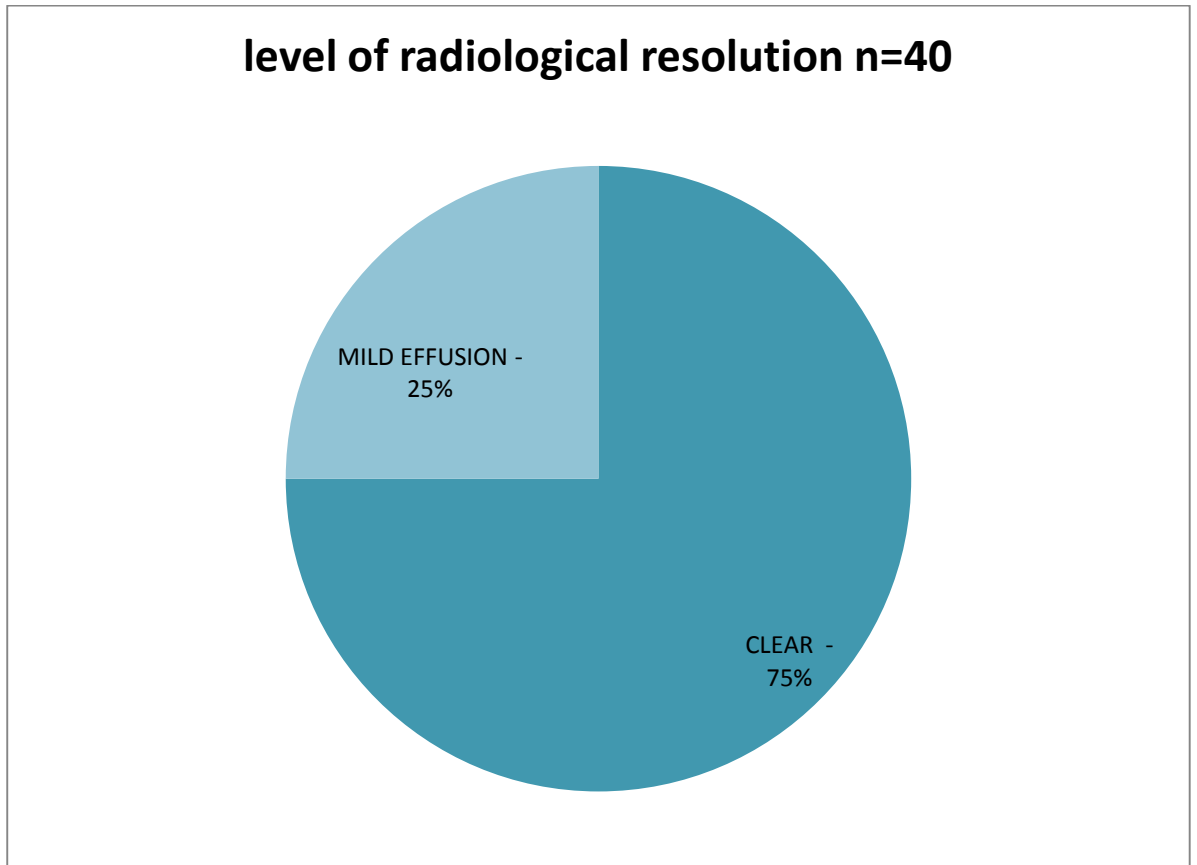
RADIOLOGICAL RESOLUTION AT TIME OF DISCHARGE**TABLE : 12**

RADIOLOGICAL RESOLUTION	TOTAL PATIENTS (n=40)	PERCENTAGE
CLEAR	30	75
MILD EFFUSION	10	25

Out of 40 patients,30 (75%) had clear chest radiograph and 10 (25%) had mild pleural effusion at time of discharge.

RADIOLOGICAL RESOLUTION AT TIME OF DISCHARGE

GRAPH: 9



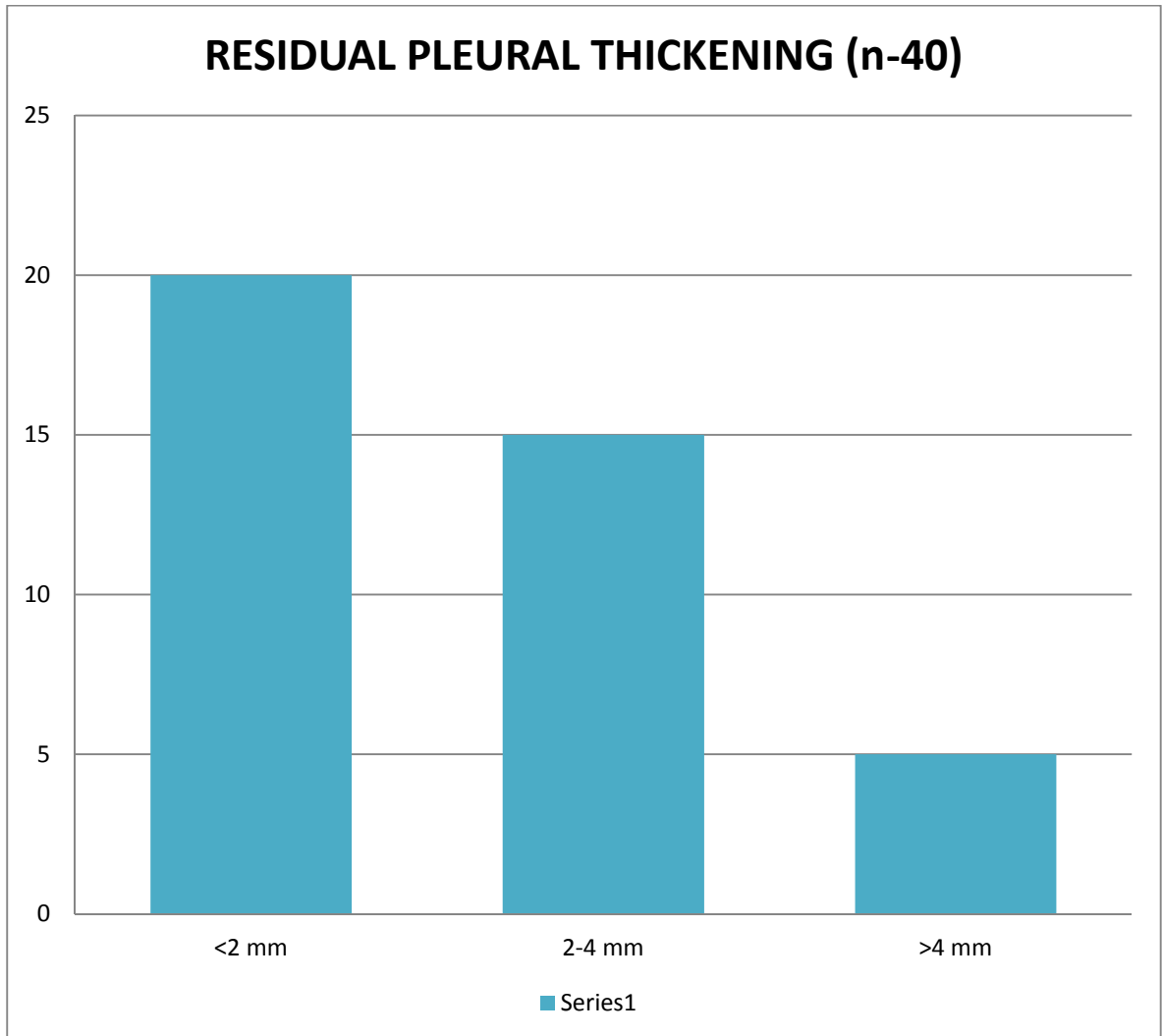
RESIDUAL PLEURAL THICKENING ASSESSED BY CHEST X-RAY**TABLE : 13**

RPT (mm)	TOTAL PATIENTS (n=40)	PERCENTAGE
<2mm	20	50
2-4 mm	15	37.5
>4mm	5	12.5

It was observed that 20 (50%) patients had residual pleural thickening <2mm, 15(37.5%) patients between 2-4 mm and 5(12.5%) patients had > 6mm.

RESIDUAL PLEURAL THICKENING ASSESSED BY CHEST X-RAY

GRAPH :10



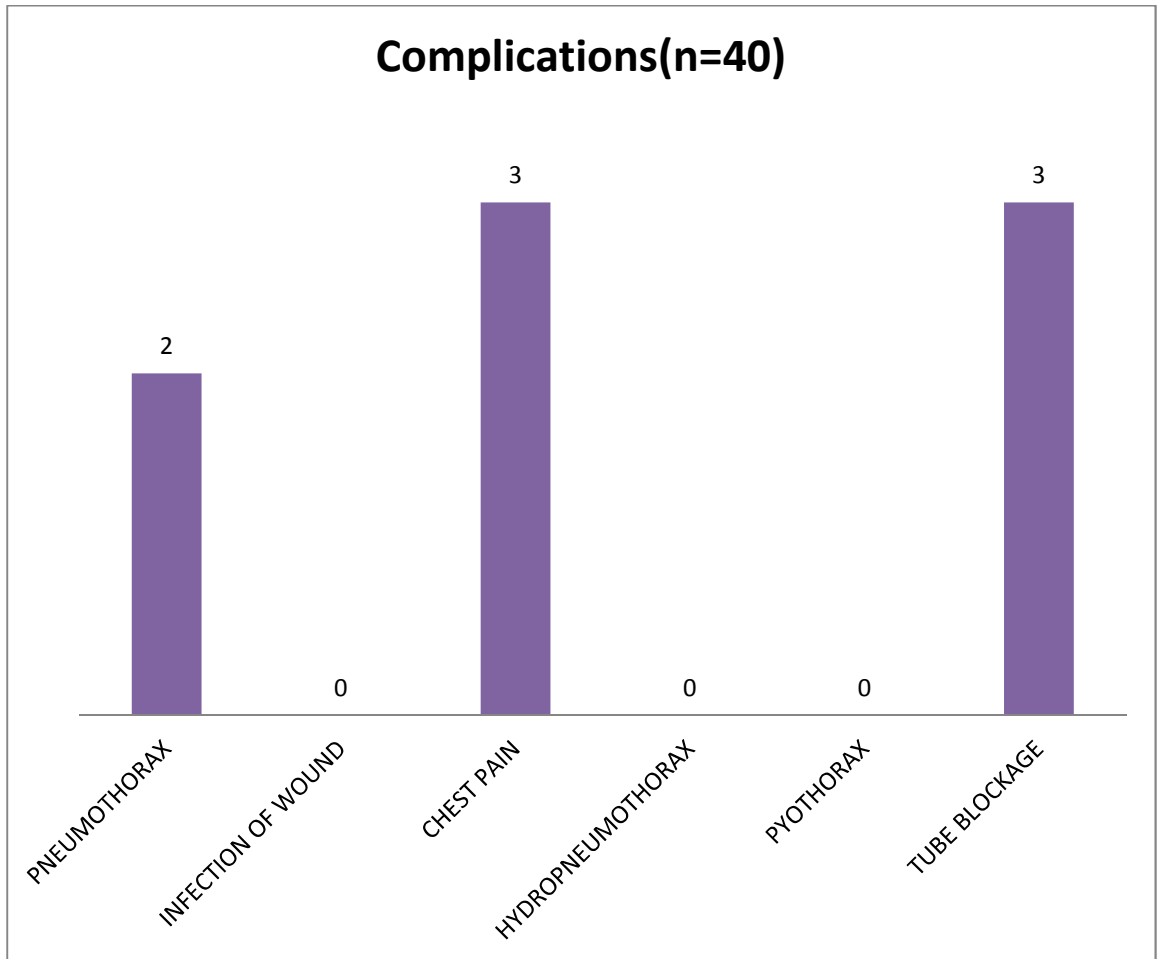
COMPLICATIONS**TABLE : 14**

COMPLICATIONS	TOTAL PATIENTS (n=40)	PERCENTAGE
PNEUMOTHORAX	2	5
INFECTION OF WOUND	0	0
CHEST PAIN	3	7.5
HYDROPNEUMOTHORAX	0	0
PYOTHORAX	0	0
TUBE BLOCKAGE	3	7.5
TOTAL	8	20

Complications like pneumothorax was seen in 2 patients and this was probably due to faulty technique, chest pain was seen in 3 patients and tube blockage was seen in 3 patients out of 40 patients. Complications like infection of wound, hydropneumothorax, pyothorax were not seen in these patients.

COMPLICATIONS

GRAPH : 11



DISCUSSION

The present study was done in a tertiary care KLES Dr Prabhakar Kore hospital and medical research center, Belgaum. A total of 40 patients were included in the study. Majority of the patients were in the age group above 51 years (30%) and overall (57.5)% of patients were above the age of 40 years. This is in contrast to study done by Ibrahim et al ⁷⁴, who observed that the mean age of 100 patients with tuberculous pleural effusion was 31.5 years. Our findings are in part consistent with the reports from the study of Baumann et al ⁷⁵ who studied 14,000 patients with tuberculous pleuritis reporting to the Communicable Disease Center in the United States between 1993 and 2003. In their study the mean age of the patients was 49.9 years. The difference in the age distribution of the present study with other studies might be due to a relatively small sample size.

In the present study, males to female ratio was 2 : 1 which was consistent with the study done by Chung et al⁶⁶ in which the bulk of the patients were males.

The major symptoms of the patients were cough (90%), fever (82%), dyspnea (70%), loss of appetite (42%), chest pain (52%) and generalized weakness (20%). These findings were consistent with the study done by Berger et al⁷⁶. They observed cough (70%), fever (68%) and chest pain to be the most common symptoms.

In our study most of the patients (90%) were with moderate and massive pleural effusion.

In our study, majority of the patients had an ADA level exceeding 40 IU/L which is in consistency with the studies done by Ocana et al ⁷⁷ . In their study of 221

patients with pleural or peritoneal effusions, all patients with a pleural fluid ADA of 70 IU/L had TB, whereas no patient with a pleural fluid ADA below 40 IU/L had tuberculous pleuritis.

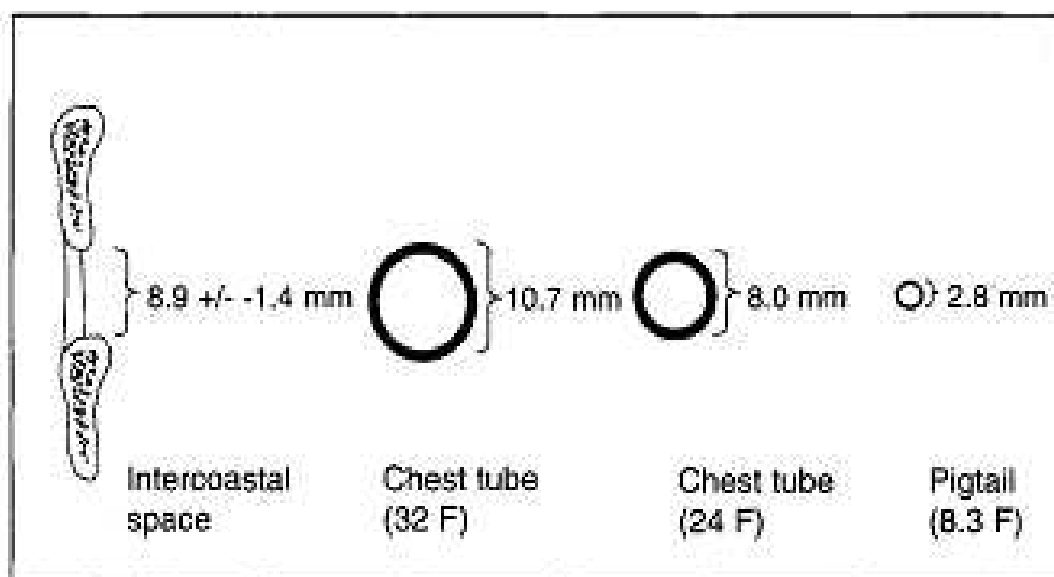
The main aim of the present study was to evaluate the early and effective drainage of tubercular pleural effusion via pigtail catheter and to evaluate the radiological response in the improvement of pleural effusion after pigtail catheter insertion. Large-bore thoracostomy tubes commonly used for draining of pleural fluid or air are introduced by blunt dissection or by trocar assistance and have significant morbidity and complications. Furham et al, Lawless et al and Vardhan et al^{60,61,62} used pigtail catheters in their studies and found them less traumatic and comfortable for patients with efficacy as good as large bore tubes and in our study we found it successful in 90% of our cases. Wescott JL et al⁶⁸ used small bore pigtail catheters for drainage of empyema in 12 cases and was successful in 11 cases and he found these tubes particularly useful for drainage of loculated effusions with difficult access

Our study Clinical success rates are comparable to previously reported rates of tube thoracostomy (90%)⁷⁸. Minor complications in our study include tube blockade, chest pain and small pneumothorax, tube blockade was flushed with normal saline and was successful in all 3 cases complications like haemothorax, rupture of diaphragm, liver, spleen and infection of wound seen with large bore tubes are not seen with pigtail catheter insertion⁶⁸. Morrison et al⁷⁹ found complications in the form of pneumothorax in 19% of cases while Chang et al⁸⁰ found the incidence to be 31%. In our study all pneumothorax were insignificant and attribute it to use of seldinger technique. Grodzin et al⁷⁰ used small bore catheter in 53 cases of pleural

effusion and found it to be safe, efficacious and cost effective with few complications which was comparable to our study.

Parulekar et al⁷¹ compared small bore and large bore tubes in malignant pleural effusion and they found there was no difference in outcome and small bore tubes were as effective as large bore tubes. In our present study we observed that mean amount of pleural fluid drained was 2575ml and mean total duration of pig tail catheter was 4.5 days and no patient was with pigtail catheter for more than 6 days and mean total duration of hospitalization was 6 days which were consistent in a study done by Chung et al⁶⁶.

In our study we used pig tail catheters of size 7-9 F. In a study done by James S Gammie et al⁷³ they said there is wide acceptance of the pigtail catheter for thoracic drainage on a variety of clinical services and they strongly believe that the pigtail catheter causes substantially less pain than traditional tube thoracostomy, by virtue of its size in relation to the normal intercostal space. The average intercostals space in an adult (measured at the 5th intercostal space in the mid-axillary line) is 8.8 ± 1.4 millimeters. A 24 F chest tube (the smallest size commonly used for the described indications) has an outer diameter of 8 mm, while a 32 F chest tube has an outer diameter of just 11 mm. Chest tubes, with their excessive size, cause pain by compressing the neurovascular bundle at the top of the interspace, as well as by levering open the interspace. In contrast, the 8.3 F pigtail catheter has a diameter of only 2.8 mm and does not impinge on the neurovascular bundle or alter the geometry of the inter-costal space. It has previously been shown that routine chest tube placement is frequently associated with an unacceptable pain level.⁸¹



(Figure-1) Relative sizes of the average intercostal distance in the adult(fifth inter costal space, mid-axillary line),two commonly used chest tube sizes (24 F and 32 F), and the 8.3 F pigtail catheter.

At the end of 6 months of anti tubercular treatment, we noted that the occurrence of residual pleural thickening in 50% of cases . this is in contrast to study done by Lai Y-F et al ⁴² that indicate RPT is clinically significant on initial presentation of tuberculous pleurisy, it may subside over time. Drainage of pleural effusions, even in the early stages does not prevent the development of RPT, but early diagnosis and early initiation of anti-TB drug treatment has been implicated in a decrease in the development of RPT in patients with tuberculous pleurisy.

We observed that radiological resolution was clear in 75% of cases during discharge.

CONCLUSION

In conclusion this study has shown early effective drainage and complete anti-tuberculosis treatment with pigtail catheter hasten clearance of pleural effusion and is safe, efficacious, comfortable, decreased hospital stay, cost-effective, minimal complications and improves patient quality of life and does not reduce residual pleural thickening occurrence.

SUMMARY

- The study was done in a tertiary care KLES Dr Prabhakar Kore hospital and medical research center, Belgaum.
- The study included 40 patients
- The majority were in the age group of above 51 (30%) and 60 % were above 35 years of age.
- The male: female ratio was 2:1.
- The major complaints of the patients were cough (90%), fever(82%), breathlessness(70%), chest pain (52%), loss of appetite (42%) and weakness (20%).
- Most of the patients were with moderate (57.5%) and massive (32.5%) tubercular pleural effusion(before procedure).
- Pleural fluid analysis of ADA levels of above 40 IU were 37 (92.5%) patients.
- In 36(90%) patients the total amount of pleural fluid drained with pigtail catheter was above 1500ml and mean drainage of pigtail catheter was 2575ml.
- In 40(100%) patients pitail catheter was removed before 6 days and mean duration of pigtail cathter was 4.5 days.No patient was with pigtail catheter for more than 6 days.
- 37(92.5%) patients were discharged from hospital before 6 days.
- Complete clearance of chest radiograph during discharge was seen in 30(75%) patients.
- Residual pleural thickening was seen in 20(50%) patients after six months of antitubercular treatment.

- Minimal complications like pneumothorax 2(5%),tube blockade 3(7.5%),and chest pain 3(7.5%) were observed.

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ANNEXURE - I

CONSENT FOR PARTICIPATION IN RESEARCH STUDY

Please help to serve you better

“EARLY AND EFFECTIVE DRAINAGE OF TUBERCULAR PLEURAL EFFUSIONS VIA PIGTAIL CATHETER IN KLES DR PRABHAKAR KORE HOSPITAL AND MRC – A DESCRIPTIVE STUDY OVER A PERIOD OF ONE YEAR”, conducted by **DR. K. SUNIL KUMAR REDDY.**, Post Graduate in M.S. General Surgery, J.N. Medical College, Belgaum under the guidance of Dr. M. S. Sangolli M.S Professor, Department of General Surgery, under KLE University, Belgaum.

Respected Sir / Madam we request you to please participate in our study as you are eligible for it. Your participation in this research is voluntary and your decision whether to participate or not in this study will in no way affect your relationship with J.N.M.C. If you decide to participate you are absolutely free to withdraw at any point of time.

Purpose :

During the study you will be asked to undergo Chest x-ray, Sputum analysis , pleural fluid analysis, which ever is advised in your case .

The purpose of research is to determine the efficacy Pigtail catheter to drain pleural effusion.

Procedure Involved :

If you agree to participate you will undergo invasive procedure under local anesthesia to drain pleural effusion. (using pigtail catheter) .

Risks and Benefits :

The procedure involves minimal risks and is less painful. (As compared to tube thorocostomy)

Alternatives :

Even if you decline the participation in the study, you will get the routine line of management.

Privacy and Confidentiality :

The only people to know that you are a research subject are members of the research team. No information about you or information provided by you during the research will be disclosed to other without your written permission except :

1. In emergency to protect your rights and welfare.
2. If required by law.

Authorization to Publish Results :

When the results of the research are published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with you will remain confidential.

Compensation :

In the event of injury or complication related to the study, treatment will be made available to you through the free wards of KLES Prabhakar Kore's Hospital & MRC, Belgaum. There is no compensation or payment for such medical treatment by law. In case of any complication or injury please do feel free to contact **DR. K. SUNIL KUMAR REDDY**. PG M.S. General Surgery, KLES Prabhakar Kore Hospital and MRC, Belgaum Phone No.9740638666

Questions :

In case you have any questions related to the study, you can contact **DR. K. SUNIL KUMAR REDDY**. on Mob : 9740638666

In case you have any question about your rights as a study participant, you can contact Dr. V. D. Patil (0831-2471350).

Consent for participation in research trial :

I, Mrs. _____ voluntarily agree for the participation as a subject of study. By signing this consent form I am not giving up any of my legal rights, I may withdraw from the study anytime. I am signing the consent form after having read or been read for me in vernacular language, including the risks and the benefits and having all my questions answered.

Subject Name : _____

Signature or the Left Thumb Print of Subject : _____

Witness Name : _____ Signature : _____

Investigators Name: _____ Signature : _____

Date : _____

Place : _____

PROFORMA

**“EARLY AND EFFECTIVE DRAINAGE OF TUBERCULAR PLEURAL EFFUSIONS
VIA PIGTAIL CATHETER IN KLES DR PRABHAKAR KORE HOSPITAL AND MRC
– A DESCRIPTIVE STUDY OVER A PERIOD OF ONE YEAR ”.**

Investigator : Dr. K. Sunil Kumar Reddy.

Guide : Dr. M. S. Sangolli M.S.

Name & Address of the patient : _____

Age of the Patient :

Years

IP. No.

Sex :

Occupation :

Date of Admission :

Date of Examination :

CHIEF COMPLAINTS

BRIEF HISTORY :

DIAGNOSIS :

BLOOD INVESTIGATIONS :

CHEST X-RAY Findings :

SPUTUM AFB FINDINGS :

PLEURAL FLUID ANALYSIS FINDINGS:

TOTAL DRAINAGE:

DURATION OF PIGTAIL CATHETER:

DURATION OF HOSPITALIZATION:

RADIOLOGICAL RESOLUTION DURING DISCHARGE:

COMPLICATIONS IF ANY:

RESIDUAL PLEURAL THICKENING (After 6 months of ATT) (mm):

PT NAME-SHRIPAD VERNEKAR

I.P NO-336957



ADMISSION



BEFORE DISCHARGE



COMPLETION OF ATT

PT NAME- NAGANNUR SHARDHA SIDDAPPA

I.P NO-325169



ADMISSION



BEFORE DISCHARGE



COMPLETION OF ATT

PT NAME-PRAKASH RANOJI

I.P NO-317190



ADMISSION

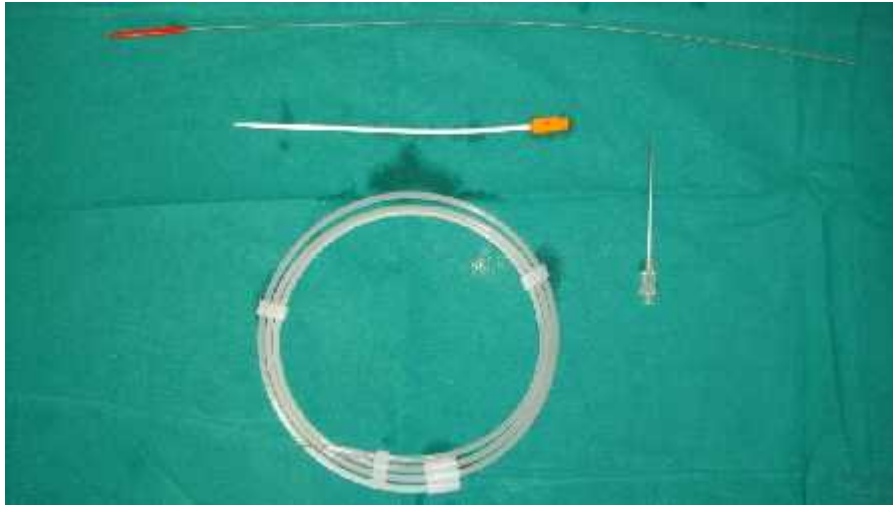


BEFORE DISCHARGE



COMPLETION OF ATT

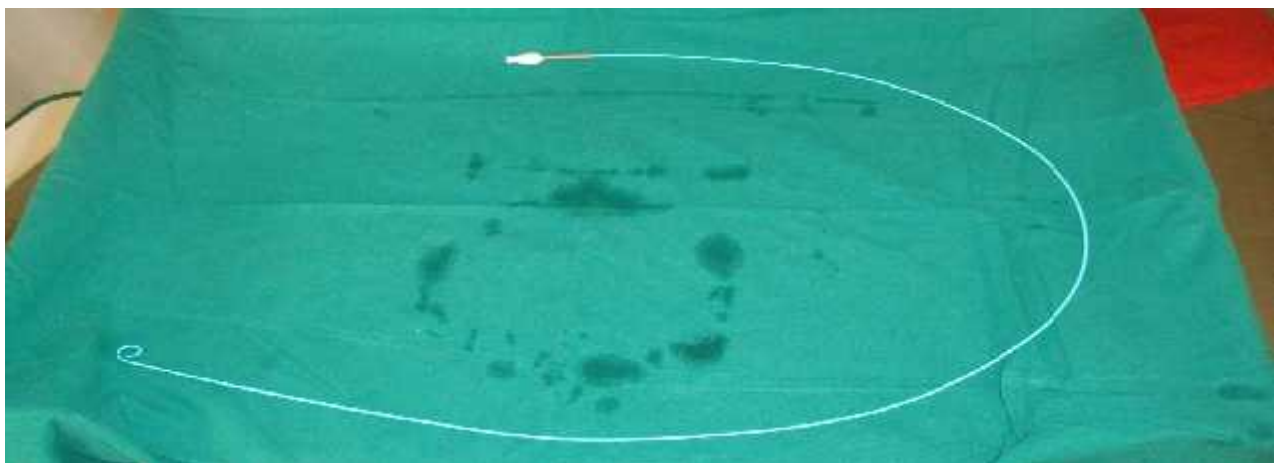
COMPLICATION--PNEUMOTHORAX



Materials required
Needle, guidewire and dilator



Pigtail catheter—7 F

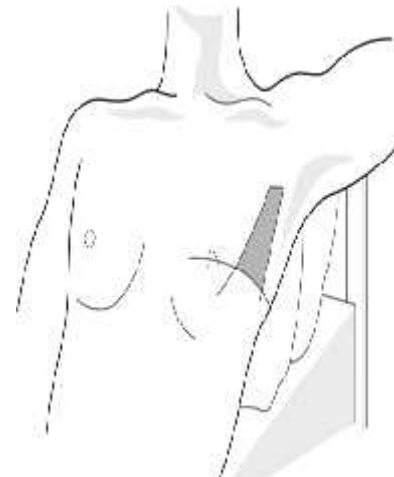




Romodrain with water sealed drainage system connected to 3 way connector.



Insertion of pigtail catheter at safe triangle





Infiltration of local anesthetic and needle



Insertion of guide wire through the needle



Tract enlarged using dilator

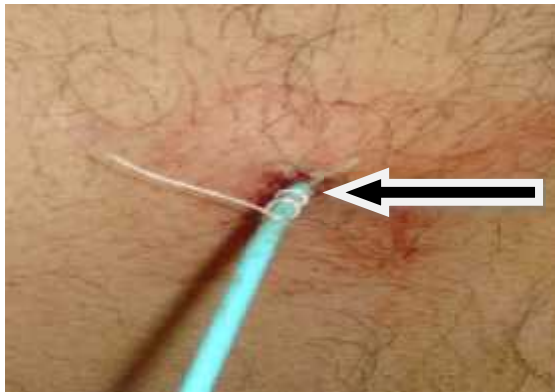


Insertion of pigtail along guide wire

Tubercular pleural fluid aspirating through pigtail catheter



Amount of trauma during pigtail catheter insertion



Freely flowing pleural fluid



ANNEXURE -4

KEY TO MASTER CHART

GENDER

- M - MALE
- F - FEMALE

MAJOR COMPLAINTS

- C - COUGH
- F v - FEVER
- B - BREATHLESSNESS
- L - LOSS OF APETITE
- P - CHEST PAIN
- W - WEAKNESS

QUANTIFICATION OF PLEURAL EFFUSION (Qty)

- MIL - MILD
- MOD - MODERATE
- MAS - MASSIVE
- PF anal of AFB sm - PLEURAL FLUID ANALYSIS OF AFB SMEAR
- PF anal of ADA - PLEURAL FLUID ANALYSIS OF ADA LEVELS
- T D (ml) - TOTAL DRAINAGE IN MILLILITRES

Dur PTC	-	DURATION OF PIGTAIL CATHETER
Dur HO	-	DURATION OF HOSPITALIZATION
RPT (mm)	-	RESIDUAL PLEURAL THICKENING

RADIOLOGICAL RESOLUTION DURING DISCHARGE (RRDD)

Clear	-	CLEAR
Mil Eff	-	MILD EFFUSION

COMPLICATIONS (Compl)

NIL	-	NO COMPLICATIONS
TB	-	TUBE BLOCKADE
CP	-	CHEST PAIN
PTx	-	PNEUMOTHORAX

Sl.No	Pt Name	IP.No	Gender	AGE (yrs)	Maj co	Side	Qty	Sputum	PF anal	PF anal	T D (ml)	Dur PTC	Dur HO	RRDD	RPT (mm)	Compl
									AFB sm	ADA (IU)						
1	LAMANI	331040	M	28	CBFvLW	Left	MOD	Neg	Neg	38.2	2300	4D	4D	Clear	4	NIL
2	MORANJ	331876	M	68	CBFvL	Left	MIL	Neg	Neg	69.2	1400	4D	4D	Clear	0	NIL
3	PATIL	331970	M	26	CFvP	Left	MOD	Neg	Neg	73	2600	4D	4D	Clear	0	NIL
4	CHIKADDI	348325	F	20	CPBFv	Left	MOD	Neg	Neg	48	2000	4D	4D	Mil Eff	1	NIL
5	POTTODAR	350880	M	66	CBFvP	Right	MIL	Neg	Neg	28.2	1250	4D	4D	Clear	1	TB
6	ARAKERI	328204	M	29	CFvPL	Right	MIL	Pos	Neg	75	1300	4D	4D	Clear	0	TB
7	UDANAYAK	303582	M	36	CWPFv	Right	MOD	Neg	Neg	14.2	1800	4D	4D	Mil Eff	3	CP
8	YANAGAYYI	304224	M	64	CBW	Right	MOD	Neg	Pos	50.8	2300	5D	5D	Clear	2	NIL
9	CHOUGALE	337202	F	28	CBFvP	Right	MAS	Neg	Neg	73	2800	4D	4D	Mil Eff	0	NIL
10	GOBAB	352276	F	56	CPBFv	Right	MOD	Neg	Neg	120	2350	4D	4D	Clear	6	NIL
11	RAVAL	353619	M	27	CBFv	Right	MAS	Neg	Neg	62.8	2800	4D	4D	Clear	8	NIL
12	MANVADDER	362207	M	41	CPFv	Right	MOD	Neg	Neg	68	1800	4D	4D	Clear	5	NIL
13	WAGGAR	320513	M	50	PCB	Left	MOD	Pos	Neg	63.2	1800	4D	4D	Clear	3	NIL
14	VADAGOANKAR	330205	M	19	CBFvL	Right	MOD	Neg	Neg	59.1	2200	5D	5D	Clear	1	NIL
15	AUARADDI	331449	M	40	CBFvL	Left	MOD	Neg	Neg	73	2400	4D	5D	Mil Eff	0	NIL
16	HASAVAKKAR	333426	M	43	CFvP	Left	MOD	Neg	Neg	68.2	1850	3D	4D	Clear	0	NIL
17	HOOLI	348746	F	43	CPFv	Right	MAS	Neg	Neg	55	2800	3D	3D	Clear	2	NIL
18	HUMUDE	346084	F	60	CBPFv	Right	MAS	Neg	Neg	48	2800	4D	4D	Mil Eff	4	NIL
19	DHUDHADE	346705	M	19	CBLW	Right	MOD	Neg	Neg	80.4	2100	4D	6D	Clear	0	CP
20	MULIN	319776	M	35	CBLFv	Right	MOD	Neg	Neg	127.6	2050	4D	4D	Clear	5	NIL
21	KHUDE	341222	M	19	CBPLW	Right	MAS	Neg	Neg	57.8	3100	4D	5D	Mil Eff	3	NIL
22	HUNASHIKATTI	293656	F	60	PCBFv	Right	MOD	Neg	Neg	67	2500	3D	5D	Clear	0	NIL
23	NAGANNUR	325169	F	63	PLFv	Left	MAS	Neg	Pos	68	3200	3D	4D	Clear	0	NIL
24	CHOUGALE	342819	M	47	CBLFv	Right	MAS	Pos	Neg	78	3000	3D	4D	Clear	1	NIL
25	BANHATTI	361648	M	29	CRFvW	Right	MAS	Neg	Neg	66.2	3700	4D	5D	Mil Eff	4	NIL
26	BHATKANDE	284774	F	50	CPBFv	Right	MAS	Neg	Neg	72	2800	3D	5D	Clear	3	CP
27	MIRACHI	297852	M	62	CBFv	Right	MAS	Neg	Neg	80	3900	4D	6D	Clear	1	NIL
28	KATAGNAWAR	257532	M	45	CPFv	Right	MOD	Neg	Neg	67.4	2700	3D	5D	Clear	0	NIL
29	HUKKERI	350037	F	20	PCBW	Left	MOD	Neg	Neg	68	1600	4D	6D	Clear	0	NIL
30	HUKKERI	352342	M	85	CBFvL	Right	MAS	Neg	Neg	84	3080	4D	6D	Clear	2	NIL
31	YADAL	362908	F	70	CBFvL	Left	MOD	Neg	Neg	48	2750	3D	4D	Mil Eff	3	NIL
32	BANYAN	355389	F	37	CFvP	Left	MAS	Neg	Neg	88.6	3800	6D	7D	Clear	0	NIL
33	JAKUTH	355223	M	45	CPFv	Right	MAS	Neg	Neg	76.2	2900	5D	8D	Mil Eff	4	PTx
34	DESAI	362933	F	45	CBPFv	Right	MOD	Neg	Neg	68.8	2200	4D	5D	Clear	3	NIL
35	NAIK	338139	M	50	CBLW	Right	MOD	Neg	Neg	79	2300	4D	5D	Clear	0	NIL

Sl.No	Pt Name	IP.No	Gender	AGE (yrs)	Maj co	Side	Qty	Sputum	PF anal	PF anal	T D (ml)	Dur PTC	Dur HO	RRDD	RPT (mm)	Compl
36	VERNEKAR	336957	M	75	CBLFv	Right	MOD	Pos	Neg	76	1900	4D	5D	Clear	0	NIL
37	MOHITHA	320502	F	24	CBPL	Right	MIL	Neg	Neg	85	1250	3D	3D	Mil Eff	1	TB
38	MADIWAL	318218	M	25	PCBFv	Right	MOD	Neg	Neg	88.2	2800	4D	4D	Clear	4	NIL
39	RANOJI	317190	M	45	PLFv	Left	MOD	Neg	Pos	95	2300	4D	9D	Clear	3	PTx
40	BANDERI	338315	M	55	CBLFv	Right	MOD	Neg	Neg	72	2100	4D	4D	Clear	5	NIL