A STUDY OF PLEURAL FLUID CHOLESTEROL IN DIFFERENTIATING TRANSUDATIVE AND EXUDATIVE PLEURAL EFFUSION

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DR. AYYALI AMBRESH

LIST OF ABBREVIATIONS

- ADA Adenosine deaminase
- AFB Acid fast bacillus
- CEA Carcino embryonic antigen
- ELISA Enzyme linked immune sorbent assay
- FNAC Fine needle aspiration cytology
- HDL High density lipoprotein
- HBD Hydroxy butyrate dehydrogenase
- LDH Lactate dehydrogenase
- LDL Low density lipoprotein
- LE Lupus erythematosis
- LVH Left ventricular hypertrophy
- RF Rheumatoid factor
- VATS Video assisted thoracic surgery
- VLDL Very low density lipoprotein

ABSTRACT

Background: Pleural effusion is one of the common condition encountered in day to day practise. Pleural effusions represent a very common diagnostic task to the physician. A correct diagnosis of the underlying disease is essential to rational management.

Today there are a number of laboratory tests available to differentiate exudates and transudates which are considered cost effective to the patients.

So this study was designed for the measurement of pleural fluid cholesterol to differentiate transudative and exudative pleural effusions (sensitivity-97.8%, specificity-100%) with the advantage that a contemporary blood sample is not required, thereby lowering cost of diagnostic procedure.

Aims and Objectives: To study the diagnostic value of Pleural fluid Cholesterol in differentiating transudative and exudative pleural effusions.

Materials and Methods :This cross sectional descriptive study was conducted in the Department of Medicine, Shri B M Patil medical college hospital and research centre, Vijayapura on patients of pleural effusion. A study design consists of 60 patients. Age >18 years and patients with with

definitive clinical diagnosis and evidenced by radiological diagnosis of pleural effusion were taken as inclusion criteria.

Results: The results showed majority of the patients were males (63.33%) and females (36.67%). According to lights criteria 46 patients were exudates and 14 patients were transudates and according to Pleural fluid Cholesterol criteria 45 patients were exudates and 15 patients were transudates with sensitivity of 97.8% and specificity of 100% and accuracy of 98.3%.

Conclusion: The pleural fluid cholesterol criteria were found to be the most efficient criteria. Since this parameter involves the measurement of only pleural fluid values of cholesterol, it has following advantages- Economically it reduces number of biochemical tests and Simpler as there is no need to take simultaneous blood sample at the time of thoracocentesis.

Key words: Pleural Effusion, Transudates, Exudates, Cholesterol.

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INTRODUCTION

Pleural effusions represent a very common diagnostic task to the physician. A correct diagnosis of the underlying disease is essential to the rational management[1].

Normally the pleural space contains only a few millimetres of fluid. Accumulation of excessive amount of fluid is a frequent manifestation of many diseases of both thoracic and extra thoracic. Indeed pleural effusion must be regarded as a trivial event but as a sign of major disorder or disease[2].

The first diagnostic step is the identification of pleural effusions as either a transudate or exudates. This is useful because it indicates the pathophysiological mechanisms involved. Exudates are secondary to alteration of capillary permeability or lymphatic drainage. Transudates are due to either alterations of hydrostatic and / or osmotic pressure in pleural capillaries or to a fluid passing from the peritoneal cavity via diaphragmatic defects.

If an exudate is present further diagnostic procedures and tests are imperative for definitive diagnosis and specific therapy. On the other hand if the fluid is clearly a transudate one need not worry about manoeuvres directed at the pleura and need to treat only the congestive cardiac failure, nephrosis, cirrhosis or hypoproteinemia[3].

Over the years many criteria have been developed by various workers for separation of exudates and transudates.

OBJECTIVE OF THE STUDY

To study the diagnostic value of Pleural fluid Cholesterol in differentiating transudative and exudative pleural effusions.

REVIEW OF LITERATURE

HISTORICAL ASPECTSOF PLEURAL DISEASE

The word ‘pleura’ means ‘rib’ in Greek. Galen used the term pleura for both the ribcage and the lining membrane of the chest wall. Later Mandino used the word pleura for the lining membrane exclusively and this has been used ever since.

Pleurisy means inflammation of the Pleura. Hippocrates was the first to use this termin 5th century. The first to establish the site of pleurisy exclusively in the Pleura was Hermann Boerhaave(1668-1730). It was Laennec in 1820 who first to describe the association between phthisis and Pleural effusion. In 1761 AD, Auenbrugger introduced chest percussion which proved to be very useful in clinical detection of Pleural effusion. GuidoBacelli’s description of aphonic pectorilquy of Pleural effusion was another landmark. Grocco (1856-1916) described the Para vertebral dullness known as Grocco’s triangle of pleural effusion. Armand trosseau (18th century) was the person to aspirate fluid from the Pleural cavity. Later on, Delafouy, his pupil improved the procedure by employing a trocar. But it was Henry Bowditch (1802-92) who perfected the procedure paracentesis thoracis. In 1925 Jacobaeus first viewed tubercles studded over the pleura through thoracoscopy. In 1954 Sutcliff introduced open pleural biopsy as a diagnostic aid. In 1954 Defrancis used liver biopsy needle for closed biopsy. In 1958 Abram developed pleural biopsy needle which is in general use till today.

TABLE 1: SHOWS THE HISTORICAL ASPECTS OF THE VARIOUS CRITERIA/ PARAMETERS

Sl. No.	WORKER & YEAR	CRITERIA / PARAMETERS	COMMENTS
1.	Paddok f.k[4] 1940	Specific Gravity	Unacceptable misclassification rates of both transudates and exudates led to recommendation of abandonment of this criteria in a later review
2.	Leuallen&carr[5] 1955	Pleural Fluid Protein Levels >3.0/100ml	Erroneous classification of both exudates and transudates of up to 10% noted in a review
3.	Carr & power[6] 1958	Pleural Fluid Protein to Serum Protein Ratio > 0.5	Somewhat better results than above but only as for as transudates were concerned. 10% of exudates still misclassified
4.	Chandras -hekar[7]J. 1958	Pleura Fluid LDH > 200 IU/L	Was found to be inferior even to Protein criteria
5.	Light R.W[8] 1972	Combination of Protein and LDH criteria. A) Pleural Fluid to - serum protein ratio >0.5 B) Pleural LDH > 200 IU/L C) Pleural Fluid serum LDH >0.6 (LIGHTS CRITERIA)	Used routinely till today. However a large number of studies have not found satisfying results. A large percentage of transudates are misclassified in C.C.F if patient is on diuretics. Both pleural fluid and serum samples required. Hence cumbersome and costly

It is clear from the above table 1 that many of the criteria have given way to the next successive criteria. It was Lights criteria which had better success since it is based on a combination of both Protein and LDH criteria. Lights[8] criteria is used routinely today as a standard method to separate transudate and exudate.

However Lights criteria has many deficiencies. Firstly, Lights excellent results have not been fully reproduced in several studies with respect to sensitivity and specificity. A large number of prospective studies have reported specificities of only between 70% and 86% in contrast to 98% specificity claimed by Lights (Hamm[9] 1987, Hirsh[10] 1979, Peterman[11] 1984, Costa, M[12] 1989, Roth[13] 1990, Valdes[14] 1991, Burgess[15] 1995). The second major disadvantage of Lights criteria is the misclassification of transudative effusions as exudates in patients with congestive cardiac failure on diuretic therapy a phenomenon first noticed by Pillay[16] in 1965 and confirmed by Chaklo[17] in 1989. Thirdly, Lights criteriarequires both Pleural and blood samples and four biochemical measurements Hence, it is both expensive and cumbersome[18, 19].

For these reasons in recent years other researchers have proposed several new parameters for separation of transudates and exudates

Table 2: SHOWS THE NEWER PARAMETERS/CRITERIA PROPOSED AS ALTERNATIVE TO LIGHTS CRITERIA

Sl. No.	NAME OF WORKER	YEAR	CRITERIA / PARAMETERS	COMMENTS
1.	Hamm H[9]	1987	Pleural cholesterol >60mg/dl Pleural Cholesterol to Serum Cholesterol ratio >0.3	First to use cholesterol, found good results
2.	Valdes L[14]	1991	Pleural Cholesterol >55 mg/dl and/or P.Cholesterol to Serum Cholesterol ratio >0.3	Confirmed Harum's routine use of this criteria since cheaper and more efficient
3.	Roth.B.J[13]	1990	Serum-effusion albumin gradient (1.2g/dl)	Specificity not affected by concomitant diuretic therapy
4.	Meisel.S[20].	1990	Pleural bilirubin to serum bilirubin ratio >0.6	Good results not obtained in subsequent studies
5.	Tahaoglu. K[21]	1994	Alkaline phosphatase levels of Pleural fluid	Further studies needed to confirm claim of authors of high efficacy

6.	Costa.M[12]	1995	Pleural. Cholesterol>45mg/dl + Pleural LDH>200 IU/L.	No simultaneous serum sample needed. Pleural fluid data alone sufficient. Hence very cost effective.
7.	Eduardo-Garcia p[22]	1996	Pleural fluid to serum Cholinesterase ratio	Claimed to be the most accurate of all. Further studies needed since it is the newest in literature

From table 2, it is evident that the recent literature has seen a plenty of reports on various alternative criteria to Lights criteria. Controversy exists as to which method is more accurate[23].

Two of the newer criteria, cholesterol criteria of Hamm and bilirubin criteria of Meisel[29] have been to test in a number of tests studies by other workers with conflicting opinions.

Regarding cholesterol, Ortega[24] 1991,Valdes[14] 1991,Gilsuay[25]1995, KNRao[26] 1995,Mohapatra[27] 1995 are of the opinion that cholesterol criteria are the best in terms of specificity, sensitivity and cost. As opposed to this, Romero[28] 1993 and Burgess[15] 1995 in their comparative analysis found Lights criteria to be better.

Regarding bilirubin criteria, Romero[28] 1993 and Burgess in 1995 found it to be inferior to Lights criteria ,where as S Rao[29] found this parameter to be useful in separation of malignant and tubercular exudates.

Going back to Lights criteria, Manuel Vives[30] 1996 attempted a modification of Lights[8] criteria using different cut off levels but found no advantage thereon.

Amidst this confusion, Marino Costa in 1995 combined the best parameters of Lights criteria and cholesterol criteria: LDH >200 IU/L in pleural fluid and cholesterol >45mg/dl in pleural fluid and studied the effectiveness of this combination. He found these criteria had a sensitivity of 99% and specificity of 98%. This combination also had the advantage that in contrast to Lights criteria, Hamm's criteria and bilirubin criteria a simultaneous blood sample is not required and the numbers of chemical tests needed are reduced to two thus lowering the cost.

V.B. Antony et al in an editorial (Chest 1996)[31, 32] "Evaluating pleural fluid" evinces interest in Costas[12] criteria and recommends further studies in this respect.

Hence it was decided to take up this study to carryout out comparative analysis of Lights criteria and cholesterol criteria.

ANATOMY AND PHYSIOLOGY OF THE PLEURAL SPACE

The Pleura is the serous membrane that covers the lung parenchyma, the mediastinum, the diaphragm and the rib cage. The visceral pleura cover the lung parenchyma while the parietal pleura line the inside of the thoracic cavity[33]. The visceral and parietal pleura meet at the lung root.

A film of fluid is normally present between the two layers called Pleural fluid (0.1-0.2 ml/kg body wt)[34]. The space between the two layers is designated as the Pleural space (approx 10-20 microns in width)[34]. The mediastinum completely separates the right from left pleural space. The total area of both pleural surfaces is approx 2000 cm^2 in the adult man[34].

EMBRYOLOGY

The pleural cavity is derived from the body cavity of the embryo, the coelomic cavity. The lining mesothelium of the primordial lung buds becomes the visceral pleura while the lining mesothelium of the pleural cavity becomes the parietal pleura[35].

HISTOLOGY

The Parietal Pleura is composed of loose irregular connective tissue covered by a single layer of mesothelial cells, blood vessels, capillaries and lymphatic lacunae. The visceral pleura are composed of two layers the mesothelium and connective tissue. Blood vessels and lymphatics are located in the connective tissue.

BLOOD SUPPLY

The parietal pleura receive its blood supply from the systemic capillaries. The costal part from the branches of intercostal arteries, the mediastinal part from the pericardiophrenic artery and the diaphragmatic part from the superior phrenic and musculophrenic arteries. The visceral pleura are supplied by the bronchial artery[36].

LYMPHATICS[36]

The lymphatic vessels of costal pleura drain into the intercostal lymph nodes and into the internal tracheobronchial and mediastinal nodes from the mediastinal pleura. The diaphragmatic pleura is drained by the parasternal, middle phrenic and posterior mediastinal lymph nodes. The lymphatic vessels of the visceral pleura eventually reach the lung root. Fluid from the pleural space does not enter the lymphatics in the visceral pleura in man.

INNERVATION

The intercostal nerves supply the costal pleura and the peripheral part of the diaphragmatic pleura. Pain from here is referred to the adjacent chest wall. The central part of diaphragmatic pleura is innervated by the phrenic nerve. Stimulation of this pleura causes pain to be referred to the ipsilateral shoulder. The visceral pleura contain no pain fibres. Therefore the Presence of pleuritic pain indicates inflammation or irritation of the parietal Pleura.

PHYSIOLOGY OF THE PLEURAL SPACE

PLEURAL FLUID FORMATION

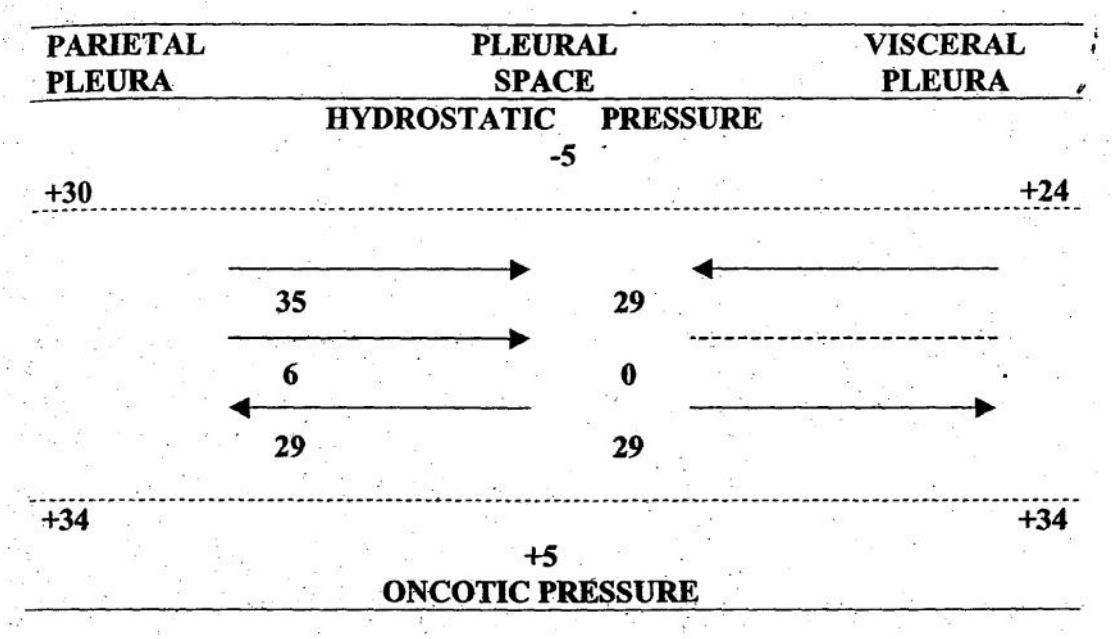
Fluid that enters the pleural space can originate in the (A) Interstitial spaces of lung, (B) the pleural capillaries, (C) the intrathoraciclymphatics and (D) the peritoneal cavity.

a) **Interstitial Origin:** In recent years it has been demonstrated that much of the fluid that enters the pleural space is from the interstitial spaces of the lungs. Either high

pressure or high permeability pulmonary oedema can lead accumulation of pleural fluid. The pulmonary interstitial spaces are also the probable origin of pleural fluid in patients with congestive cardiac failure. The likelihood of pleural effusion increases as the severity of pulmonary oedema increases[37].

b) Pleural Capillaries: The movement of fluid between the pleural capillaries and the pleural space is governed by the Starling'slaw of transcapillary exchange. When the parietal pleura is considered a net gradient for fluid formation is normally present. The net gradient for fluid movement across the visceral pleura is probably zero[38].

FIGURE 1: SHOWING VARIOUS PRESSURES THAT NORMALLY INFLUENCE THE MOVEMENT OF FLUID IN AND OUT OF THE PLEURAL SPACE.



The hydrostatic pressure in the parietal pleura is 30cm of water whereas the pleural pressure is about -5cm of water. The net hydrostatic pressure is therefore35cm

of water. Opposing this is the oncotic pressure gradient. The oncotic pressure of plasma is 34cm of water, the pleural fluid oncotic pressure is 5cm of water, the net oncotic gradient thus is 34-5 i.e.29cm of water.

The net gradient is 35-29 .i.e.6cmof water favouring movement of fluid from the capillaries into the pleural space. The pressure in the parietal pleural capillaries being 6cm of water less than that in the parietal pleural capillaries, it follows that the net gradient across the visceral pleura is zero.

c) **IntrathoracicLymphatics:** Fluid (lymph) accumulates rapidly if the thoracic duct is disrupted.

d) **Peritoneal Cavity:** Pleural fluid accumulation can occur if there is free fluid in the peritoneal cavity through the openings in the diaphragm.

Rate of pleural fluid formation is approximately 0.0lml /kg/hr[38].

PLEURAL FLUID ABSORPTION

Fluid clearance via the pleural lymphatics explains the lack of fluid accumulation normally. Most of the pleural fluid is in fact removed via the lymphatics as has been shown by several studies[38, 39]. A smaller amount of fluid is removed by the capillaries in the visceral pleura[38].

PATHOGENESIS OF PLEURAL EFFUSIONS

Normally a small amount (0.0l ml/kg/hr) of fluid which enters the space is removed by the parietal pleural lymphatic's which have a capacity to remove at least 0.20 m1/kg/hr (providing a safety factor of nearly 20).

Pleural fluid will accumulate when the rate of pleural fluid formation exceeds the rate of absorption. The main factors that lead to increased pleural fluid formation or decreased pleural fluid absorption are tabulated below.

GENERAL MECHANISMS OF PLEURAL EFFUSION[38]

A) Increased Pleural Fluid Formation:

1) **Increased interstitial fluid in the lung:** e.g. left ventricular failure, pneumonia, pulmonary embolus.

2) **Increased intravascular pressure in the pleura:** e.g. Right or left ventricular failure, SVC obstruction syndrome.

3) **Increased pleural fluid protein level:**

   Protein leak through capillaries

   Protein exudation due to local pleural inflammation

   Defective lymphatic absorption

4) **Decreased pleural pressure** : e.g. Lung atelectasis

5) **Increased fluid in the peritoneal cavity:** e.g. Ascites or peritoneal dialysis.

6) **Disruption of the thoracic duct:** Trauma, tumour, lymphomas, congenital absence of thoracic duct.

B) Decreased Pleural Fluid Absorption:

1) **Obstruction of draining lymphatics:** Tumour-lymphomas, tuberculosis, lymphangiomatosis, yellow nail syndrome, filariasis.

2) **Elevation of systemic vascular pressure:**Right ventricular failure and SVC syndrome.

The most common cause of increased pleural fluid formation is increased interstitial fluid in the lung. Whenever the amount of oedema in the lung exceeds 5g/g of lung dry weight pleural fluid accumulates. This is the predominant mechanism for the formation of pleural effusions with congestive heart failure and parapneumoniceffusions.

Increased intravascular pressure in the pleura will also lead to increased pleural fluid formation through its influence on Starling's (figure 1). Such increases occur with right ventricular failure, left ventricular failure or the superior vena cavasyndrome.

Increased pleural fluid protein level also leads to increased pleural fluid formation through its influence on Starling's equation. This occurs in increased permeability pulmonary oedema, hemothorax and with conditions where the permeability of the pleural capillaries is increased.

Decreased pleural pressure in the pleural fluid also increases pleural fluid formation through its influence on starling's equation. The most common situation is

bronchial obstruction leading to atelectasis of a lower lobe or a complete lung. In these instances, the pleural pressure can become negative, below -50cm of water.

If there is free fluid in the peritoneal cavity, it will lead to pleural fluid accumulation if there is a hole in the diaphragm. Chylewill accumulate in the pleural space if there is a disruption in the thoracic duct.

The most common cause of a decrease in the pleural fluid absorption is obstruction of the lymphatic's draining the parietal pleura. Normally the lymphatic flow from the pleural space is about 0.01 ml/kg/hr but the capacity of the lymphatics is about 0.20 ml/kg/hr unless the lymphatic flow is markedly impaired, another factor must be present in addition to lymphatic disease to produce a pleural effusion given the excess capacity of the lymphatics.

The lymphatics drain into the systemic venous circulation, elevation of the pressures in the central veins will decrease the lymphatic flow and hence pleural effusions develop when the pressure in the superior vena cava is increased.

DIAGNOSTIC EVALUATION OF PLEURAL EFFUSIONS

The presence of moderate to large pleural effusions produces symptoms and characteristic changes on physical examination. However, the relative lack of sensitivity and specificity of symptoms and signs necessitates additional tests to confirm the presence of an effusion. The following are useful in the diagnostic evaluation of pleural effusion.

1. Radiographic examination

2. Ultrasonography and tomography

3. Thoracocentesisand fluid analysis

4. Pleural biopsy

5. Thoracoscopy/bronchoscopy

RADIOGRAPHY[40,41,42]: Radiologic appearances of pleural effusions have long been recognized by chest x-ray including PA and Lateral view and if necessary lateral decubitus views are valuable tools for evaluation, While clinical examination can detect effusion only of more than 500ml, chest x-ray can detect lesser amounts of pleural fluid (100-200 ml)[43].

Pleural effusion appears as a dense homogenous opacity .A very small effusion may present as obliteration of the costophrenic angle. Moderate effusion appears a triangular lateral opacity with the base obscuring the hemi diaphragm and with a curved upper border, concave medially with extension upwards into axilla (Ellis 'S' shaped curve)[44].

For evaluating small or equivocal effusions, a lateral decubitus film is helpful. This position enables detection of less than 10 ml of pleural fluid[38,43].

At times large effusions are trapped between the intrapulmonary margin of the lung and the diaphragm. Typical signs such as blunting of costophrenic angles and Meniscus sign may be absent. The following characteristics are useful as indicators of sub pulmonic effusion

a) Apparent elevation of the diaphragm

b) Lateral displacement of the apex of diaphragmatic dome.

c) A distance of more than 2cm between the air bubble stomach and the top of left diaphragm in PA view.

d) In the lateral projection, the major fissure often bows anteriorly where it meets the convex upper margin of the fluid. A small amount of fluid is usually apparent at the lower end of major fissure.

e) In the absence of adhesions, a subpulmonic effusion can be confirmed by a lateral decubitus X-ray.

Upright chest X-ray may be difficult to obtain in acutely ill patients. Presence of free fluid elicits several signs in the supine radiograph they are: a) increased homogenous opacity with blunting of CP angle b) loss of hemi diaphragm silhouette C) apical capping d) decreased visibility of lower lobe vasculature[45].

Interlobar effusions also called “Phantom Tumours” occur in congestive heart failure[46,47]. Loculated pleural effusions both in the pleural space and in the interlobar fissures are often seen. Loculation may be differentiated from parenchymal infiltrates by the absence of air bronchogram. A definitive diagnosis of loculated pleural effusion is however done by ultrasonography.

ULTRASONOGRAPHY: Is very useful in the study of pleural disease[48, 49]. It is used in A) Identification of appropriate location for thoracocentesis/ pleural biopsy/ chest tube

placement. B) Identification of loculated effusions and C) Distinction of pleural effusion from pleural thickening[50].

It may be said that considering the advantages of ultrasound such as speed, lack of radiation, portability and lower cost, ultrasound has been underused for assessment of pleural disease. Recently it has been shown that Colour Doppler ultrasound is superior to conventional ultrasound because pleural fluid provides colour signal which can be more easily identified[43].

THORACOCENTESIS AND FLUID ANALYSIS: Thoracocentesis should be done whenever the thickness of pleural fluid on the decubitus film is greater than 10 mm or whenever loculated pleural fluid is demonstrated on ultrasound[51].Thoracocentesis is diagnostic in 75% of cases and is useful in management of another 15-20%. There are no absolute contraindications for diagnostic thoracocentesis. Relative contra-indications include bleeding diathesis, small volume of pleural fluid, anticoagulation, patients on mechanical ventilation and a low benefit to risk ratio.

The Pleural fluid is analysed under the following categories:

a) **Appearance of fluid :**Colour, Turbidity and Viscosity

b) **Total cell count:** - RBC count

- WBC count and Differential white cell count

- Test for malignant cells

c) Biochemical analysis and Immunological studies

- Protein measurement
- Glucose measurement
- Amylase determination
- Lactate Dehydrogenase Measurement
- PH Measurement
- Cancer Associated Antigens
- Adenosinedeaminase (ADA) levels
- Gamma Interferon
- ELISA for TB Antigens
- Rheumatoid Factor (RF)
- LE cells
- Anti Nuclear Antibodies
- Lipid studies

d) **Microbiologic studies:**

- Culture for bacteria, fungi, AFB
- Gram stain of fluid
- Culture of Biopsy material for AFB

3a. Appearance of fluid

Colour - most transudative and many exudative effusions are clear, straw colour and non-viscid. Any deviations should be noted and investigated. Reddish colourindicates blood while brownish tinge indicates that the blood has been present for a long time. A bloody effusion in the absence of trauma is most likely to be due to malignancy. A whitish pleural effusion is due to chyle, cholesterol or empyema. Black pleural fluid suggests aspergillus involvement of pleura. Yellow-green fluid suggests rheumatoid pleurisy. Turbid fluid can occur due to increased lipid or cellular content. Anchovy sauce pus is suggestive of amoebiasis. A bloody viscous fluid is suggestive of malignant mesothelioma[41,43]. A feculent odour indicates anaerobic infection of pleural space.

3b. Pleural fluid cell counts:

Red Blood Cell Count: When only 1 ml of blood leaks into pleural space with an effusion of 500 ml, it results in a blood tinged pleural effusion. For this reason, the mere fact that a pleural effusion is blood tinged is of little diagnostic significance Over 15% of transudative and 40% all effusions are blood tinged[52]. Grossly bloody pleural effusions having RBC count above 100,000/mm^3 suggest malignant disease, trauma or pulmonary embolism[52, 53].

White Cell Count: The total WBC counts is of little diagnostic value although a total count of above 1000/mm^3 are seen in most exudates[54, 55].Examination of a Wright stain of pleural fluid is one of the most useful tests. The following types of cells should be looked for Neutrophils, Eosinophils, Basophils, Lymphocytes, Mesothelial cells, Macrophages and Malignant cells.

Lymphocytes: If more than 50% of WBC in an exudative pleural effusion are small lymphocytes it narrows down the diagnosis to Malignancy or Tuberculosis and is an indication for pleural biopsy.

Separation of pleural lymphocytes into T and B Lymphocytes has in general been of little use. However in Chronic Lymphatic Leukaemia or Lymphoma, B Lymphocytes predominate[57].

Neutrophils: Are the cellular component of the acute inflammatory response. They predominate in pleural fluid resulting from acute inflammation. If neutrophils predominate in a case of congestive cardiac failure, the possibility of pulmonary embolism should be entertained[53].

Eosinophils: Pleural fluid eosinophilia (more than 50% of the total count) may occur in parasitic disease, pneumothorax, pulmonary infarction, traumatic hemothorax, previousthoracocentesis, and asbestos related pleural effusions. Pleural effusions secondary to drug reactions are frequently eosinophilic.

Basophils: Basophilic pleural effusions are uncommon. They may be present in eosinophilic effusions. Basophil counts over 10% are most common with Leukemic pleural involvement.

Mesothelial cells: They are significant for two reasons. First, their presence or absence is useful diagnostically because they are uncommon in tubercular effusions[53,58,59]. This is because of extensive involvement of pleural surfaces by the disease process denying entry of mesothelial cells into the pleural space. Secondly, mesothelial cells particularly in their activated form may be confused with malignant cells.

Macrophages: There are of little diagnostic value but they are likely to be confused with mesothelial cells.

Malignant cells: Are very important in the diagnosis of pleural effusions because with it a definitive diagnosis can be made in 50% of malignant effusions. When 3 separate pleural fluid specimens are submitted to an experienced cytologist, one should expect a positive diagnosis in about 80% of patients.

3c. Biochemical analysis

Protein measurement: The pleural fluid protein levels are generally higher in exudative effusions but are not useful in separating the various types of exudative effusions. But if a pleural fluid meets the exudative criteria with its LDH but not its protein level then such effusions are almost always Para pneumonic or malignant[60, 61, 62.]

Glucose measurement[63]: Is useful in the differential diagnosis because a low pleural fluid glucose level (less than 60 mg/dl) indicates that the patient has one of following i.e. Tubercular, Malignant, Rheumatoid or Para pneumonic effusion though rare causes include hem thorax, Chug-Strauss syndrome, Paragnomiasis etc.

Amylase Determination: Pleural fluid amylase above upper normal limits indicates pancreatic effusions like (i) acute pancreatitis (ii) pancreatic pseudocyst, oesophageal rupture and malignant effusions (in malignant effusions it is only moderately elevated) Amylase Isoenzyme determinations are useful in distinguishing malignant and pancreatic effusions[64].

Lactate Dehydrogenase Measurement: The level of pleural fluid LDH is used to separate transudate from exudate, but is of no use in the differentiation of various exudates. It is a reliable indicator of the degree of pleural inflammation. If serial LDH levels are progressively higher in a patient, the degree of inflammation is increasing and one should be aggressive in pursuing the diagnosis. The presence of blood in the pleural fluid usually does not adversely affect the LDH levels.

The LDH isoenzyme is of limited value in the differential diagnosis of exudative effusions, though most malignant effusions have a higher percentage of LDH-4 LDH-5 levels than the corresponding serum value[65].

PH measurement[63, 66, 67, 68:] pH less than 7.2 is seen in complicated parapneumonic effusions, oesophageal rupture, rheumatoid pleuritis, tubercular pleuritis, hemothorax, systemic acidosis, lupus pleuritis and urinothorax. If the pleural fluid pH is less than 7.00 in a parapneumonic effusion, then the patient invariably has a complicated parapneumonic effusion and is an indication for tube thoracostomy. With a malignant pleural effusion, a pH of less than 7.30 predicts a short survival, an increased yield on biopsy and cytology, and a poor response to sclerosing agents.

Carcino Embryonic Antigen[69]: CEA levels are elevated in malignant effusions. If it is above 10mg/ml it is highly suggestive of malignancy though not diagnostic. When CEA is elevated cytology is usually positive.

Adenosine deaminase measurement [70, 71, 72, 73:] ADA levels are higher in tuberculous exudates than in other exudates. ADA is the enzyme that catalyses the conversion of adenosine to inosine. Many studies have confined the early reports. It appears that pleural

fluid ADA level above 70 u/l is highly suggestive of tuberculouspleuritis while a pleural ADA level below 40 u/l virtually rules out this diagnosis. High ADA levels in pleural fluid have also been seen in rheumatoid pleurisies and are a small percentage of neoplasms.

Gamma Interferon[71]: Levels of Gamma Interferon are higher with tuberculouspleuritis(above 200 pg/ml) and is apparently very useful in the diagnosis of tubercular effusions.

LE cells: Most pleural effusions secondary to SLE contain LE cells. With the development of better immunological tests for SLE, LE preparations are being done less and less frequently nowadays.

Rheumatoid Factor: Rheumatoid Factor though high in rheumatoidpleuritis is not pathognomonic since elevated levels are seen in tuberculosis, carcinomatous effusions and in empyema. However Light[8] recommends that the demonstration of Pleural fluid RF $\geq$ 1:320 and RF $\geq$ serum level is strong evidence for rheumatoid pleural effusion.

Complement levels: Most patients with rheumatoid Pleural effusions and pleural effusions secondary to SLE have reduced pleural fluid complement levels. But this does not absolutely separate SLE and RA effusions from other exudates and hence routine determinations are not advocated.

Lipid studies: The diagnosis of chylothorax is best made by the triglyceride levels of pleural fluid. Pleural Fluid Triglyceride levels > 110 mg/dl: indicates chylothorax, if less 50 mg/dl rules out chylothorax. The demonstration of chylomicrons in the pleural fluid on lipoprotein analysis is diagnostic of chylothorax. Patients with chyliform effusions have

pleural fluid triglyceride levels > 250 mg/dl. The cholesterol levels in pleural fluid are elevated in high- lipid pleural effusions due to high numbers of cholesterol crystals or lecithin globulin complexes. Cholesterol levels may also be elevated in chylous effusions. When the turbidity of pleural fluid is due to cholesterol crystals, examination of pleural fluid sediment reveals cholesterol crystals which are large polyhydric or rhomboid crystals.

3d. Microbiologic studies on pleural fluid

Cultures: Pleural fluid from undiagnosed exudative effusions should be cultured for bacteria, mycobacteria and fungi. For bacterial cultures bedside inoculation of culture media can be done. For mycobacterial cultures both conventional cultures (yield 21-48 days) and Bactec system (yield 3-40 days) can be used. In tubercular effusions, sample of pleural biopsy can also be sent for culture for mycobacterium tuberculosis[74].

PLEURAL BIOPSY[75, 76, 77, 78:] Needle biopsy of pleura is useful is establishing the diagnosis of malignant or tuberculous pleural effusions. Sample of pleura can be easily obtained with Abram's or Cope's pleural biopsy needle.

With TB pleuritis the needle biopsy is positive for granulomas in 50-80% of patients. Demonstration of granulomas on pleural biopsy is virtually diagnostic of TB pleuritis; caseation or AFB need not be demonstrated. Culture of biopsy specimens yields positivity in 75% of cases. Biopsy of pleura in malignant effusion is recommended only when initial cytology of pleural fluid is negative. Needle biopsy of thickened pleura is also useful to establish etiology. Other diagnosis where pleural Biopsy is useful

occasionally issarcoidosis, fungal disease of pleura (Coccidiomycosis), rheumatoidpleurisy.

Contraindications to pleural biopsy are as follows

i. Transudative effusion

ii. Syn-pneumonic effusions secondary to pneumonia, embolism, SLE, pancreatitis.

iii. Associated bleeding diathesis or anticoagulation

iv. Obliterated pleural space

v. Un-cooperative patient.

Complications of pleural biopsy include:

- Pneumothorax 3-15%, Site pain 1-15%
- Vasovagal reaction 1-5%, Hemothorax<2%
- Subcutaneous emphysema < 1%, Air Embolism <1%
- Biopsy of extra pleural tissue <1%

False positive biopsies have been found most often when reactive mesothelial cells are often confused with tumour cells. Open pleural biopsy has been largely supplemented by VATS (Video Assisted Thoracoscopy). The main indication nowa day is progressive undiagnosed pleural disease.

THORACOSCOPY AND BRONCHOSCOPY[79, 80, 81]: Thoracoscopy also called pleuroscopy is useful in cases where etiology of effusion remains obscure after both

routine analysis and biopsy. With the advent of Video-assisted Thoracic Surgery (VATS) there has been renewed interest in the use of thoracoscopy. Thoracoscopy is excellent in diagnosing malignant disease of pleura for managing patients with complex pneumothorax and for breaking down loculation in complicated parapneumonic effusions. Bronchoscopy is useful in patients with pleural effusion who have associated parenchymal abnormalities such as atelectasis or masses. It is also indicated if the patient hashemoptysis and in the diagnosis of massive effusions even with no parenchymal abnormality or hemoptysis.

DIFFERENTIAL DIAGNOSIS OF PLEURAL EFFUSION

A.Transudative pleural effusion

1. Congestive heart failure

2. Cirrhosis

3. Nephrotic syndrome

4. Superior venacaval obstruction

5. Fontan procedure

6. Urinothorax

7. Peritoneal dialysis

8. Glomerulonephritis

9. Myxoedema.

10. Pulmonary emboli

11. Sarcoidosis.

B. Exudative pleural effusion

I .Neoplastic disease

1. Metastatic disease eg: Ca Lung, CaBreastand lymphoma.

2. Mesothelioma

II. Infectious diseases.

1. Bacterial infections

2. Tuberculosis

3. Fungal infections

4. Parasitic infections

5. Viral infections

III. Pulmonary embolization

IV. Gastrointestinal diseases

1. Pancreatic diseases

2. Subphrenic abscess

3. Intrahepatic abscess

4. Intrasplenic abscess

5. Oesophageal perforation

6. After abdominal surgery

7. Diaphragmatic hernia

8. Endoscopic variceal sclerosis

9. After liver transplant

V. Collagen vascular diseases

1. Rheumatoid pleuritis

2. Systemic lupus erythematosis

3. Drug induced lupus

4. Immunoblastic lymphadenopathy

5. Sjogrenssyndrome

6. Familial Mediterranean fever

7. ChurgStrauss syndrome

8. Wegenersgranulomatosis

VI. Drug induced pleural disease

1. Nitrofurantoin

2. Dantrolene

3. Methysergide

4. Bromocryptine

5. Amiodarone

6. Procarbazine

7. Methotrexate

VII. Miscellaneous diseases and conditions

1. Asbestos exposure

2. Meig syndrome

3. Post myocardial infarction syndrome

4. Yellow nail syndrome

5. Sarcoidosis

6. Pericardial disease

7. After coronary artery bypass surgery

8. After lung transplant

9.Fetal pleural effusions

10. Uremia

11. Trapped lung

VIII.Haemothorax

IX .Chylothorax

CHOLESTEROL IN PLEURAL FLUID

It has long been known that cholesterol is constantly present in all pleural fluids. Until recently the cholesterol content of pleural fluid has been used, together with the concentrations of other lipid fractions to distinguish between Chylothorax and pseudochylothorax. Chylothorax occurs when the thoracic duct is disrupted causing chyle to enter the pleural space. A Pseudochylothorax occurs due to accumulation of large amounts of cholesterol in long standing effusions.

CHOLESTEROL PLEURAL EFFUSION[84,85,86,87,83]

(Syn-pseudochylous effusion, cholesterol thorax, chyliform effusion)

History: Cholesterol pleural effusion is a rare condition. Until 1961 only 99 cases had been reported in literature .The first description was by Nauyn in 1865 and then by Guneau de Moussy in 1874. Churton published the first detailed description in 1882 in his case he found that there was degeneration of cells leading to formation of cholesterol. Malgatti reviewed 44 cases from literature up to 1929, in his series the commonest associated condition was tuberculosis. Stein in 1932 reported a male of 45 years with 25 years history of pleural effusion. Pleural fluid cholesterol was 2353 mg%, autopsy revealed gross pleural thickening with calcification enabling the pleural sac to be removed like a cast. Durham and Diamond (1939), Moll and Fowweather 1940, Erwin l941 have all described cholesterol thorax. In most of their cases the pleural effusion was long standing (mean 7 years).

Aetiology: The most common causes are tubercular pleuritis and rheumatoid pleuritis.

Pathogenesis[85,86,88]: The precise mechanism of chyliform effusions is not known. Most of the cholesterol is associated with HDL in contrast to the cholesterol in acute exudates where it is mostly bound to LDL[89]. It has been hypothesized that cholesterol that enters the pleural space with acute inflammation becomes trapped and undergoes changein lipoprotein binding characteristics. The diseased pleura results in abnormally slow transfer of cholesterol out of the pleural space resulting in accumulation.

The origin of the cholesterol and other lipids is not definitely known but one possibility is from degenerating red and white blood cells in the pleural fluid. Most patients with cholesterol effusions have no altered cholesterol metabolism because the serum cholesterol levels are normal. Some chyliform effusions contain cholesterol crystals.

Diagnosis: is based on pleural fluid appearance, microscopic detection of cholesterol crystals and by elevated pleural fluid cholesterol levels. Lipoprotein analysis may have to be performed if doubt exists whether the fluid is chylous or pseudochylous because only chylous fluid contains chylomicrons. Triglyceride levels may be elevated even in cholesterol effusions and hence are not useful in differentiation from chylous effusion.

Treatment: is both by specific therapy (eg Anti Tubercular Therapy) and by therapeutic thoracocentesis. Decortication may result in markedly improved functional status in many cases.

CHYLOUS PLEURAL EFFUSIONS[90,91] (Syn-chylothorax)

A chylothorax is formed when the thoracic duct is disrupted and chyle enters the pleural space.

Etiology: In over 50% of cases the cause is Tumour, mainly Lymphomas. Chylothorax may be the presenting symptom of Lymphoma. The second leading cause is surgical and penetrating trauma to chest andthirdly, congenital chylothorax.

Diagnosis: is by appearance of fluid, and by triglyceride measurement. If the pleural fluid triglyceride level is above 110 mg/dl chylothorax is ruled out. In those with levels in between, lipoprotein analysis establishes the diagnosis since chylomicronsare present only in chillies effusions.

Treatment: In Traumatic chylothorax therapeutic thoracocentesis, pleuroperitoneal shunt, maintenance of nutrition and hydration and in some by tube thoracotomy. The defect in the thoracic duct closes spontaneously by itself in time. Recently Video-assisted Thoracic surgery (VATS) has been used in better management of chylous effusions.

CHOLESTEROL CRITERIA FOR THE SEPARATION OF PLEURAL TRANSU DATES AND EXUDATES.

It was well known that cholesterol levels are high in pseudochylothorax which is seen in Tuberculosis effusions and rheumatoid effusions of long standing duration. Recently Hinrich Hamm[9] was the first to conduct a systematic study of pleural fluid cholesterol levels in transudative and exudative pleural effusions. In his study he showed that cholesterol levels are already elevated in exudative effusions of much shorter duration. He found that a cut off value of 60 mg/dl was highly effective in the separation of transudates and exudates and simpler and cost effective compared to Lights[8] criteria.

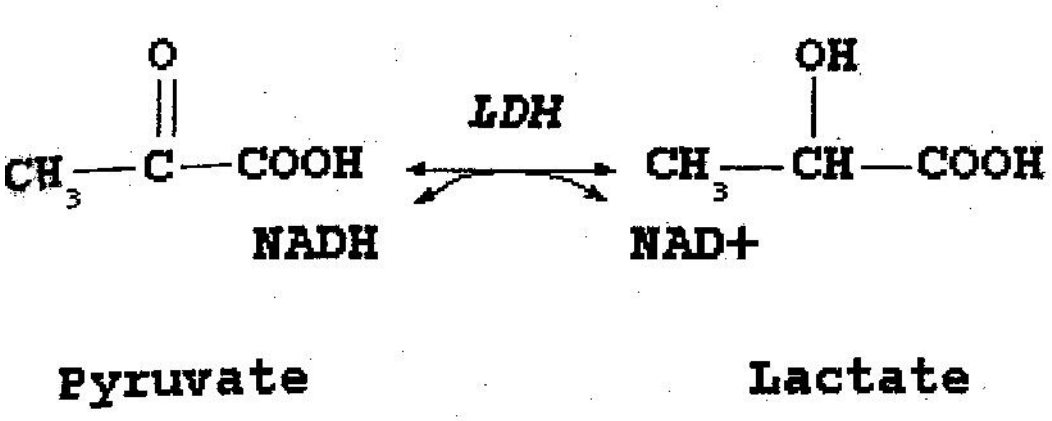
His study was validated by another study of Valdes[14] who found that determination of pleural cholesterol was of great value and recommended its inclusion in routine laboratory analysis of pleural effusions in view of better efficacy and cost efficiency.

Regarding the cause of rise in cholesterol levels in pleural exudates Hamm[9] proposed two possible hypotheses. One is cellular degeneration mainly of white cells and red cells as assumed for pseudochylothorax. Another is that the accumulation of cholesterol reflects a “serum leakage” (an analogy to the postulated mechanism for protein). Some 70 percentage of plasma cholesterol is bound to low density, high molecular weight lipoproteins (LDL) and the rest to HDL or very low density lipoproteins (VLDL), and the increased permeability of pleural capillaries in pleural exudate would allow plasma cholesterol to enter the pleural cavity. Valdes adds that the local synthesis of cholesterol by the pleural cells themselves.

LACTATE DEHYDROGENASE IN PLEURAL FLUID[65]

Lactate dehydrogenase (LDH) is an enzyme that is found in almost all body tissues but only a small amount of it is usually detectable in the blood. It usually stays contained within the tissue cells. When cells are damaged or destroyed however, they release LDH into the bloodstream causing blood levels to rise.

It catalyses the interconversion of pyruvate and lactate with concomitant interconversion of NADH and NAD+. As it can also catalyse the oxidation of hydroxybutyrate, it is occasionally called HydroxybutyrateDehydrogenase (HBD).



Lactate dehydrogenase is a widespread cytosolic enzyme found in the greatest concentrations in heart, skeletal muscle, liver, kidney and red blood cells. Many of these tissues rely on anaerobic glycolysis for energy.

LDH is a tetramer made up of combinations of 2 polypeptide chains. Five isoenzymes are recognized, designated LD1 to LD5, according to the Number of each chain, type resulting in different molecular mass and electrophoretic mobility.

- LD1 and LD2 are predominant in the heart, red cells and kidney

- LD4 and LD5 are predominant in the liver and some skeletal muscles
- No single isoenzyme predominate in the lung and spleen

Reference/ normal values for entire LDH	Normal values
Men	135-225 U/L
Women	135-215 U/L

MATERIALS AND METHODS

1. SOURCE OF DATA:

- Data is collected from patients who are attending Medicine OPD and admitted in BLDEU'S Shri B. M. Patil medical college hospital and research centre, VIJAYAPURA.
- Period of study is from November 2016 to July 2018.

2. METHOD OF COLLECTION OF DATA:

Inclusion Criteria:

- Age >18years
- Patients with definite clinical diagnosis and Pleural effusion evidenced by radiological imaging.

Exclusion criteria:

- Age <18years
- Patients without definitive clinical diagnosis
- Patients previously diagnosed and already on treatment.

3. TYPE OF STUDY

Cross sectional descriptive study

4. SAMPLE SIZE:

Using expected incidence of exudates cases among pleural effusion as 69.4%,expected sensitivity as 88%, expected specificity as 100% and desired precision as +/-10%,

The minimum sample is 60.

This sample size will give the precision of 10%for both sensitivity and specificity.

Formula used:

$$N=z^2 (1-p)/d^2$$

Z-value of z statistic at 5% level of significance

d-margin of error

p-expected incidence rate

Statistical Analysis:

Data will be analysed using mean+/-SD Chi square test for association, comparison of means using test,ANOVA for comparison between and within groups and diagrammatic presentation.

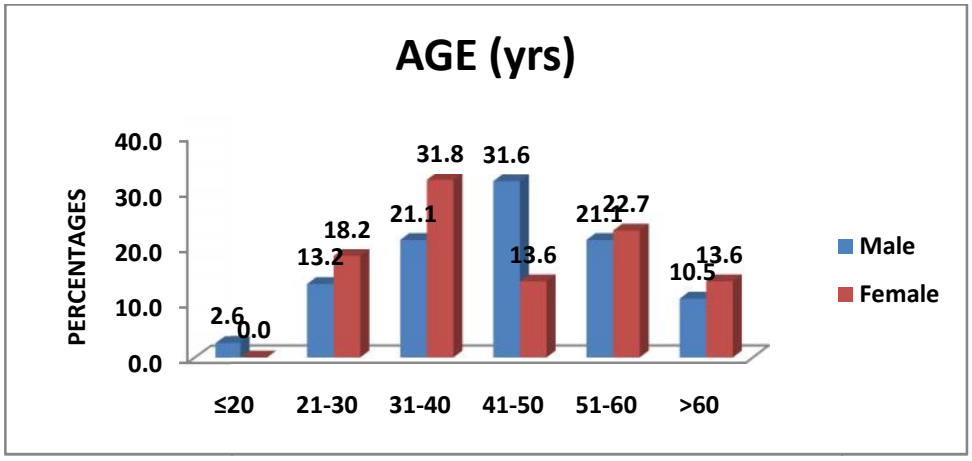
RESULTS AND OBSERVATION

The present study was undertaken in 60 cases of Pleural Effusion over a period of 2 and half years from November 2016 to July 2018, the results of which are given below.

TABLE 1: AGE AND SEX DISTRIBUTION

AGE (years)	Male		Female		p value
	N	%	N	%	
18-20	1	2.6	0	0.0	0.641
21-30	5	13.2	4	18.2	
31-40	8	21.1	7	31.8	
41-50	12	31.6	3	13.6	
51-60	8	21.1	5	22.7	
>60	4	10.5	3	13.6	
Total	38	100.0	22	100.0	

FIGURE 1a: AGE AND SEX DISTRIBUTION

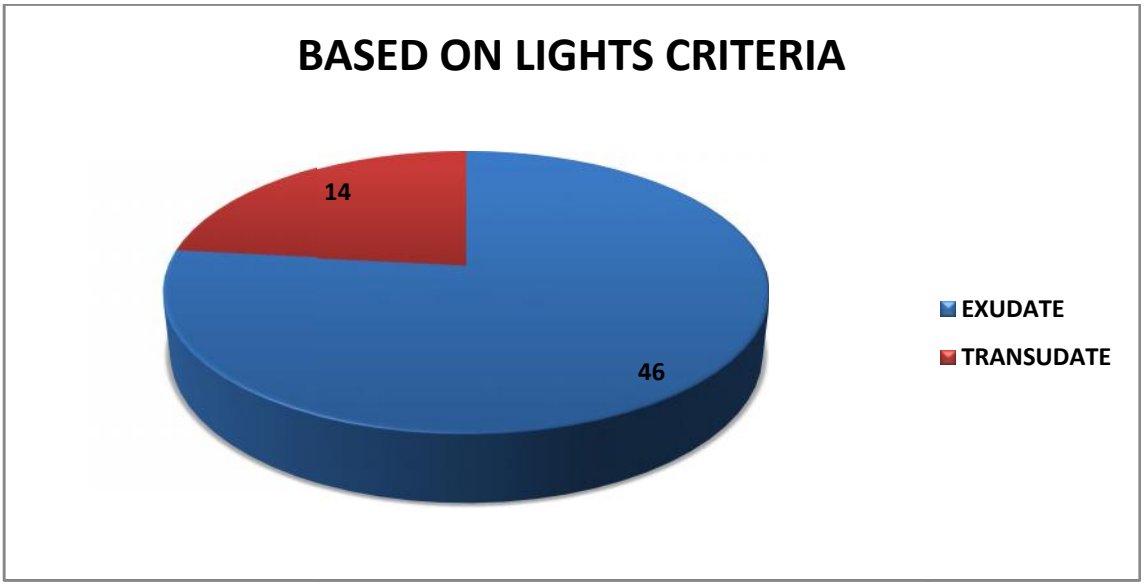


The age of the patient in this study ranged from 18years to 75 years. 1 patient was 18 years,9 patients were under 21-30years,15 patients were under 31-40 years,15 patients were under 41-50years,13 patients were under 51-60 years,7 patients were above 60 years. Out of 60 patients there were 38 males and 22 females.

TABLE 2: DISTRIBUTION OF EXUDATES AND TRANSUDATE ACCORDING TO LIGHTS CRITERIA

BASED ON LIGHTS CRITERIA	N	%
EXUDATE	46	76.7
TRANSUDATE	14	23.3
Total	60	100

FIGURE 2a: DISTRIBUTION OF EXUDATES AND TRANSUDATE ACCORDING TO LIGHTS CRITERIA

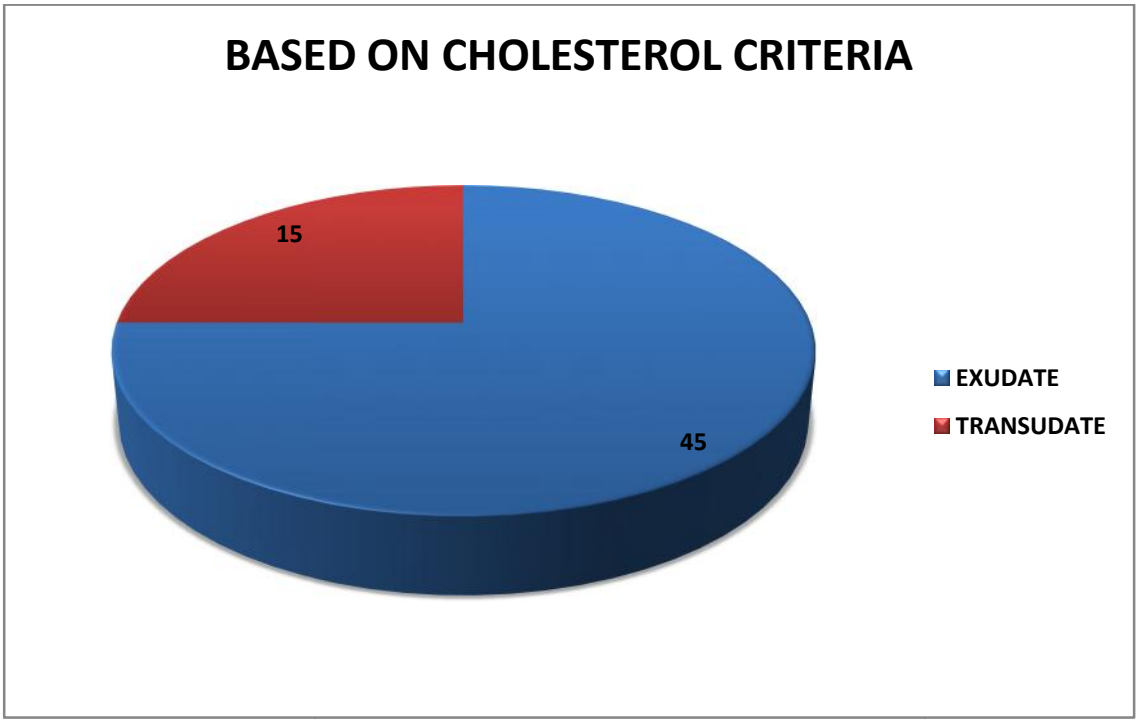


Based on Lights criteria, out of 60 patients 46 were exudates(76.7%) and 14 were transudates (23.3%).

TABLE 3: DISTRIBUTION OF EXUDATES AND TRANSUDATE ACCORDING TO PLEURAL FLUIDCHOLESTEROL CRITERIA

BASED ON CHOLESTEROL CRITERIA	N	%
EXUDATE	45	75
TRANSUDATE	15	25
Total	60	100

FIGURE 3a: DISTRIBUTION OF EXUDATES AND TRANSUDATE ACCORDING TO PLEURAL FLUIDCHOLESTEROL CRITERIA



Based on pleural cholesterol level criteria, out of 60 patients 45(75%) were exudates and 15(25%) were transudates.

TABLE 4: DISTRIBUTION OF SYMPTOMS IN PLEURAL EFFUSION AT PRESENTATION

PRESENTING SYMPTOMS	NUMBER (N=60)	PERCENTAGE
1. COUGH	50	83.3
2. FEVER	22	36.7
3. CHEST PAIN	34	56.7
4. DYSPNOEA	47	78.3
5. SWELLING OF LIMBS	10	16.7
6. DISTENSION OF ABDOMEN	10	16.7
7. FACIAL PUFFINESS	6	10
8. LOSS OF APPETITE	60	100
9. LOSS OF WEIGHT	40	66.7

Cough was present in 50 patients (83.3%) , fever in 22 patients (36.7%), chest pain in 34 patients (56.7%) , dyspnoea in 47(78.3%) , swelling of limbs and abdominal distension each in 10 patients (16.7%) , facial puffiness in 6 patients , loss of appetite in 60 patients (100%) , loss of weight in 40 patients (66.7%).

FIGURE 4a: DISTRIBUTION OF SYMPTOMS IN PLEURAL EFFUSION AT PRESENTATION

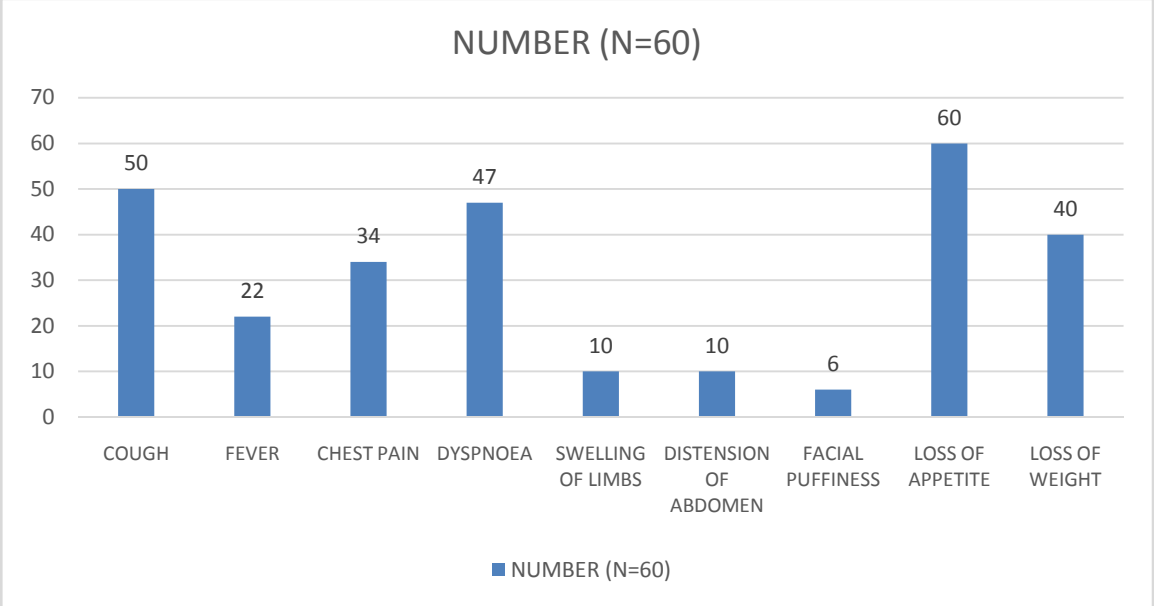


TABLE 5: DISTRIBUTION OF SIGNS IN PLEURAL EFFUSION AT PRESENTATION

CLINICAL SIGNS	NUMBER(N=60)	PERCENTAGE
1. STONY DULLNESS	60	100
2. ABSENT BREATH SOUND	50	83.3
3. DECREASED VF/VR	52	86.7
4. MEDIASTINAL SHIFT	50	83.3
5. PLEURAL RUB	4	6.6
6. CREPITATIONS	5	8.3

Stony dullness in 60 patients (100%) , Decreased / absent breath sounds in 50 patients (83.3%) , Mediastinal shift in 33 patients (83.3%), Decreased vf/vr in 52 patients (86.7%), Pleural rub in 4 patients (6.6%), Crepitations in 5 patients (8.3%).

FIGURE 5a: DISTRIBUTION OF SIGNSIN PLEURAL EFFUSION AT PRESENTATION

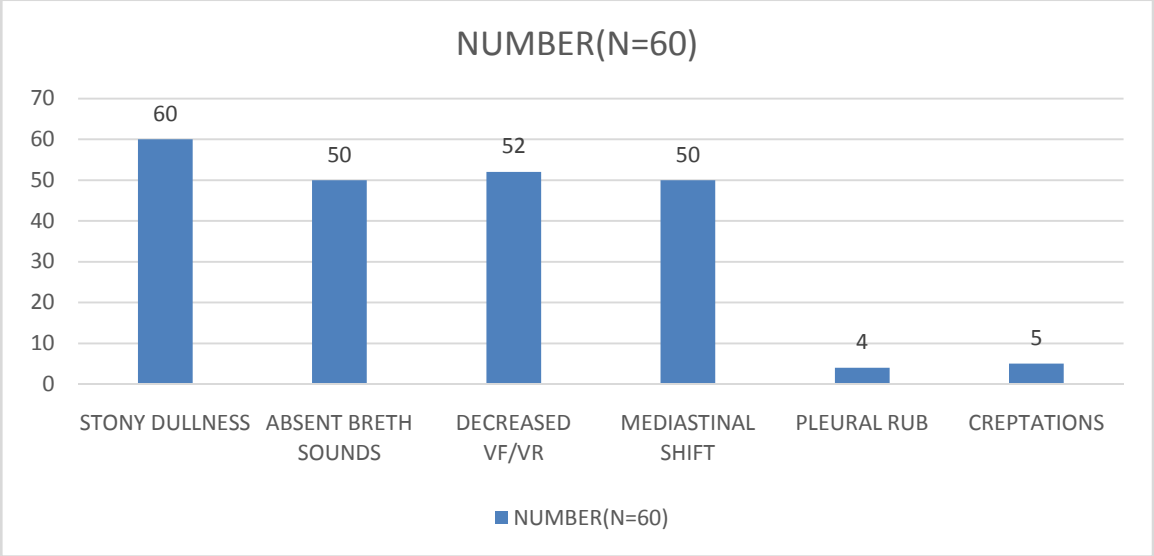


TABLE 6: PLEURAL EFFUSION RIGHT AND LEFT SIDE DISTRIBUTION

SIDE OF EFFUSION	NUMBER(N=60)	PERCENTAGE
RIGHT	36	60.0
LEFT	19	31.7
BILATERAL	5	8.3

Out of 60 patients, 36 had right sided effusion ,19 had left sided effusion ,5 patients had bilateral pleural effusion.

FIGURE 6a: PLEURAL EFFUSION RIGHT AND LEFT SIDE DISTRIBUTION

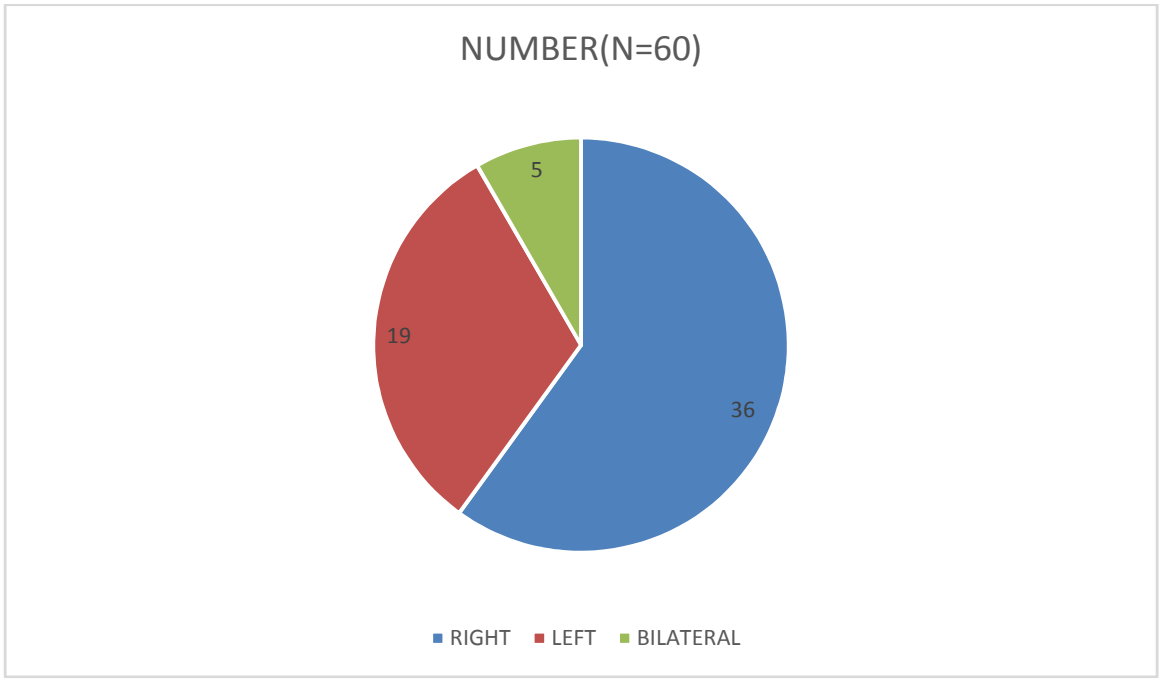
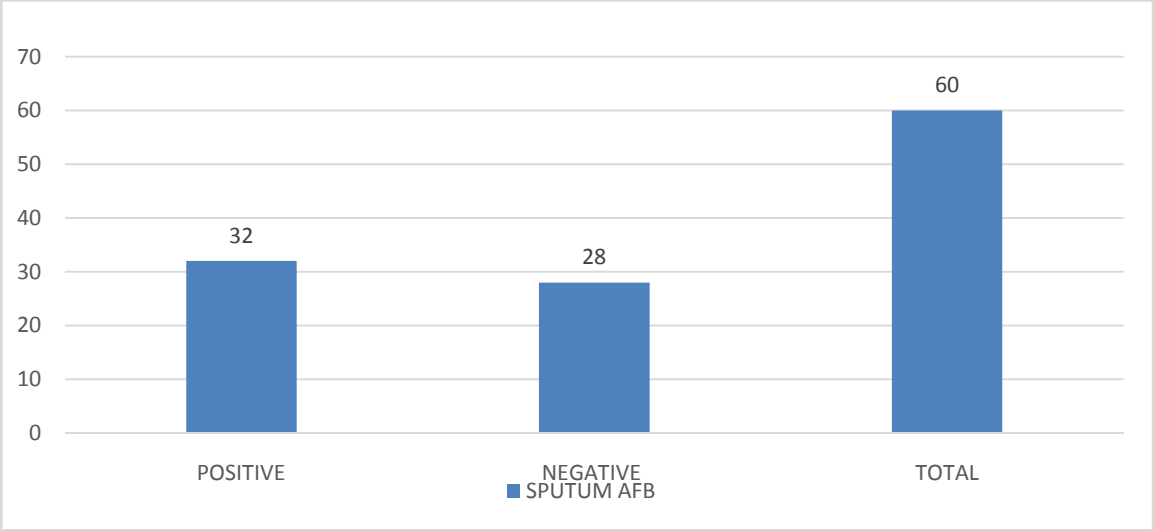


TABLE 7: RESULT OF SPUTUM AFB

SPUTUM AFB	TOTAL		P VALUE
	N	%	
NEGATIVE	28	46.7	0.232
POSITIVE	32	53.3	
TOTAL	60	100.0	

FIGURE 7a: RESULT OF SPUTUM AFB

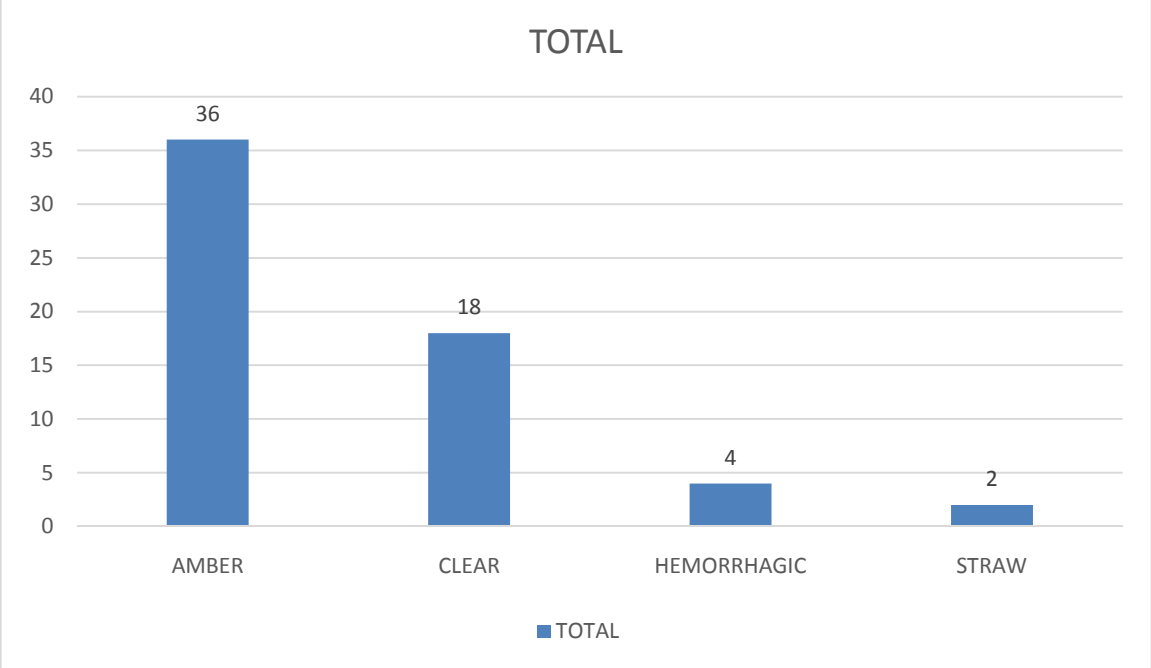


In the study group of 60 patients, sputum AFB was positive in 32 (53.3%) patients and 28 (53.3%) patients had sputum AFB was negative.

TABLE 8: APPEARANCE OF PLEURAL EFFUSION

COLOUR	Total		p value
	N	%	
AMBER	36	60.0	0.864
CLEAR	18	30.0	
HAEMORRHAGIC	4	6.7	
STRAW	2	3.3	
Total	60	100.0	

FIGURE 8a: APPEARANCE OF PLEURAL EFFUSION

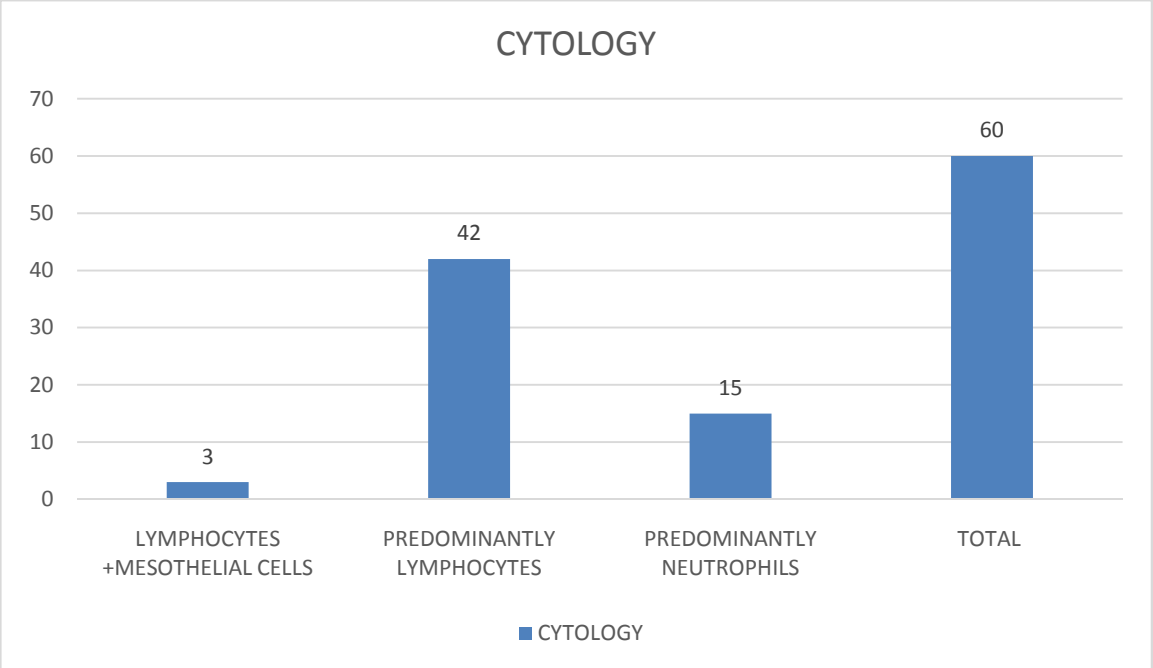


Colour of pleural effusion – 36 patients had amber colour, 18 patients had clear, 4 patients had haemorrhagic and straw colour in 2 patients.

TABLE 9: CYTOLOGY OF PLEURAL EFFUSION

CYTOLOGY	Total		p value
	N	%	
LYMPHOCYTES + MESOTHELIAL CELLS	3	5.0	0.476
PREDOMINANTLY LYMPHOCYTES	42	70.0	
PREDOMINANTLY NEUTROPHILS	15	25.0	
Total	60	100.0	

FIGURE 9a: CYTOLOGY OF PLEURAL EFFUSION



Out of 60 patients, 3 patients had lymphocytes plus mesothelial cells, 42 patients had predominantly lymphocytes and 15 patients had predominantly neutrophils.

TABLE 10: DISTRIBUTION OF PLEURAL PROTEIN

PLEURAL PROTEIN (gram/dl)	Number (n=60)
1-2	6
2-4	16
4-6	34
>6	4
Total	60

The above table shows the values of pleural protein. 6 patients had pleural protein values ranging from 1-2 gram/dl, 16 patients of pleural protein ranging from 2-4 gram/dl, 34 patients ranging from 4-6 gram/dl and 4 patients had protein levels above 6 gram/dl.

FIGURE 10a: DISTRIBUTION OF PLEURAL PROTEIN

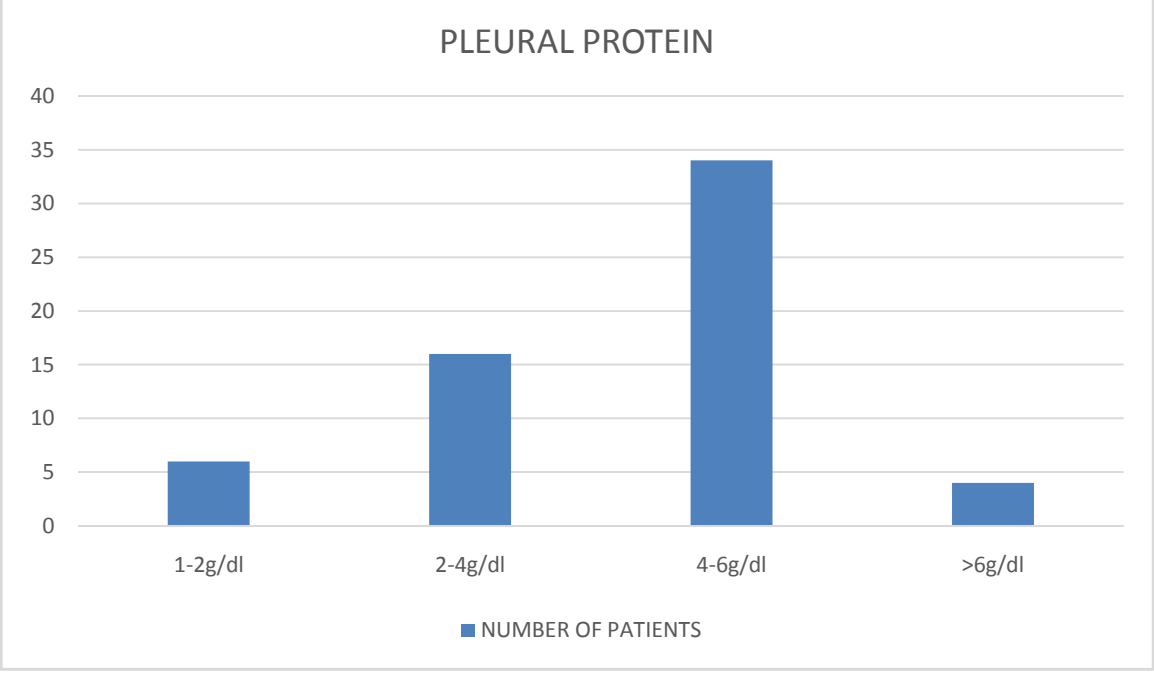


TABLE 11: DISTRIBUTION OF PLEURAL CHOLESTEROL

PLEURAL CHOLESTEROL	NUMBER (N=60)
<45 mg/dl	15
>45 mg/dl	45
Total	60

15 patients had pleural cholesterol levels less than 45 mg/dl and 45 patients had cholesterol level above 45 mg/dl.

FIGURE 11a: DISTRIBUTION OF PLEURAL CHOLESTEROL

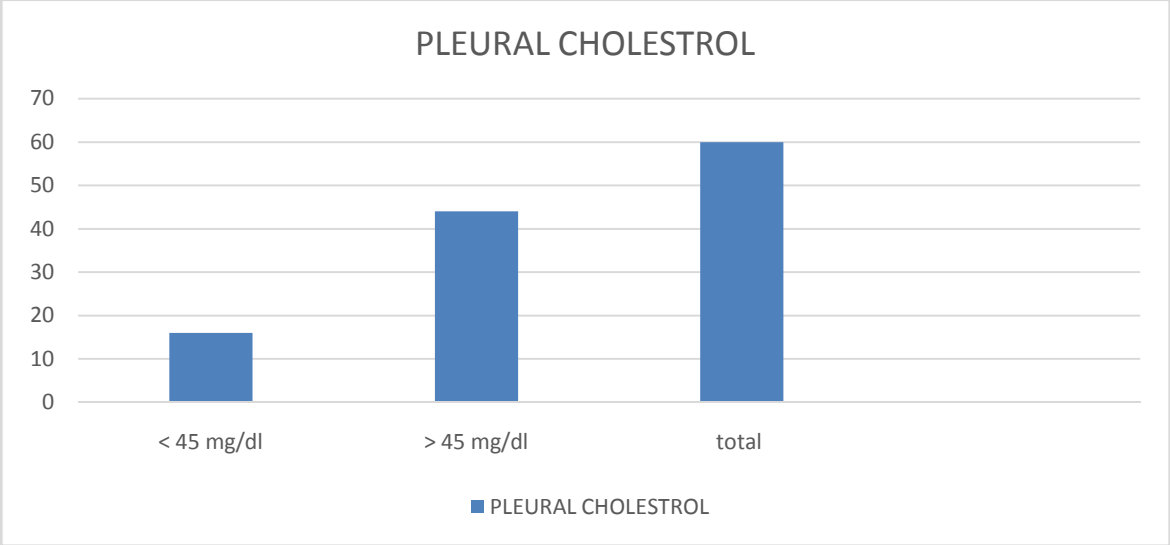


TABLE 12: BIOCHEMICAL ANALYSIS OF PLEURAL EFFUSION

PARAMETERS	EXUDATES		TRANSUDATE		P VALUE
	MEAN	SD	MEAN	SD	
LIGHTS CRITERIA (TRANSUDATE=14 EXUDATE=46)					
SERUM PROTEIN	5.7	1.0	6.1	1.1	0.215
PLEURAL PROTEIN(G/DL)	4.7	1.0	2.4	0.9	<0.001*
PLEURAL SUGAR	68.2	40.1	126.7	75.9	<0.001*
PLEURAL FLUID PROTEIN:SERUM PROTEIN	0.8	0.2	0.3	0.1	<0.001*
PLEURAL CHOLESTEROL CRITERIA(TRANSUDATE=15 EXUDATE=45)					
PLEURAL CHOLESTEROL	78.2	23.7	21.9	9.2	<0.001*

Note: * significant at 5% level of significance (p<0.05). The p value of serum protein is 0.215, pleural protein is <0.001, pleural sugar is <0.001, pleural cholesterol is <0.001, pleural fluid protein: serum protein is <0.001. P value of <0.001 is statistically significant.

FIGURE 12a: BIOCHEMICAL ANALYSIS OF PLEURAL EFFUSION

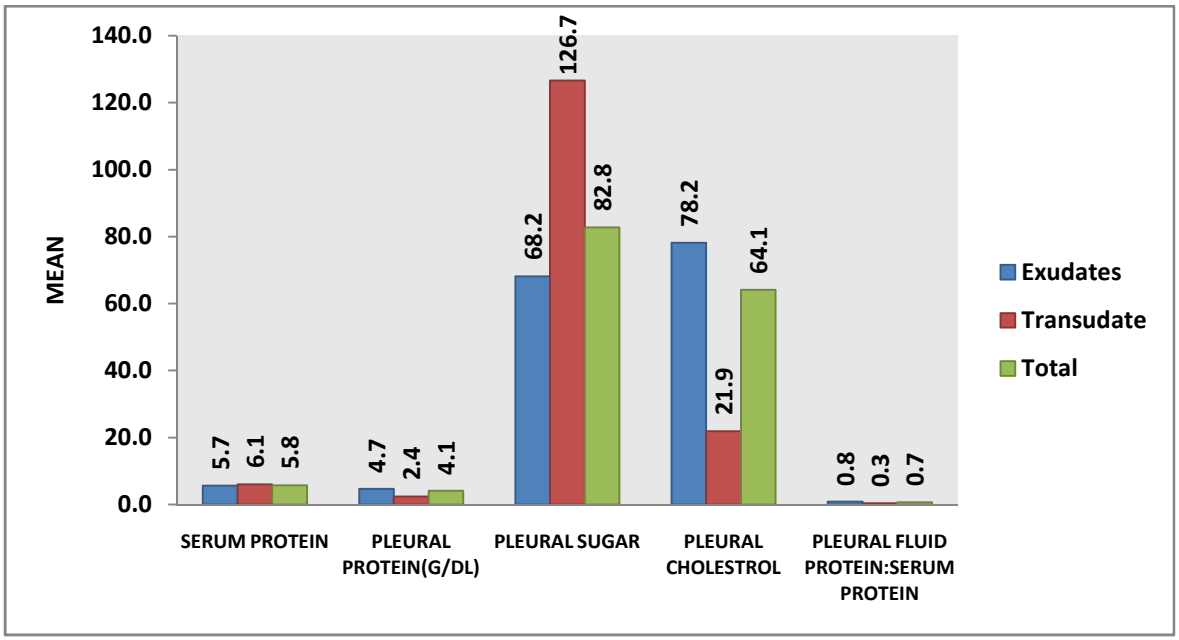
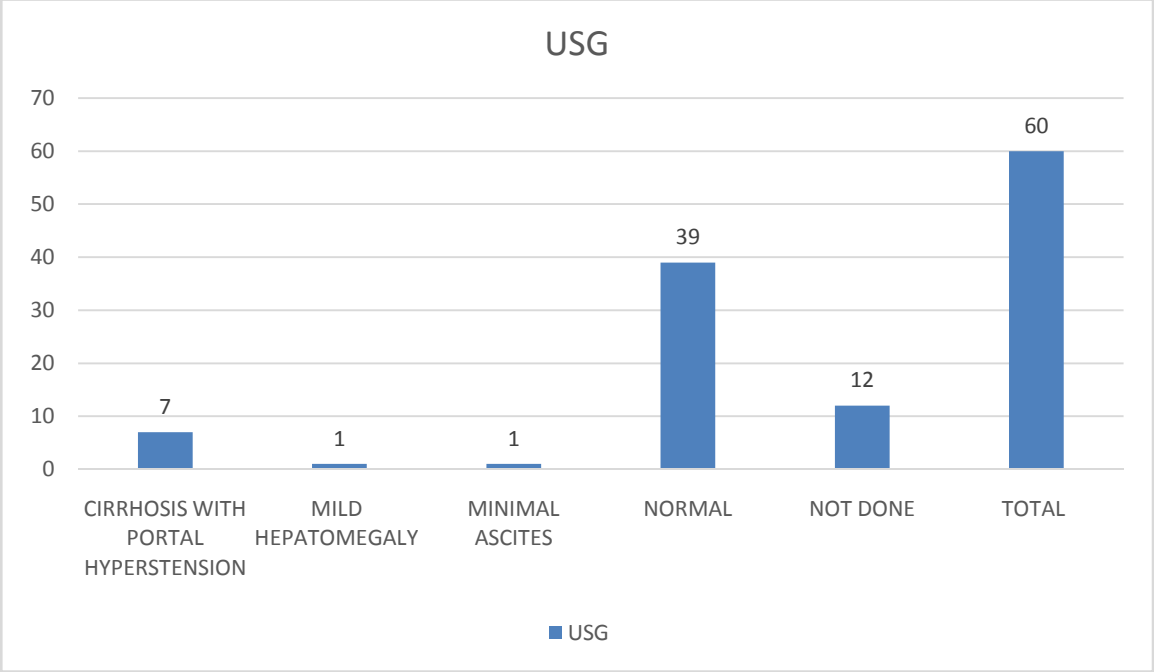


TABLE 13: USG ABDOMEN EXAMINATION IN PLEURAL EFFUSION

USG	Total		p value
	N	%	
CIRRHOSIS WITH PORTAL HYPERTENSION	7	11.7	0.182
MILD HEPATOMEGALY	1	1.7	
MINIMAL ASCITES	1	1.7	
NORMAL	39	65.0	
NOT DONE	12	20.0	
Total	60	100.0	

FIGURE 13a: USG ABDOMEN EXAMINATION IN PLEURAL EFFUSION



In the study group of 60 patients, cirrhosis was present in 7 patients, hepatomegaly in 1 patient, ascites in 1 patient, and normal in 39 patients.

TABLE 14: COMPARISON OF EXUDATIVE AND TRANSUDATIVE PLEURAL EFFUSION ACCORDING TO PLEURAL FLUID CHOLESTEROL CRITERIA AND LIGHTS CRITERIA

	ACCORDING TO PLEURAL FLUID CHOLESTEROL CRITERIA (N=60)		ACCORDING TO LIGHTS CRITERIA (N=60)		P VALUE
	N	%	N	%	
EXUDATE	45	75.0	46	76.66	<0.001*
TRANSUDATE	15	25.0	14	23.33	
TOTAL	60	100.0	60	100.0	

Note: * significant at 5% level of significance ($p<0.05$)

Based on lights criteria 46 patients were exudate and 14 patients were transudative pleural effusion , based on cholesterol criteria 45 patients were exudative and 15 were transudative pleural effusion.

The p value is < 0.001 which is statistically significant.

TABLE 15: SENSITIVITY ANALYSIS OF PLEURAL CHOLESTEROL CRITERIA

TP (true positive)	45
FN (false negative)	1
FP (false positive)	0
TN (true negative)	14
Sensitivity	97.8%
Specificity	100.0%
PPV(positive predictive value)	100.0%
NPV(negative predictive value)	93.3%
Accuracy	98.3%

DISCUSSION

A total of 60 patients were taken up for this study. Out of 60, 46 were exudates and 14 were transudates. Among 46 exudates, 40 were tubercular effusions, 5 patients were synpneumonic effusion and 1 patient with malignant effusion.

Among 14 transudative, 7 patients were congestive cardiac failure, 7 patients were cirrhosis.

AGE AND SEX: The age of the patient in this study ranged from 18 years to 75 years. 1 patient was 18 years, 9 patients were between 21-30 years, 15 patients were between 31-40 years, 15 patients were between 41-50 years, 13 patients were between 51-60 years and 7 patients were above 60 years. Out of 60 patients, males were 38 and females were 22.

PRESENTING SYMPTOMS: Cough was present in 50 patients (83.3%) , fever in 22 patients (36.7%) , chest pain in 34 patients (56.7%) , dyspnoea in 47 (78.3%) , swelling of limbs and abdominal distension each in 10 patients (16.7%) , facial puffiness in 6 patients , loss of appetite in 60 patients (100%) , loss of weight in 40 patients (66.7%).

SIGNS: Mediastinal shift is seen in 50 patients opposite to the pleural effusion. Over affected side of chest, fullness of chest in 48 patients, decreased chest movements in 50 patients, expansion of chest reduced in 50 patients, decreased vocal fremitus in 52 patients, stony dullness in 60 patients, absent breath sounds in 50 patients, decreased vocal resonance in 52 patients, pleural rub in 4 patients and Crepitations in 5 patients.

SIDE OF PLEURAL EFFUSION: Out of 60 patients, 36 had right side effusion, 19 had left side effusion, and 5 patients had bilateral pleural effusion.

SPUTUM AFB ANALYSIS: In the study group of 60 patients, sputum AFB was positive in 32 (53.3%) patients and 28 (53.3%) patients had sputum AFB was negative.

COLOUR OF PLEURAL EFFUSION DISTRIBUTION: Colour of pleural effusion 36 patients had amber colour, 18 patients had clear fluid, 4 patients had haemorrhagic and straw colour in 2 patients.

CYTOLOGY OF PLEURAL EFFUSION: Out of 60 patients, 3 patients had predominantly lymphocytes plus mesothelial cells, 42 patients had predominantly lymphocytes and 15 patients had predominantly neutrophils.

USG ANALYSIS: In the study group of 60 patients, cirrhosis was present in 7 patients, hepatomegaly in 1 patient, ascites in 1 patient, and normal in 39 patients.

BIOCHEMICAL ANALYSIS OF PLEURAL EFFUSION:

Table 16: Biochemical analysis of pleural effusion

PARAMETERS	**EXUDATES**		**TRANSUDATE**		**P VALUE**
	MEAN	**SD**	**MEAN**	**SD**	
LIGHTS CRITERIA (TRANSUDATE=14 EXUDATE=46)					
SERUM PROTEIN	5.7	1.0	6.1	1.1	0.215
PLEURAL PROTEIN(G/DL)	4.7	1.0	2.4	0.9	<0.001*
PLEURAL SUGAR	68.2	40.1	126.7	75.9	<0.001*
PLEURAL FLUID PROTEIN:SERUM PROTEIN	0.8	0.2	0.3	0.1	<0.001*
PLEURAL CHOLESTEROL CRITERIA(TRANSUDATE=15 EXUDATE=45)					
PLEURAL CHOLESTEROL	78.2	23.7	21.9	9.2	<0.001*

According to Lights Criteria, the mean serum protein is 5.7±1.0 in exudates and 6.1±1.1 has p value of 0.215. The mean pleural protein is 4.7±1.0 in exudates and 2.4±0.9 has p value of 0.001. The mean pleural sugar is 68.2±40.1 in exudates and 126.7±75.9 has p value of 0.001. The mean pleural protein: serum protein is 0.8±0.2 in exudates and 0.3± 0.1 has p value of 0.001. According to pleural cholesterol criteria, the mean pleural cholesterol is 78.2±23.7 in exudates and 21.9±9.2 and has p value of 0.001 which is statistically significant.

PLEURAL FLUID CHOLESTEROL:

Table 17: Comparison of pleural fluid cholesterol values between the studies

Sl.no	AUTHORS	SENSITIVITY	SPECIFICITY	PPV	NPV	ACCURACY
1	Hamm[9]	93.5	100	100	91	96
2	Valdes[14]	92.5	87.6	95	80	91.3
3	Ram[103]	96	93	96	92.6	95
4	B N Mohaptra[27]	92	100	100	99	93
5	Burgess[15]	54	92.2	87.3	50	66
6	Present study	97.8	100	100	93.3	98.3

Hamm[9] first used pleural cholesterol as a parameter. In his study of 150 patients he found excellent results (Sensitivity 93%, Specificity 100%, Accuracy 96%). Following Hamm's[9], Valdes[14] aimed to validate this parameter. In his study of 74 patients pleural cholesterol had good results as shown in the above table. Similar results were obtained from studies by Ram[103] in 100 patients and B N Mohaptra[27] in his study of 132 patients. The studies of Burgess[15] and Remero[28] of 124 patients, results were in favour of lights criteria but they had less sensitivity, specificity and accuracy. As a result the present

study of 60 patients which contains Pleural Cholesterol criteria has more sensitivity, specificity and accuracy when compared to other studies done by Burgess and Remero which contains Lights criteria.

The study shows that pleural fluid cholesterol criteria (cholesterol >45 mg/dl - exudate and cholesterol <45 mg/dl – transudate) constitute a useful tool for the separation of pleural effusions.

CONCLUSION

The pleural fluid cholesterol criteria were found to be the most efficient criteria.

Since this parameter involves the measurement of only pleural fluid values of cholesterol, it has following advantages

1) Economically , it reduces number of biochemical tests
2) Simpler, as there is no need to take simultaneous blood sample at the time of thoracocentesis.

It is concluded that the determination of pleural fluid cholesterol criteria can be included in routine analysis of pleural fluid samples in place of presently used Lights Criteria.

SUMMARY

This was a cross sectional descriptive study of 60 cases of pleural effusion. The parameter pleural fluid cholesterol levels are used in comparison with Lights criteria to distinguishing transudative and exudative pleural effusion. The following results were obtained in the present study.

- True positive in 45 cases
- False negative 1 case
- False positive 0 case
- True negative 14 case
- Sensitivity 97.8%
- Specificity 100%
- Positive predictive value 100%
- Negative predictive value 93.3%
- Accuracy 98.3%

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ANNEXURE 1

ETHICAL CLEARANCE CERTIFICATE

B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 04-10-2016 *at* 03 pm *to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.*

Title "A Study of Pleural fluid Cholestrol in differentiating exudative and transudative pleural effusion"

Name of P.G. student Dr Ayyali Ambrose
Dept of Medicine

Name of Guide/Co-investigator Dr M.S. Mulimani
Prof & HOD of Medicine

DR.TEJASWINI. VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Researeh project.
2) Copy of informed consent form
3) Any other relevant documents.

ANNEXURE 2

BLDEU'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA- 586103

INFORMED CONSENT FOR PARTICIPATION DISSERTATION/ RESEARCH

I, the undersigned _____ S/O.D/O.W/O _____,aged ____years ordinarily resident of _____do here by state/declare that Dr Ayyali Ambresh of Shri B.M.Patil Medical College and Hospital has examined me thoroughly on _____at_____(place) and has explained to me in my own language _____that I am suffering from _____ disease (condition) and this disease/condition mimic following diseases _____Further Dr Ayyali Ambresh informed me that she is conducting dissertation/research titled

"TO STUDY THE DIAGNOSTIC VALUE OF PLEURAL FLUID CHOLESTROL IN DIFFERENTIATING EXUDATIVE AND TRANSUDATIVE PLEURAL EFFUSION" of Shri B.M.Patil Medical College, VIJAYAPURA. Under the guidance of Dr M S Mulimani requesting my participation in the study.

Doctor has also informed me that during conduct of this procedure _____ like adverse results may be encountered. Among the above complications most of them are treatable but are not anticipated hence there is chance of aggravation of my condition and in rare circumstances it may prove fatal in spite of anticipated diagnosis and best treatment made available.

Further Doctor has informed me that my participation in this study help in evaluation of results of the study which is useful reference for treatment of other similar cases in near future, and also I may be benefited in getting relieved of suffering or cure of the disease I am suffering.

The Doctor has also informed me that information given by me, observations made/ photographs/video graphs taken upon me by the investigator will be kept secret and not accessed by the person other than me or my legal hirer except for academic purposes.

The Doctor did inform me that though my participation is purely voluntary based on information given to me, I can ask any clarification during the course of treatment/study related to Diagnosis, Procedure of treatment, result of treatment or prognosis. At the same time I have been informed that I can withdraw from my participation in this study at any time if I want or investigator can terminate me from the study at any time from the study but not the procedure of treatment & follow up unless I request to discharge.

In the view of anticipated or unexpected complications during the course of study, that I will be treated free of cost, as explained by the investigator.

After understanding the nature of dissertation or research, Diagnosis made, mode of treatment I the under signed Shri/Smt _____under my full conscious state of mind I agree to participate in the said research/Dissertation .

Signature of patient:

Signature of Doctor:

Witness 1

Witness 2

Date:

Place:

ANNEXURE 3

BLDE'S SHRI B.M.PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA

CASE PROFORMA

"TO STUDY THE DIAGNOSTIC VALUE OF PLEURAL FLUID CHOLESTROL IN DIFFERENTIATING EXUDATIVE AND TRANSUDATIVE PLEURAL EFFUSION"

Name: IP No:

Address: Date:

Age: Sex:

History

Presenting complaints

Chest pain:

Dyspnoea:

Syncope:

Vomitting:

Fever:

Sweating:

Others:

Personal Habits

Smoking: Alcoholism:

General physical examination

Height (Cms): Weight (Kgs):

Body Mass index:

Pulse rate (bpm):

Blood pressure (mm Hg): SBP: DBP:

SYSTEMIC EXAMINATION:

Respiratory System

Upper respiratory tract :

Lower respiratory tract:

A. INSPECTION:

1. Shape of chest:
2. Drooping of shoulder:
3. Lye of ribs:
4. Trachea:
5. Apex beat:
6. Chest movements:
7. Any engorged veins:
8. Visible pulsations/scars:
9. Spine

B. PALPATION:

1. Trachea:
2. Apex beat:

3. Chest movements:

4. Tactile vocal fremitus:

	Right	Left
Supraclavicular		
Infraclavicular		
Mammary		
Inframammary		
Axillary		
Infraaxillary		
Suprascapular		
Interscapular		
Infrascapular		

5.Measurements:

AP diameter:

Transverse diameter:

Right hemithorax:

Left hemithorax:

Circumference:

On inspiration:

On expiration:

Expansion:

C. PERCUSSION:

	Right	Left
Supraclavicular		
Infraclavicular		
Mammary		
Inframammary		
Axillary		

Infraaxillary

Suprascapular

Interscapular

D.AUSCULTATION:

1. Air entry:
2. Breath sounds:
3. Rhonchi
4. Crepitations:
5. Wheeze:
6. Pleural rub:
7. Bronchophony:
8. Egophony:
9. Whispering pectoriloquy:
10. Vocal resonance:

	Right	Left
Supraclavicular		
Infraclavicular		
Mammary		
Inframammary		
Axillary		
Infraaxillary		
Suprascapular		
Interscapular		
Infrascapular		

Cardiovascular System

Central Nervous System

Per abdomen

Provisional diagnosis:

Investigations

CBC: TC

DC

Hb

ESR

UR: Albumin

Sugar

Pus cells

Epitelial cells

Serum Sodium:

Serum Potassium:

Serum Creatinine:

Pleural fluid analysis:

Cell count	Protein	Glucose	LDH	pCHOL	Gram stain	Culture	Acid fast stain	cytology

Pleural fluid cholesterol:

MASTER CHART

SL.NO	NAME	AGE	SEX	COUGH	FEVER	CHEST PAIN	DYSPNOEA	SWELLING OF LIMBS	ABD DISTENSION	FACIAL PUFFINESS	LOSS OF APPETITE	LOSS OF WEIGHT	STONY DULLNESS	DECREASE/ABSENT BREATH SOUNDS	VF/VR	MEDIASTINAL SHIFT	PLEURAL RUB	CREPTS	SIDE OF EFFUSION	HB	ESR	WBC TC	CREATININE	SPUTUM AFB	CXR	COLOUR	ADA	CYTOLOGY	PLEURAL PROTEIN(G/DL)	PLEURAL SUGAR	PLEURAL CHOLESTROL	USG
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33
1	PEERAPPA	65	M	P	P	P	P	A	A	A	P	P	P	P	P	P	A	A	RIGHT	10.9	100	6370	0.7	POSITIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	65	PREDOMINANTLY LYMPHOCYTES	5.4	95	110	NORMAL
2	LAXMAN	35	M	P	A	P	P	A	A	A	P	P	P	P	P	P	A	A	RIGHT	3.4	150	5000	0.5	POSITIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	9	PREDOMINANTLY LYMPHOCYTES	2.5	50	52	NORMAL
3	MUDUKAPPA	68	M	P	A	A	A	P	P	P	P	A	P	P	P	P	P	A	RIGHT	11.1	70	9770	1	POSITIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	32	PREDOMINANTLY LYMPHOCYTES	5.1	95	100	NORMAL
4	ITABHAI	55	F	P	P	P	P	A	A	A	P	P	P	P	P	P	A	A	LEFT	12.2	10	11070	1.2	NEGATIVE	LEFT SIDE PLEURAL EFFUSION	AMBER	45	PREDOMINANTLY LYMPHOCYTES	3.5	85	80	NOT DONE
5	PARSAPPA	44	M	P	P	P	P	A	A	A	P	P	P	A	A	A	A	P	RIGHT	13.3	85	10750	1	NEGATIVE	RIGHT SIDE PLEURAL EFFUSION	CLEAR	5	PREDOMINANTLY LYMPHOCYTES	4.7	49	96	NOT DONE
6	BASAMMA	35	F	P	P	A	P	A	A	A	P	P	P	P	P	P	A	A	RIGHT	10.9	55	10330	0.7	NEGATIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	31	LYMPHOCYTES + MESOTHELIAL CELLS	5.6	57	69	NOT DONE
7	MALLAPPA	46	M	P	A	A	A	A	A	A	P	P	P	P	P	P	P	A	LEFT	10.7	90	8400	0.8	POSITIVE	LEFT SIDE PLEURAL EFFUSION	CLEAR	31	LYMPHOCYTES + MESOTHELIAL CELLS	5.6	57	69	NOT DONE
8	AMBOJI	50	M	P	A	P	P	A	A	A	P	A	P	A	A	A	A	A	RIGHT	12.1	60	12040	0.6	POSITIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	29	LYMPHOCYTES + MESOTHELIAL CELLS	4.2	184	62	NOT DONE
9	HANMANTH	42	M	P	P	A	P	A	A	A	P	P	P	P	P	P	A	P	LEFT	9.2	110	6010	1.6	NEGATIVE	LEFT SIDE PLEURAL EFFUSION	AMBER	8	PREDOMINANTLY LYMPHOCYTES	4.7	40	47	NOT DONE
10	DAVALSAB A	70	M	P	A	P	P	P	P	A	P	A	P	A	A	P	A	P	RIGHT	9.7	65	16160	1	POSITIVE	RIGHT SIDE PLEURAL EFFUSION	CLEAR	40	PREDOMINANTLY LYMPHOCYTES	1.2	160	22	NORMAL
11	YALLAMMA	68	F	P	A	A	P	A	A	A	P	A	P	P	P	P	A	A	RIGHT	11.6	30	8250	2.5	NEGATIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	68	PREDOMINANTLY LYMPHOCYTES	5.4	87	101	NORMAL
12	NINGAPPA	25	M	P	P	P	P	A	A	P	P	A	P	P	P	A	A	A	RIGHT	11.4	100	7450	0.6	NEGATIVE	RIGHT SIDE PLEURAL EFFUSION	STRAW	74	PREDOMINANTLY LYMPHOCYTES	5.6	105	25	NOT DONE
13	BHIMAVVA	31	F	P	A	P	P	A	A	A	P	P	P	P	P	P	A	A	LEFT	12	50	11234	1.9	NEGATIVE	LEFT SIDE PLEURAL EFFUSION	AMBER	73	PREDOMINANTLY LYMPHOCYTES	5.9	54	98	NORMAL
14	MAHADEVI	25	F	P	A	A	A	A	A	A	P	A	P	P	P	P	P	A	RIGHT	9.2	70	8400	0.7	POSITIVE	RIGHT SIDE PLEURAL EFFUSION	CLEAR	80	PREDOMINANTLY LYMPHOCYTES	5.7	103	35	NORMAL
15	ALLABAKSHI	27	M	P	P	A	P	A	A	A	P	P	P	P	P	P	A	P	BILATERAL	13.4	20	7560	0.6	POSITIVE	BILATERAL EFFUSION	HEMORRHAGIC	40	PREDOMINANTLY LYMPHOCYTES	4.6	115	92	NORMAL
16	SUJATA	30	F	P	A	P	P	A	A	A	P	P	P	P	P	A	A	A	RIGHT	7.8	65	6140	1.1	NEGATIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	56	PREDOMINANTLY NEUTROPHILS	5.4	58	102	CIRRHOSIS WITH PORTAL HTN
17	SOMAKKA	40	F	P	A	A	P	A	A	A	P	A	P	P	P	A	A	A	LEFT	12	10	5870	1	NEGATIVE	LEFT SIDE PLEURAL EFFUSION	CLEAR	15.9	PREDOMINANTLY LYMPHOCYTES	3	163	65	NORMAL
18	HANMESHA	28	M	P	P	P	P	A	A	A	P	P	P	P	P	P	A	A	LEFT	14	40	4350	0.9	NEGATIVE	LEFT SIDE PLEURAL EFFUSION	AMBER	10	PREDOMINANTLY NEUTROPHILS	1.7	150	20	NORMAL
19	ARUNA	34	F	A	A	P	P	A	A	A	P	A	P	P	P	P	A	A	RIGHT	8.6	95	15030	0.6	POSITIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	78	PREDOMINANTLY LYMPHOCYTES	5.8	50	25	NORMAL
20	SANTOSH	45	M	P	A	P	A	A	A	A	P	P	P	P	P	A	P	P	RIGHT	8.3	120	8060	0.9	NEGATIVE	RIGHT SIDE PLEURAL EFFUSION	CLEAR	70	PREDOMINANTLY NEUTROPHILS	5.9	68	55	CIRRHOSIS WITH PORTAL HTN
21	JAYASHRI	40	F	P	A	A	P	A	A	P	P	P	P	P	P	A	A	A	LEFT	9	50	7650	1.2	NEGATIVE	LEFT SIDE PLEURAL EFFUSION	AMBER	13.4	PREDOMINANTLY LYMPHOCYTES	6.4	91	114	NORMAL
22	SIDAMMA S	45	F	P	A	A	P	A	A	A	P	A	P	P	P	P	A	A	BILATERAL	6.3	95	3100	0.8	NEGATIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	18	PREDOMINANTLY LYMPHOCYTES	2.5	95	100	NORMAL
23	RUDRAYYA	50	M	P	A	P	P	A	A	A	P	A	P	P	P	P	A	A	LEFT	11.2	60	7520	0.8	POSITIVE	LEFT SIDE PLEURAL EFFUSION	CLEAR	49	PREDOMINANTLY LYMPHOCYTES	4.7	57	50	NORMAL
24	MEHBOOBSAB	44	M	P	A	A	A	A	A	A	P	A	P	P	P	P	A	A	RIGHT	12	40	11250	0.4	NEGATIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	21	PREDOMINANTLY LYMPHOCYTES	5.3	121	92	NOT DONE
25	BASAYYA	36	M	A	P	A	P	A	A	A	P	P	P	A	A	P	A	A	RIGHT	8.8	90	15190	1	NEGATIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	18	PREDOMINANTLY LYMPHOCYTES	4.2	30	60	NOT DONE
26	MALLAYYA	60	M	P	A	P	P	A	A	A	P	P	P	P	P	P	A	P	RIGHT	9	100	32000	0.9	NEGATIVE	RIGHT SIDE PLEURAL EFFUSION	CLEAR	70	PREDOMINANTLY LYMPHOCYTES	6.4	113	100	NOT DONE
27	DAVALAMMA	50	F	P	P	A	P	A	A	A	P	P	P	P	P	A	A	A	BILATERAL	12	60	6600	0.7	POSITIVE	BILATERAL EFFUSION	HEMORRHAGIC	16.5	PREDOMINANTLY LYMPHOCYTES	4.1	49	65	NOT DONE
28	BASSAMMA	75	F	P	A	A	P	A	A	A	P	A	P	P	P	A	A	A	RIGHT	12.3	35	14110	0.9	POSITIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	13	PREDOMINANTLY LYMPHOCYTES	4.4	65	88	NORMAL
29	KAJAPPA	70	M	P	P	A	A	P	P	A	P	P	P	P	P	P	P	A	RIGHT	13.7	60	19530	1	NEGATIVE	RIGHT SIDE PLEURAL EFFUSION	CLEAR	150	PREDOMINANTLY NEUTROPHILS	5.2	10	100	CIRRHOSIS WITH PORTAL HTN
30	YASHWANTH	46	M	A	A	P	P	A	A	P	P	A	P	P	P	P	A	P	LEFT	9.1	40	14980	0.7	POSITIVE	LEFT SIDE PLEURAL EFFUSION	AMBER	67	PREDOMINANTLY NEUTROPHILS	4.6	10	45	NORMAL
31	LAKAPPA	60	M	P	A	P	P	P	P	A	P	P	P	P	P	P	A	A	RIGHT	10	90	9800	1.4	POSITIVE	RIGHT SIDE PLEURAL EFFUSION	CLEAR	107	PREDOMINANTLY NEUTROPHILS	3.6	10	62	NORMAL
32	SUNITHA	24	F	P	A	A	P	A	A	A	P	A	P	P	P	A	A	A	LEFT	13	70	12000	0.6	POSITIVE	LEFT SIDE PLEURAL EFFUSION	AMBER	100	PREDOMINANTLY NEUTROPHILS	4.8	27	50	CIRRHOSIS WITH PORTAL HTN
33	SHRANKAPPA LOGAVI	54	M	P	P	A	P	A	A	A	P	P	P	P	P	P	P	A	RIGHT	13.7	60	19530	0.4	NEGATIVE	RIGHT SIDE PLEURAL EFFUSION	CLEAR	150	PREDOMINANTLY LYMPHOCYTES	5.2	10	115	NORMAL

34	RAJANI	60	F	A	P	A	A	A	A	A	P	P	P	P	P	A	A	P	LEFT	13.7	5	11300	0.6	POSITIVE	LEFT SIDE PLEURAL EFFUSION	AMBER	60	PREDOMINANTLY NEUTROPHILS	5	44	123	NORMAL
35	MALLIKARJUN	30	M	P	P	P	P	A	A	A	P	P	P	P	P	P	A	A	RIGHT	11	80	4500	0.8	POSITIVE	RIGHT SIDE PLEURAL EFFUSION	STRAW	54	PREDOMINANTLY NEUTROPHILS	1.4	76	88	CIRRHOSIS WITH PORTAL HTN
36	SHARANAPPA	45	M	P	A	P	P	A	A	A	P	P	P	A	A	P	A	A	LEFT	12.9	25	29170	0.7	NEGATIVE	LEFT SIDE PLEURAL EFFUSION	CLEAR	60	PREDOMINANTLY LYMPHOCYTES	4	48	55	NORMAL
37	BASAVARAJ	32	M	P	A	P	P	A	A	A	P	A	P	P	P	A	A	A	RIGHT	10	120	6880	1.8	NEGATIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	38	PREDOMINANTLY NEUTROPHILS	5.2	61	76	NORMAL
38	SHIVALEELA	36	F	P	A	A	P	A	A	A	P	P	P	P	P	P	A	A	RIGHT	10.3	100	6470	1	NEGATIVE	RIGHT SIDE PLEURAL EFFUSION	CLEAR	12	PREDOMINANTLY LYMPHOCYTES	2.4	98	74	NORMAL
39	SIDRAM	40	M	A	P	A	P	A	A	A	P	P	P	P	P	A	A	P	BILATERAL	11	75	6870	1.7	POSITIVE	BILATERAL EFFUSION	AMBER	57	PREDOMINANTLY LYMPHOCYTES	4.9	95	78	NORMAL
40	SIDRAMMAPPA	55	M	P	A	P	A	A	A	A	P	P	P	P	P	A	A	A	LEFT	11.4	50	10330	2	POSITIVE	LEFT SIDE PLEURAL EFFUSION	HEMORRHAGIC	21	PREDOMINANTLY NEUTROPHILS	3	10	89	NORMAL
41	SHANTABAI	60	F	P	A	P	P	A	A	A	P	P	P	P	P	A	A	A	RIGHT	13.8	40	10730	7.9	POSITIVE	RIGHT SIDE PLEURAL EFFUSION	CLEAR	17	PREDOMINANTLY LYMPHOCYTES	4.8	115	101	NOT DONE
42	BASAVARAJ	38	M	P	A	A	P	P	P	P	A	P	P	P	A	A	A	LEFT	10	120	6880	2.3	NEGATIVE	LEFT SIDE PLEURAL EFFUSION	AMBER	38	PREDOMINANTLY LYMPHOCYTES	5.2	61	76	NORMAL
43	REKHA METI	30	F	A	P	A	A	A	A	A	P	P	P	P	P	A	A	A	RIGHT	12.6	100	24090	1	NEGATIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	51	PREDOMINANTLY LYMPHOCYTES	3.4	89	25	NORMAL
44	SATISH	48	M	P	A	P	P	A	A	A	P	P	P	P	P	A	A	A	LEFT	11	90	20000	1	NEGATIVE	LEFT SIDE PLEURAL EFFUSION	CLEAR	23	PREDOMINANTLY LYMPHOCYTES	6.1	70	40	NORMAL
45	ANNAPURNA	35	F	P	A	P	A	A	A	A	P	P	P	P	P	A	A	P	RIGHT	12.2	20	23290	0.6	POSITIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	10	PREDOMINANTLY NEUTROPHILS	1	124	10	MILD HEPATOMEGALY
46	REKHA M	67	F	P	A	A	P	A	A	A	P	P	P	P	P	P	P	A	BILATERAL	12.6	50	6070	9.2	POSITIVE	BILATERAL EFFUSION	HEMORRHAGIC	51	PREDOMINANTLY LYMPHOCYTES	3.4	89	15	NORMAL
47	ARUNKUMAR DESAI	56	F	A	P	A	P	P	P	A	P	P	P	P	P	P	A	A	RIGHT	13	45	7700	0.5	NEGATIVE	RIGHT SIDE PLEURAL EFFUSION	CLEAR	16	PREDOMINANTLY LYMPHOCYTES	7.4	53	23	NORMAL
48	BHARATHI	45	F	P	P	P	P	A	A	A	P	A	P	P	P	P	A	A	LEFT	4.3	40	7510	2	POSITIVE	LEFT SIDE PLEURAL EFFUSION	AMBER	6	PREDOMINANTLY NEUTROPHILS	1.7	91	11	CIRRHOSIS WITH PORTAL HTN
49	SHARANAPPA	54	M	P	P	P	P	P	P	P	P	P	A	A	A	A	P	RIGHT	6	100	10000	1.2	POSITIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	6.2	PREDOMINANTLY LYMPHOCYTES	5.7	328	22	NORMAL
50	SUNITA	55	F	P	A	P	A	A	A	A	P	P	P	P	P	A	A	A	RIGHT	12	60	8800	2	POSITIVE	RIGHT SIDE PLEURAL EFFUSION	CLEAR	4.1	PREDOMINANTLY LYMPHOCYTES	1.1	84	45	NORMAL
51	SUDHA M K	49	M	A	A	A	P	A	A	A	P	P	P	P	P	A	A	A	RIGHT	13.2	50	17330	0.8	POSITIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	23	PREDOMINANTLY LYMPHOCYTES	2.6	269	14	NORMAL
52	GURAPPA S R	59	M	P	A	P	P	A	A	A	P	P	P	P	P	P	A	A	LEFT	14.8	10	10700	0.7	POSITIVE	LEFT SIDE PLEURAL EFFUSION	AMBER	11	PREDOMINANTLY LYMPHOCYTES	3.3	83	20	NORMAL
53	KASAPPA	56	M	P	P	P	P	P	P	A	P	A	P	A	P	P	P	A	RIGHT	12.5	30	7600	0.6	POSITIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	47	PREDOMINANTLY LYMPHOCYTES	5.8	99	34	NORMAL
54	LAKAPPA	37	M	P	A	P	A	A	A	A	P	P	P	A	A	P	A	A	RIGHT	10	90	9800	0.8	NEGATIVE	RIGHT SIDE PLEURAL EFFUSION	CLEAR	107	PREDOMINANTLY NEUTROPHILS	3.6	10	83	CIRRHOSIS WITH PORTAL HTN
55	BASAGOND	27	M	A	A	P	P	A	A	A	P	P	P	P	P	A	A	P	RIGHT	12.5	25	10460	0.9	POSITIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	64	PREDOMINANTLY LYMPHOCYTES	4.6	67	82	NORMAL
56	ISHWAR	18	M	P	A	P	P	A	A	A	P	P	P	P	P	P	A	A	RIGHT	13.2	80	11610	0.6	POSITIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	63.3	PREDOMINANTLY LYMPHOCYTES	5.9	91	10	NORMAL
57	SIDAMMA S	39	M	P	A	P	P	A	A	A	P	A	P	P	P	A	A	A	LEFT	11.5	50	9820	0.4	POSITIVE	LEFT SIDE PLEURAL EFFUSION	AMBER	18	PREDOMINANTLY NEUTROPHILS	2.7	23	124	NORMAL
58	ABBASBER	50	M	P	P	A	A	A	A	A	P	P	P	A	A	P	A	A	RIGHT	13	90	7860	0.9	NEGATIVE	RIGHT SIDE PLEURAL EFFUSION	AMBER	133	PREDOMINANTLY LYMPHOCYTES	5.5	91	50	NORMAL
59	SHIVAGONDAPPA	59	M	A	A	P	P	P	P	A	P	P	P	A	P	A	A	A	LEFT	10.5	10	11920	0.6	NEGATIVE	LEFT SIDE PLEURAL EFFUSION	AMBER	6.9	PREDOMINANTLY LYMPHOCYTES	2	130	50	NORMAL
60	MARUTHI T B	40	M	P	P	P	P	P	P	A	P	A	P	P	P	P	P	A	RIGHT	15.8	5	11350	1.4	NEGATIVE	RIGHT SIDE PLEURAL EFFUSION	PALE YELLOW	18	PREDOMINANTLY LYMPHOCYTES	2.1	91	50	NORMAL