

**PLEURAL FLUID CHOLESTEROL AND LACTATE
DEHYDROGENASE TO DIFFERENTIATE EXUDATES AND
TRANSUDATE AND COMPARING IT WITH LIGHT'S CRITERIA**

**Dissertation submitted to
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In partial fulfillment of regulations

**For award of the degree of
M.D (GENERAL MEDICINE)**

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BONAFIDE CERTIFICATE

This is to certify that dissertation named “**PLEURAL FLUID CHOLESTEROL AND LACTATE DEHYDROGENASE TO DIFFERENTIATE EXUDATES AND TRANSUDATE AND COMPARING IT WITH LIGHT’S CRITERIA**” is a bonafide work performed by DR.R.B.S.MANIAN, post graduate student, Department of Internal Medicine, Kilpauk Medical College, Chennai-10, under my guidance and supervision , in fulfillment of regulations of the Tamil Nadu Dr. M.G.R Medical University for the award of M.D. Degree Branch I (General Medicine) during the academic period from 2013 to 2016.

Prof.Dr.T.S SANTHI,M.D.,
M IV Unit Chief
Government Royapettah Hospital.
Kilpauk Medical College.

Prof.Dr.S.MAYILVAHANAN,M.D
Professor of medicine,
Guide for the study
Government Royapettah hospital ,
Kilpauk Medical College.

Prof.Dr.S.USHALAKSHMI,MD,FMCC
Professor and Head of the Department
Department of Medicine,
Kilpauk Medical College.

Prof. Dr. R.NARAYANA BABU,M.D,DCH.
THE DEAN,
KILPAUK MEDICAL COLLEGE,
CHENNAI-10

DECLARATION

I solemnly declare that this dissertation named “**PLEURAL FLUID CHOLESTEROL AND LACTATEDEHYDROGENASE TO DIFFERENTIATE EXUDATES AND TRANSUDATE AND COMPARING IT WITH LIGHT’S CRITERIA**” was prepared by me at Government Kilpauk Medical College, Chennai, under the guidance and supervision of Dr.S.MAYILVAHANAN,M.D., Professor, Department of Internal Medicine, Government Royapettah Hospital, Chennai.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the University regulations for the award of the degree of M.D. Branch I (General Medicine).

Place: Chennai

Date:

(Dr. R.B.S MANIAN)

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ABBREVIATIONS

LDH -LACTATE DEHYDROGENASE

SD - STANDARD DEVIATION

CT - COMPUTED TOMOGRAPHY

SLE - SYSTEMIC LUPUS ERYTHEMATOSIS

CONTENTS

1. INTRODUCTION

2. AIM AND OBJECTIVES

3. REVIEW OF LITERATURE

4. MATERIALS AND METHODS

5. OBSERVATION AND RESULTS

6. DISCUSSION

7. CONCLUSION

8. LIMITATIONS OF THE STUDY

9. BIBLIOGRAPHY

10. ANNEXURES

PROFORMA

ETHICAL COMMITTEE APPROVAL CERTIFICATE

PLAGIARISM CERTIFICATE AND RECIEPT

MASTER CHART

INTRODUCTION

Pleural effusion is one of the common clinical disorders encountered in the medical wards. In a patient with pleural effusion diagnosis can be arrived by history, clinical examination and radiological techniques. Pleural effusion occurs due to various etiologies.

Pleural effusion is of two different types depending on pathophysiology. They are Transudate and Exudate. To find out etiology, first step is to differentiate whether the pleural effusion is of exudate or transudate type. Etiology of pleural effusions differs in different parts of the world.

In Indian scenario, infectious causes of pleural effusion particularly tuberculosis and pneumonic consolidation leading on to parapneumonic effusion will be the leading cause of pleural effusion.

Though worldwide the incidence of empyema, has come down due to early initiation of antibiotic therapy, in India still many cases of empyema and loculated pleural effusion are encountered in tertiary care hospitals.

After diagnosing pleural effusion in a patient, thoracentesis should be done under strict aseptic precautions and after getting concurrence from the patient. Pleural fluid aspirate is analysed for various parameters like protein, glucose, LDH etc.

Pleural fluid protein was used to differentiate exudate and transudate, which later on was found to be less sensitive. Then Light et al established diagnostic criteria to separate exudate and transudate in 1972. They used serum protein, serum LDH, pleural fluid protein and pleural fluid LDH, all these four parameters to differentiate exudative and transudate pleural effusion. Since then Light's criteria is used as the most accepted method to differentiate between exudate and transudate pleural effusion.

Light et al established their criteria with sensitivity and specificity of 99% and 98%. But other community studies cannot reproduce this much of results and they showed results with lesser specificity of around 70-86%.

Then newer parameters like ADA, interferon gamma assay, ANA, Rheumatoid factor, tumor markers etc. are analysed in the pleural fluid to separate exudates and transudate and they helped to arrive at an etiological diagnosis very easily.

Apart from routine pleural fluid analysis of protein and LDH, various researchers studied role of various parameters to find out the set of indicators which will be simple and cost effective. Many newer investigations for diagnosis of pleural effusion are in pipeline but nothing has been widely accepted among the clinicians.

Various authors extended their work on establishing newer diagnostic tests to separate exudate and transudate. Pleural fluid cholesterol in isolation and in combination with other parameters was tried in many studies. Pleural fluid cholesterol and lactate dehydrogenase test was found to be the best alternative parameter and was in comparable to Light's criteria in differentiating exudate and transudate.

So this study 'PLEURAL FLUID CHOLESTEROL AND LACTATE DEHYDROGENASE TO DIFFERENTIATE EXUDATES AND TRANSUDATE AND COMPARING IT WITH LIGHT'S CRITERIA' is taken to assess the usefulness of this test in differentiating pleural effusion into exudate and transudate and its comparison with established Light's criteria.

AIMS AND OBJECTIVES

AIM

To find the role of pleural fluid cholesterol and Lactate dehydrogenase in differentiating exudate and transudate.

OBJECTIVES:

- To find the diagnostic value of pleural fluid Cholesterol and Lactate dehydrogenase in differentiating exudative and transudative effusion and is compared with Light's criteria.
- To find the place of pleural fluid cholesterol and Lactate dehydrogenase in diagnostic algorithm of pleural effusion.

REVIEW
OF
LITERATURE

REVIEW OF LITERATURE

PLEURAL ANATOMY ¹:

Pleural cavity is derived from the primitive body cavity called coelom, which in turn is derived from primitive mesoderm of embryo.

The pleura have two membranes:

- Inner visceral pleura cover the lung surface and inter lobar fissures.
- Outer parietal pleura which lies outside the visceral pleura and it covers thoracic cavity's inner surface, diaphragm and mediastinum.

The part of parietal pleura that covers inner surface of the ribs and intercostal muscles is called costal pleura, the one which covers the convex surface of the diaphragm is called diaphragmatic pleura. Cervical pleura is the one which extends into the neck and the part of the parietal pleura which covers mediastinum is called mediastinal pleura ².

Surface of the pleura is glistening, semitransparent and wet. The potential space between these two membranes is named as pleural space.

PLEURAL SPACE:

Pleural space is slit like which is filled with a thin layer of fluid, called pleural fluid. The function of this pleural fluid is mainly lubrication so as to reduce and eliminate friction forces. It allows movement of the lung with the chest wall during inspiratory and expiratory movements.

FUNCTIONAL ANATOMY OF PLEURA ³:

Whenever pleura are examined in light microscopy it is found to have five layers.

Five layers of pleura are:

- (i) The mesothelium
- (ii) Submesothelial connective tissue
- (iii) Layer of elastic tissue
- (iv) Another layer of loose connective tissue containing blood and lymph vessels and nerves.
- (v) Deep fibroelastic layer adherent to the underlying tissue.

The two important functions that the connective tissue layer in the visceral pleura has:

- (a) Elastic recoil of the lung, in expelling air from the lung,
- (b) Restricting the volume of the lung to be inflated, thereby protecting the lungs from over inflation.

MESOTHELIAL CELLS OF PLEURA:

Mesothelial cells covers surface of both parietal and visceral pleura. These cells are polygonal in shape. Surface of mesothelial cell is bushy and are 0.1 micrometer in diameter and 3 micrometer in length. These mesothelial cells are metabolically active cells⁴. There exists tight junction between these mesothelial cells.

BLOOD SUPPLY OF PLEURA ⁵:

Parietal pleural Blood supply:

Parietal pleural blood supply is very rich and supplied mainly by systemic vessels.

- ❖ Intercostal and internal mammary vessels supply costal pleura.
- ❖ Subclavian arteries supply cervical pleura.
- ❖ Bronchial, internal mammary, mediastinal vessel supply mediastinal pleura.
- ❖ Superior phrenic branch of internal mammary artery, posterior mediastinal artery which is from aorta in thorax, inferior phrenic artery a branch from abdominal aorta –all these supply diaphragmatic pleura.

VENOUS DRAINAGE OF PARIETAL PLEURA:

Venous drainage of parietal pleura is mainly to azygous vein. From there, it drains to superior vena cava. From diaphragmatic pleura some of the venous blood drains to inferior vena cava also.

VISCERAL PLEURAL BLOOD SUPPLY:

In human being visceral pleura is thick and the main blood supply is from bronchial artery.

VENOUS DRAINAGE OF VISCERAL PLEURA:

Venous blood from visceral pleura is drained mainly through pulmonary veins.

SURFACE ANATOMY OF PLEURA

At the apices of the upper lobe and along the inner margins, the pleura lies very close to the lung. It rises 2-3 cm above the clavicle.

In the lower aspect, pleura extends below the lung 4 to 5 cm anteriorly and 9-10cm posteriorly.

It lies at the level of 8th rib in the mid clavicular line, 10th rib in the mid axillary line, and the 12th rib at the scapular line.

LYMPHATICS OF PLEURA ⁶:

The costal pleura is surrounded by a lymphatic plexus which is mainly present in the intercostal spaces only and are absent below the ribs. These lymphatic vessels of the pleura covering the costal surface drain towards the lymph nodes those are present along the internal thoracic vessels and dorsally run towards the heads of the ribs to the internal intercostal nodes.

From mediastinal pleura, the lymphatic vessels pass to the tracheobronchial and mediastinal nodes. The lymphatic drainage of the pleura that cover the diaphragm, pass to the parasternal, posterior mediastinal nodes and middle phrenic nodes.

The parietal pleural lymphatic drainage plays a vital role in the formation and removal of fluid in pleural space, both in physiological and pathological condition.

There are abundant lymphatic vessels in visceral pleura. These visceral pleural lymphatics form a plexus that intercommunicate with all vessels that run on the surface of the lung and they run towards the hilum and by penetrating the lung, they join the bronchial lymph vessels and then pass via the interlobular septa.

INNERVATION OF THE PLEURA⁷:

PARIETAL PLEURAL INNERVATION:

Nerve endings that are present in the costal and diaphragmatic parietal pleura are sensory nerve endings. The somatic type intercostal nerves supply the pleura which covers the costal surface and also the outer part of the pleura covering the diaphragm. So when these areas are stimulated, person will complain of pain in the adjacent chest wall mainly.

Phrenic nerve mainly innervates the central part of the diaphragm and pain is perceived in the ipsilateral shoulder if the central part of diaphragm is stimulated or irritated.

VISCERAL PLEURAL INNERVATION:

- (i) There are no pain nerve fibers for visceral pleura and so it is pain insensitive. It can be manipulated without any unpleasant sensation.
- (ii) Vagus nerve by its pulmonary branches and sympathetic trunk supplies visceral pleura.

Therefore, the main source of catchy pain is due to disease process that causes inflammatory reaction in the parietal pleura like tuberculous pleuritis, rheumatoid pleuritis etc.

PLEURAL FLUID:

***VOLUME*⁸:**

In physiological state, very minimal pleural fluid is present in between the two pleura. The mean amount of pleural fluid in the pleural space in normal human being is 8.4 ± 4.3 ml. The quantity of fluid in both the pleural spaces is similar. While expressing per kg of body mass, a non-smoking, normal individual has the total pleural fluid volume of 0.26 ± 0.1 ml per Kg.

CELLS IN PLEURAL FLUID:

In humans, normally pleural fluid contains 75% macrophages and 25% are lymphocytes. Less than 2% of the cells that are present in pleural fluid are mesothelial cells, neutrophils and eosinophils.

BIOCHEMICAL FACTORS:

Protein:

- Normally in humans, pleural fluid contains 1 to 2 gm. /dl of protein.
- Hco_3^+ : Normal level is 20 -25% higher than plasma.
- K^+ : Identical to plasma.
- Na^+ : 3-5% less than plasma.
- Glucose: Identical to plasma.

These electrolyte gradients suggest that an active process is involved in formation of pleural fluid.

PLEURAL FLUID PRESSURE AND DYNAMICS^{9,10:}

Pleural fluid movement within pleural membranes is mainly based on the balance of hydrostatic and oncotic pressure that exists between the microvasculature and the pleural space. Starling's Law describes the fluid movement across the pleural membranes.

FORMATION OF PLEURAL FLUID:

Pleural fluid is formed in the following manner and enters into the pleural cavity from,

- The pleural capillaries
- The interstitial spaces of the lung
- The intrathoracic lymphatics
- The intrathoracic blood vessels
- The peritoneal cavity

PLEURAL CAPILLARIES^{9:}

A gradient for fluid formation is normally present in the parietal pleura. In the parietal pleura the hydrostatic pressure is nearly 30 cm H₂O. The pleural pressure is approximately - 5 cm H₂O. So the net hydrostatic pressure will be $30 - (-5) = 35$ cm H₂O. This will favour the movement of fluid from the capillaries in the parietal pleura to the pleural space.

There is an oncotic pressure gradient opposing this hydrostatic pressure. This oncotic pressure in the plasma is nearly 34 cm H₂O.

Normally, the pleural fluid contains a small amount of protein. It has an oncotic pressure of approximately 5 cm H₂O and so it will yield a net oncotic pressure gradient of $34 - 5 = 29$ cm H₂O. This net gradient is $35 - 29 = 6$ cm H₂O, which favours the movement of pleural fluid to the pleural space from the capillaries in the parietal pleura.

As the parietal pleura has systemic blood supply, it is found to be the source of normal pleural fluid. Moreover the parietal pleural microvessels will lie closer to the pleural space. The parietal pleural blood vessels have high micro vascular pressure as they drain into and are in continuity with systemic venules.

PLEURAL FLUID FROM INTERSTITIUM:

In diseased conditions like congestive heart failure¹⁰, the pleural fluid originates from the interstitial spaces of the lung. Most of the fluid from the interstitial space of the lung will be cleared through the pleural space. The volume of pleural fluid collected will be directly proportional to elevated wedge pressure. As the interstitial fluid increases, the sub pleural interstitial pressure will increase and the fluid will move via thick visceral pleura to the pleural space.

FROM PERITONEAL CAVITY¹¹:

Pleural fluid accumulation can occur if there is free fluid in the peritoneal cavity and if there are openings in the diaphragm. Under these conditions, the fluid will flow from the peritoneal space to the pleural space because the pressure in the pleural cavity is less than the pressure in the peritoneal cavity.

THORACIC DUCT INJURY:

If the thoracic duct is disrupted, lymph will accumulate in the pleural space, producing a chylothorax. The rate of fluid accumulation with chylothorax can be more than 1,000 mL/day.

PLEURAL FLUID ABSORPTION:

- Lymphatic clearance
- Clearance via capillaries in visceral pleura.

LYMPHATIC CLEARANCE¹²

The amount of fluid that can be cleared through these lymphatics is substantial. Clearance of pleural fluid through the pleural lymphatics explains the reason for fluid not accumulating in the pleural space in normal individuals. The pleural space has very good communication with the lymphatic vessels that are present in the parietal pleura, by means of stomas. Such stomas are not seen in the

visceral pleura. Cells, proteins and all the particulate matters are removed from the pleural space by the lymphatics in the parietal pleura.

Lymphatics in the pleural space have the ability to work to a maximum whenever excess fluid accumulates in the pleural cavity. Lymphatics have nearly 28 times higher capacity to remove pleural fluid than they do during normal fluid formation.

VISCERAL CAPILLARIES:

Previously it was thought that the main route of exit of pleural fluid from pleural space is through visceral pleural capillaries. As visceral pleura in human beings is very thick later it was found that, most of the pleural fluid gets absorbed via lymphatics in the parietal pleura.

PATHOGENESIS OF PLEURAL EFFUSION FORMATION:

The basic pathogenesis in the accumulation of the excess pleural fluid which leads to the formation of the pleural effusion are:

- Formation of increased pleural fluid.
- Decreased absorption of the pleural fluid.

(I). FORMATION OF INCREASED PLEURAL FLUID:

A. INCREASED LUNG INTERSTITIAL FLUID¹³

- Pneumonic consolidation
- Pulmonary vascular embolus
- Left ventricular failure

B. INCREASED PRESSURE IN INTRAPLEURAL VESSELS:

When there is increased gradient between intrapleural and intravascular pressure, there is a tendency towards formation of increased pleural fluid.

- Right or left ventricular failure.
- Superior vena caval syndrome.

C. INCREASED CAPILLARY PERMEABILITY:

- Inflammation of the pleura
- Elevated vascular endothelial growth factor level.

D. INCREASED PROTEIN LEVEL IN PLEURAL FLUID

E. DECREASED LEVEL OF PLEURAL PRESSURE

- Atelectasis of lung.
- Increased elastic recoil of the lung.

F. *LARGE VOLUME OF FLUID IN PERITONEAL CAVITY*

- Peritoneal dialysis.
- Ascites

G. *THORACIC DUCT DISRUPTION*

H. *THORACIC BLOOD VESSELS DISRUPTION*

(II) DECREASED ABSORPTION OF PLEURAL FLUID:

A. *PARIETAL PLEURAL LYMPHATICS OBSTRUCTION*

The most common cause of a decrease in pleural fluid absorption is obstruction of the lymphatics draining the parietal pleura. Normally, the lymphatic flow from the pleural space is approximately 0.01 mL/kg/hour or 15 mL/day, because this is the amount of pleural fluid formed. However, the capacity of the lymphatics is approximately 0.20 mL/kg/hour or 300 mL/day. Lymphatic blockade is an important factor that contributes to the development of a malignant pleural effusion.

B. INCREASED PRESSURE OF SYSTEMIC VASCULATURE.

- Superior venacava syndrome.
- Right heart failure.

PLEURAL EFFUSION CAUSES:

Pleural fluid that gets accumulated due to pathological reasons may be either an exudates or a transudate.

TRANSUDATIVE PLEURAL EFFUSION¹⁴

1. CONGESTIVE HEART FAILURE
2. NEPHROTIC SYNDROME.
3. HEPATIC HYDROTHORAX.
4. PERITONEAL DIALYSIS
5. PULMONARY EMBOLISM
6. SUPERIOR VENA CAVA OBSTRUCTION
7. MYXEDEMA
8. URINOTHORAX
9. AMYLOIDOSIS
10. SARCOIDOSIS
11. LEAKAGE OF CEREBROSPINAL FLUID INTO PLEURAL SPACE
12. ATELECTASIS
13. CENTRAL LINE MISPLACEMENT.

EXUDATIVE PLEURAL EFFUSION¹⁵

1. MALIGNANT CAUSES:

- i. Mesothelioma
- ii. Lymphoma
- iii. Metastasis to lung and pleura

2. INFECTIOUS DISEASES:

- i. Tuberculosis
- ii. Bacterial pneumonia
- iii. Invasive fungal disease
- iv. Viral infection
- v. Parasitic disease

3. EMBOLI IN PULMONARY VASCULATURE.

4. ABDOMINAL CONDITIONS

- i. Oesophageal perforation
- ii. Acute and chronic pancreatitis
- iii. Abdominal abscesses
- iv. Diaphragmatic hernia
- v. Post abdominal surgery
- vi. Variceal sclerotherapy
- vii. Hepatic transplantation

5. COLLAGEN VASCULAR DISEASES

- i. Rheumatoid pleuritis
- ii. SLE
- iii. Wegener's granulomatosis
- iv. Churg-Strauss syndrome

6. POST CORONARY ARTERY BYPASS GRAFT OPERATION

7. ASBESTOS RELATED LUNG DISEASES

8. YELLOW NAIL SYNDROME

9. UREMIC PLEURAL DISEASE

10. SARCOID LUNG

11. MEIGS SYNDROME

12. DRUG INDUCED

13. TRAPPED LUNG SYNDROME.

14. RADIATION EXPOSURE

15. HEMOTHORAX

16. IATROGENIC INJURY

17. OVARIAN HYPERSTIMULATION SYNDROME

18. DISEASES OF PERICARDIUM

19. CHYLOTHORAX

PLEURAL EFFUSION DUE TO DRUGS¹⁶:

The pathogenesis of pleural effusion due to drugs is not understood well.

The probable mechanism is

- i) Mesothelial injury due to oxidants
- ii) Acute hypersensitivity reactions
- iii) Direct dose related toxicity.

Following drugs which are used commonly will lead on to pleural effusion.

- a. Nitrofurantoin
- b. Clozapine
- c. Methotrexate
- d. Interleukin
- e. Dantrolene
- f. Methysergide
- g. Bromocriptine
- h. Procarbazine
- i. Minoxidil
- j. Amiodarone.
- k. Interferons.

EVALUATION OF THE PATIENT WITH PLEURAL EFFUSION¹⁷

Patients with pleural effusion may be asymptomatic or symptomatic. For proper evaluation of the various causes for pleural effusion the following details are needed.

- i) Detailed history
- ii) Physical examination
- iii) Diagnostic radiology
- iv) Pleural fluid analysis

HISTORY

If the patients are symptomatic the common symptoms will be chest pain, breathlessness and dry cough. If there is any underlying parenchymal disease, then patient will complaint of productive cough and fever.

Pleuritic chest pain¹⁸ is described by the patient mainly as shooting pain or stabbing pain which is increased on deep inspiration, coughing or sneezing. This pleuritic chest pain radiates to the shoulder if the central part of the diaphragmatic pleura is inflamed. If costal pleura is involved it is more of a localized pain.

BREATHLESSNESS:

Patient's dyspnea in pleural effusion is due to decreased movement of the thoracic wall due to chest pain, collapse of the underlying lung or due to underlying parenchymal disease such as pneumonia.

DRY COUGH:

Dry cough is due to pleural inflammation or the compression of the lung by the fluid will bring the opposing bronchial walls into contact so that stimulating cough reflex.

ASYMPTOMATIC PATIENTS WITH PLEURAL EFFUSION

- a. Nephrotic syndrome
- b. Rheumatoid pleurisy
- c. Yellow nail syndrome
- d. Trapped lung
- e. Urinothorax
- f. Peritoneal dialysis
- g. Benign asbestos related pleural effusion

PATIENTS PRESENTING WITH TYPICAL SYMPTOMS

- a. Bacterial pneumonia
- b. Lupus pleuritis
- c. Pulmonary embolism
- d. Carcinomatous pleural effusion
- e. Malignant mesothelioma

PHYSICAL EXAMINATION ¹⁹

On examination of the patient, the physical signs of effusion will depend on

- a) Volume of the pleural fluid
- b) Underlying lung compression
- c) Patency of bronchus

Whenever about 500ml of pleural fluid is there, the findings will be

- a) Percussion dullness
- b) Decreased intensity of breath sounds
- c) Decreased fremitus.

When more than 1000ml is there

- a) Bulging of the intercostal spaces.
- b) Decreased expansion of chest
- c) Dullness upto scapula
- d) Egophony at the upper level of pleural effusion.
- e) Absent vocal and tactile fremitus.

With massive pleural effusion

- a) Bronchial breath sounds at the apex.
- b) Liver and Spleen palpable due to depressed diaphragm.
- c) Totally absent breath sounds in entire hemithorax.
- d) Egophony at upper level of effusion.
- e) Bulging of intercostal spaces.

DIAGNOSIS OF PLEURAL EFFUSION BY RADIOLOGY

The radiological tests used are

- a) Chest x ray
- b) Ultrasonogram chest
- c) CT-Chest.

CHEST X RAY:

The postero-anterior view, Lateral view, lateral decubitus view are the various views used to find out pleural fluid accumulation.

- a) Whenever 175ml of fluid gets accumulated, lateral costophrenic sulcus is blunted, but there are chances that even with 500ml fluid, there won't be any blunting.
- b) Costophrenic and cardiophrenic angles are obliterated.
- c) Fluid may get collected in the interlobar fissures which will disappear on treatment and is called as vanishing tumour or phantom tumour.

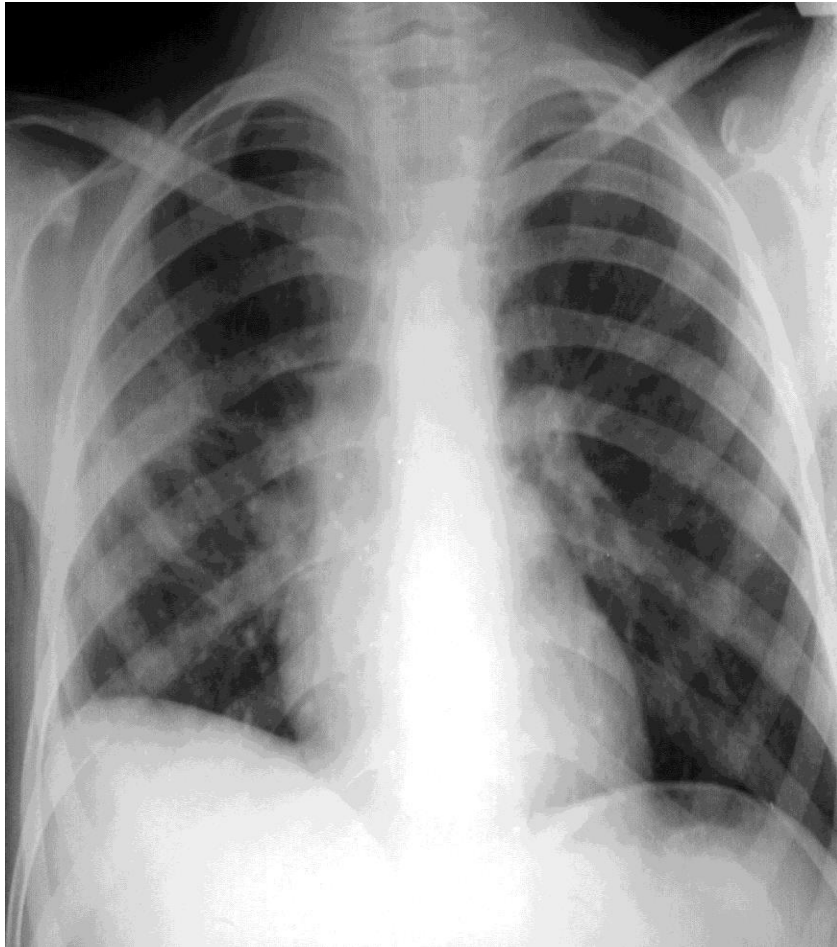
MENISCUS SIGN

In chest x ray²⁰ postero anterior view, as the fluid increases in amount **MENISCUS SIGN** is seen. It is nothing but homogeneous opacity in lower zone with concave upward sloping which extends more laterally than medially.



SUBPULMONIC EFFUSION²¹

Pleural fluid will get accumulated in between diaphragm and lung surface and is called as subpulmonic effusion. Chest x ray features will be elevation of the ipsilateral hemidiaphragm, flattening of medial aspect and lateral peaking of the hemidiaphragm



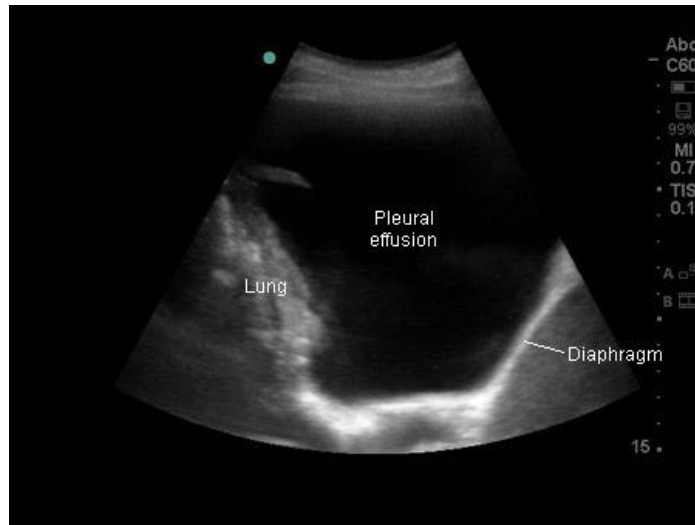
LOCULATED EFFUSION

Sometimes effusion is of loculated type, in which it is D shaped along lateral chest wall without obliterating costophrenic angle.



ULTRASONOGRAM CHEST

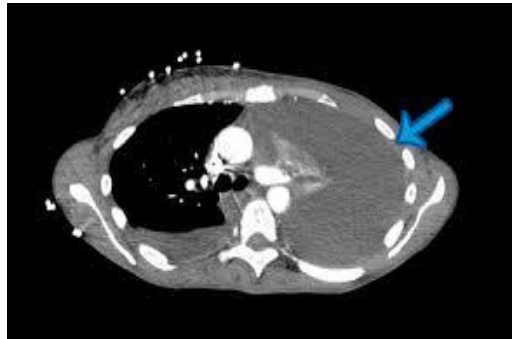
Chest ultrasound will detect very minimal fluid in pleural space. It is seen as anechoic or hypoechoic shadow which is delineated from echogenic visceral pleura.



- Ultrasound is useful in detecting small effusion.
- Loculated effusions are detected by ultrasonogram.
- Ultrasound also detects tumors associated with pleura.
- In diagnosing effusions in bedridden patients
- Septations when present will be picked up by ultrasonogram.
- Parietal and visceral pleural thickening, diaphragmatic thickening, nodules in diaphragm are detected along with effusion.
- Hemorrhagic effusion or empyema is detected as homogenous echoes.
- Ultrasonogram is useful in aspiration of minimal pleural fluid under direct guidance and for inserting chest tube in case of empyema.²²

CT-THORAX

- CT thorax is able to detect even very small effusion. CT will identify presence of inflammation clearly by the presence of thickening of visceral and parietal pleura and enhancement after injection of the contrast²³. CT also helps to find out any underlying lung parenchymal lesions.
- A pleural effusion on CT will look like dependent sickle-shaped opacity, with a lower attenuation number compared to adjacent pleural thickening or mass.



- Loculated pleural effusions will be lenticular shape and smooth margins with displacement of adjacent parenchyma. Loculated effusion which looks like a mass in chest x-ray can be clearly diagnosed with CT chest which shows homogenous attenuation.
- Also CT chest is helpful in detecting mass lesion associated with pleural effusion. Contrast enhanced CT will show pleural enhancement and is more than 80% sensitive and is also highly specific in evaluation of pleural effusion due to malignancy.

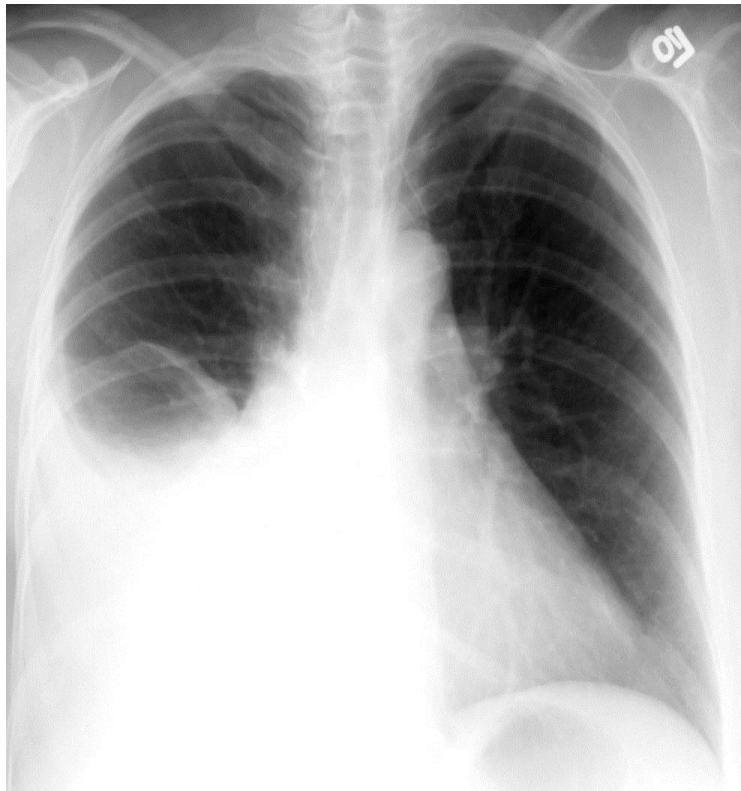
BILATERAL EFFUSION²⁴:



1. CONGESTIVE HEART FAILURE
2. MALIGNANCY.
3. RHEUMATOLOGICAL DISEASES
4. VIRAL INFECTIONS. .
5. NEPHROTIC SYNDROME.
6. EMBOLISM IN PULMONARY VESSELS.
7. EOSINOPHILIC PNEUMONIA.
8. CIRRHOSIS LIVER.
9. AMYLOIDOSIS

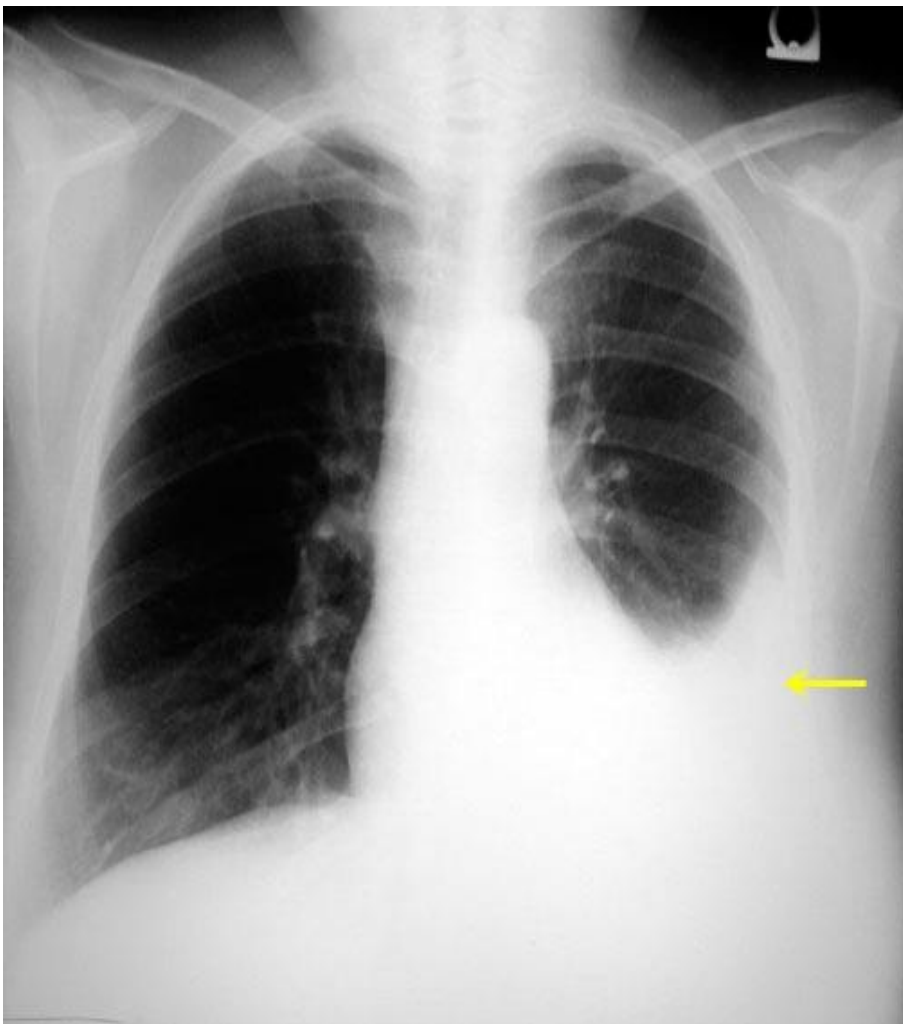
RIGHT SIDED PLEURAL EFFUSION²⁵:

1. CONGESTIVE HEART FAILURE
2. LIVER CIRRHOSIS WITH PORTAL HYPERTENSION
3. MEIGS SYNDROME
4. SUBPHRENIC ABSCESS
5. LIVER ABSCESS RUPTURE
6. THORACIC DUCT INJURY BELOW D5 LEVEL.



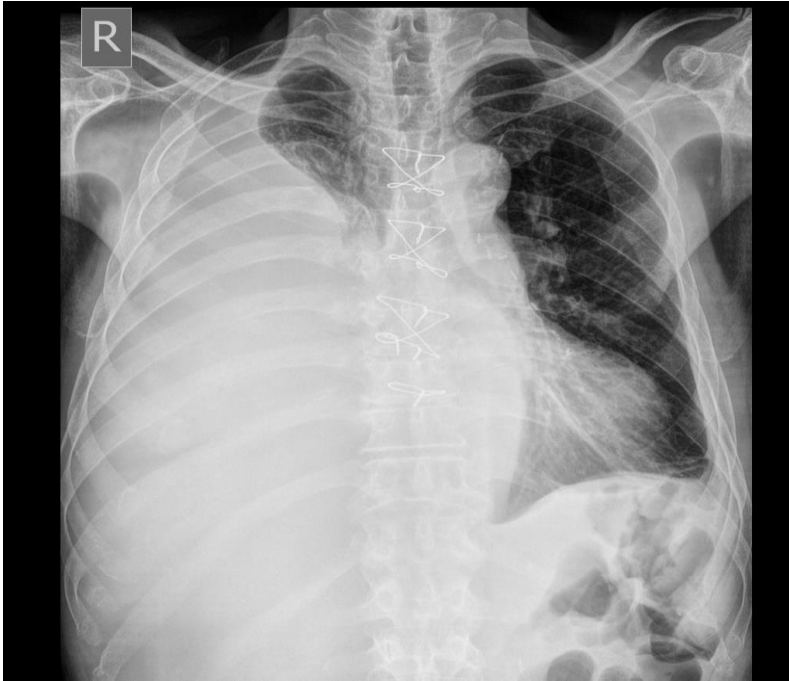
LEFT SIDED EFFUSION:

1. PANCREATITIS.
2. THORACIC DUCT INJURY ABOVE D5 LEVEL.
3. DRESSLERS SYNDROME.
4. LEFT SUBDIAPHRAGMATIC ABSCESS.
5. ESOPHAGEAL RUPTURE.



MASSIVE EFFUSION²⁶

Pleural effusion is said to be massive when it occupies entire hemithorax.



CAUSES:

1. Malignancy.
2. Meig's syndrome
3. Hepatic hydrothorax.
4. Chylothorax.
5. Hemothorax

THORACENTESIS²⁷:

Thoracentesis is defined as a drainage procedure in which needle is inserted into the pleural cavity to remove the fluid. It can be done as a bedside procedure, after explaining about it to the patient. It should be done under strict aseptic precaution. Thoracentesis is an important diagnostic procedure in a patient with pleural effusion. Pleural fluid tapping and analysis will help us to categorize as either transudate or exudate. Thoracentesis can also be done for therapeutic purpose to decrease respiratory distress in patients with massive effusion. There is insufficient data on the safety of this procedure in patients who are using anticoagulants or decreased platelet count. But studies show that it is very safe procedure, even with platelet count of less than $25000/\text{mm}^3$. It should be performed cautiously in patients who are on ventilators because of increased risk of tension pneumothorax.

COMPLICATIONS OF THORACENTESIS:

1. Pneumothorax
2. Iatrogenic infections.
3. Hemothorax.
4. Pain shock.
5. Injury to the neurovascular bundle

DIAGNOSIS OF PROBABLE ETIOLOGY OF PLEURAL EFFUSION BY GROSS APPEARANCE, ODOUR AND CHARACTER OF PLEURAL FLUID

GROSS APPEARANCE

The transudate effusion will show a clear fluid and colour will be like a straw. Exudate will be more amber like and it appears cloudy. On prolonged standing, a freshly drawn exudate effusion clots, on standing for a long time, while the transudative effusion which has less fibrin content remains in a fluid state. Bloody effusion can occur due to damage to a vessel during thoracentesis and as a result of pleural or lung biopsy.

If the fluid is red uniformly, the most probable cause is malignancy. Other conditions like tuberculosis, benign asbestos pleural effusion, leukemia, pulmonary infarction and rheumatoid pleuritis will also lead to hemorrhagic effusion.

A frank hemothorax on aspiration will be seen in trauma, invasive procedures, pleural metastasis, anticoagulation for pulmonary infarction and catamenial hemothorax.

Aspiration of white or milk like fluid is usually due to chylothorax or cholesterol effusion. Sometimes longstanding effusions of any cause may mimic chyle and is called as pseudo-chyle. It may be due to the presence of globules of fat formed from dying cells.

Brown colored fluid is due to long standing bloody fluid or amoebic liver abscess.

CHARACTER OF PLEURAL FLUID:

- Purulent fluid or gross pus is seen in frank empyema.
- Viscous fluid is seen in mesothelioma.
- Anchovy sauce like character in ruptured amoebic liver abscess.
- Turbid fluid character in inflammatory exudate.

ODOUR OF FLUID:

- Ammonia odour indicates urinothorax.
- Putrid odour will be in anaerobic empyema.

Therefore while doing thoracentesis itself, observation of the fluid color, character and odour, the probable type of pleural effusion and its possible cause can be ascertained. But further biochemical tests will be needed to confirm etiology.

PLEURAL FLUID pH²⁸:

Normally pleural fluid is of alkaline character. pH decreases in pleural fluid if there is high metabolic activity in pleural space. pH is considered low if value is less than 7.3 with a normal blood pH range. Most transudative effusion will have pH ranging from 7.45 to 7.55. But pH of most exudative effusion will be in the range of 7.30-7.45.

CAUSES OF pH less than 7.2

1. Complicated parapneumonic effusion
2. Oesophageal rupture.
3. Rheumatoid pleuritis.
4. Tuberculous pleuritis.
5. Malignancy
6. Hemothorax
7. Systemic acidosis.
8. Paragonimiasis
9. Lupus pleuritis
10. Urinothorax

Pleural fluid pH measurement will be of useful in deciding management of complicated pleural effusion. If the pH is less than 7.2 there is an indication for intercostal tube drainage. With pleural fluid pH we can determine whether invasive pleural procedure is needed or not.

PLEURAL FLUID GLUCOSE²⁹:

Pleural fluid glucose level of <60 mg/dl or pleural fluid to serum glucose less than 0.5 is called low pleural fluid glucose.

COMMON CAUSES:

1. Tuberculosis
2. Parapneumonic effusion,
3. Pleural tumors
4. Rheumatoid disease.

RARE CAUSES OF PLEURAL FLUID GLUCOSE <60mg/dl

1. Parasitic infections like paragonimiasis infection
2. Hemothorax
3. Pleuritis in SLE
4. Churg-Strauss syndrome.

The lower the pleural fluid glucose, the more chance that we are dealing with a complicated parapneumonic effusion. Low glucose in parapneumonic effusion is associated with bad prognosis and is the indication for intercostal drainage tube insertion as the prime line of treatment.

In rheumatoid pleurisy pleural fluid glucose level is less than 30 mg/dl in three fourth of patients.

PLEURAL FLUID PROTEIN:

The total amount of protein in pleural fluid was used to differentiate exudates and transudate previously. By comparing protein level in pleural fluid with that of serum protein we can diagnose exudative and transudative effusion.

Tubercular effusion almost always will have protein concentration of more than 4 g/dl. Parapneumonic effusion and effusion due to malignancy will have wide range of pleural fluid protein level.

When more than 7 gm. /dl of total protein is there in pleural fluid, we have to think of Waldenstroms macroglobulinemia and multiple myeloma.

Light's criteria uses ratio of pleural fluid to serum protein as one of its criteria to differentiate exudate and transudate effusion.

PLEURAL FLUID LDH

Pleural fluid LDH is the important parameter in separation of pleural fluid into exudates and transudate. LDH is increased in pleural fluid whenever there is a effusion due to inflammatory process. It is included as one of the parameters in routinely used Light's criteria and also in combination with other parameters like protein and cholesterol.

Pleural fluid LDH concentration of >1000 IU/L can be found in one of the following disorders

- Complicated parapneumonic effusion
- Empyema
- Pleural paragonimiasis.
- Malignancy.

PLEURAL FLUID TOTAL AND DIFFERENTIAL CELL COUNT³⁰:

Analysis of pleural fluid cell count is useful in many circumstances. If the total nucleated cell in effusion is more than 1000 per microliter, it is more in favour of exudates and in transudate it is mainly less than 1000 total nucleated cells. Pleural fluid nucleated cell count of more than 10000 is seen with parapneumonic effusion, acute pancreatitis and sub diaphragmatic abscess.

In Transudative effusion predominant cell type are mononuclear cell, lymphocytes, macrophages and mesothelial cells.

PLEURAL FLUID EOSINOPHILIA³¹:

Eosinophilic pleural effusion is the one when pleural fluid eosinophil count is more than 10% of the total nucleated cells. Interleukin-5 acts as chemotactic factor for eosinophils and it will attract eosinophils from bone marrow into the pleural cavity.

INCREASED EOSINOPHILS WITH PERIPHERAL EOSINOPHILIA

1. Hodgkin disease
2. Fungal infection
3. Paragonimiasis
4. Benign asbestos pleural effusion
5. Churg Strauss syndrome
6. Tropical eosinophilia
7. Drugs –Dantrolene, Nitrofurantoin.

PLEURAL EOSINOPHILIA WITHOUT PERIPHERAL EOSINOPHILIA:

1. Trauma
2. Pulmonary infraction
3. Pneumothorax
4. Hemothorax

PLEURAL FLUID NEUTROPHILIA

1. Pulmonary infections
2. Acute pulmonary embolism
3. Very early stage of pleural effusion

LYMPHOCYTIC PLEURAL EFFUSION³²:

When lymphocytes in fluid are more than 80% of total cells it is called as lymphocytic effusion.

1. Tuberculosis - commonest cause.
2. Malignancy
3. Chylothorax.
4. Lymphoma.
5. Yellow nail syndrome
6. Sarcoidosis
7. Rheumatoid disease

MESOTHELIAL CELLS

Pleural cavity is lined by mesothelial cells. They are present in small amount in normal pleural fluid. Mesothelial cells are uncommon in tuberculous pleural effusion. But patients with AIDS may have increased amount of mesothelial cells when they develop tuberculous pleural effusion.

Mesothelial cells paucity is seen in following conditions

1. Empyema
2. Chemical pleurodesis
3. Rheumatoid pleuritis
4. Chronic malignant effusion.

LIPID ANALYSIS

Analysis of pleural fluid triglyceride is must for the diagnosis of a suspected chylothorax. Pleural fluid triglyceride level of more than 110mg/dl will strongly support the diagnosis of chylothorax³³. If triglyceride level is less than 50mg/dl, chances of chylothorax are less.

If triglyceride level is between 50-110mg/dl the next step is to do lipoprotein electrophoresis to detect presence of chylomicrons. Presence of chylomicrons in lipoprotein analysis is diagnostic of chylothorax

INTERFERON –GAMMA³⁴

CD4+ T cells produce Interferon –gamma which migrates into the pleural cavity. It is mainly used in diagnosing of tuberculous pleural effusion. Interferon-gamma seems to be a useful defence mechanism. Interferon-gamma increases polymyristate acetate-induced hydrogen peroxide production in macrophages, so that facilitating elimination of intracellular parasites. This lymphokine will also inhibit mycobacterial growth in monocytes. Interferon gamma more than 140pg/ml is significant for the diagnosis of tuberculous pleural effusion.

ADA IN PLEURAL FLUID³⁵

Adenosine deaminase is important in the degradation of purine and is also needed for lymphocyte differentiation. It is involved in maturation of monocyte-macrophage lineage. In tuberculous effusions ADA level is higher than other exudative pleural effusion.

The cut off level between 40 to 60 U/L is used with levels above indicating tuberculous pleural effusion. The sensitivity and specificity of ADA level in pleural fluid for the diagnosis of tuberculous pleural effusion is more than 90%.

CAUSES OF ELEVATED ADA

1. Tuberculous pleurisy
2. Rheumatoid pleurisy
3. Empyema
4. After coronary bypass surgery
5. Malignancy

RARE CAUSES OF ELEVATED ADA

1. Brucellosis
2. Q fever

ADA 1 and ADA 2 are the two isoenzymes in which ADA 2 is of monocyte and macrophage origin.

PLEURAL FLUID CHOLESTEROL³⁶

Cholesterol level in pleural effusion is studied by many authors to differentiate exudate and transudate. Cholesterol in pleural effusion is thought to be derived by

1. Degenerating cells
2. Increased permeability

Cholesterol is synthesized by cells lining the pleura, depending on metabolic activity and needs. Also cholesterol in pleural fluid increases by degenerated leucocytes and erythrocytes. Increased vascular permeability of pleural capillaries also lead to increased pleural fluid cholesterol levels.

Hamm and his coworkers studied about cholesterol in differentiating transudate and exudate and found it to be simple and cost effective.

Cholesterol cut off value of more than 45mg/dl is taken in studies to differentiate exudative and transudative effusion. Pleural fluid cholesterol of more than 200mg/dl suggests chyloform effusion.

PLEURAL FLUID AMYLASE³⁷:

The amylase rich pleural effusion is defined as a pleural fluid-serum amylase ratio more than 1 or increased amylase in pleural fluid more than upper limit of normal for serum amylase.

COMMON CAUSES:

1. Pancreatic disease,
2. Carcinoma
3. Esophageal rupture.

RARE CAUSES:

1. Pneumonia
2. Ruptured ectopic pregnancy
3. Cirrhosis
4. Hydronephrosis

IMMUNOLOGICAL TESTS^{38,39}

The two diseases where the immunological tests are useful are systemic lupus erythematosus and rheumatoid arthritis. Anti-nuclear antibody (ANA) and Rheumatoid factor (RF) are assessed in pleural fluid.

ANA IN PLEURAL FLUID

Pleural fluid ANA of more than 1: 160 or pleural fluid to serum ANA more than 1 will is suggestive of systemic lupus erythematosus.

Rh FACTOR IN PLEURAL FLUID

When Rh factor in pleural fluid is more than 1:320 and also greater than serum rheumatoid factor, it is likely due to rheumatoid arthritis.

DIFFERENTIATION OF EXUDATIVE AND TRANSUDATIVE PLEURAL EFFUSION:^{40,41}

After complete analysis of pleural fluid, the important step will be differentiation of pleural effusion into exudates and transudate, based on the various parameters tested in the pleural fluid.

Initially pleural fluid specific gravity was taken into account to differentiate exudative and transudative effusion. Then pleural fluid protein was taken as an important parameter which supplanted measurement of specific gravity.

Later it was found that, differentiating exudates and transudate using pleural fluid protein alone was not quite sensitive and may lead on to misclassification.

Light's and his coworkers published that pleural fluid protein and LDH to be measured to compare it with serum values in a combination test that used an "or" diagnostic test.

LIGHT'S CRITERIA⁴¹

1. Pleural fluid to serum protein ratio more than 0.5
2. Pleural fluid to serum LDH ratio more than 0.6
3. Pleural fluid LDH more than 2/3 of upper limit of normal serum LDH.

Presence of **exudative effusion** is established by Light's criteria, if any one of the above is present. Transudative effusion will not meet any criteria.

Most of the exudates were correctly diagnosed by Light's criteria. In patients with cirrhosis and congestive heart failure, who are started on diuretics, light's criteria misclassified transudate as exudates. Though it has high sensitivity of 98% specificity was found to be 74% in subsequent validation studies.

It was observed by Chakko⁴² and his coworkers that transudative pleural effusions that is due to congestive heart failure, after treating with diuretics, light's criteria will show it as exudate⁴⁴. This is due to rapid pleural fluid absorption from pleural space.

To overcome this pitfall of Light's criteria, Roth et al suggested to do serum effusion albumin gradient but that also needs blood sampling.

As Light's criteria has high sensitivity but low specificity, many other studies are also done to find out simple tests, comparable to light's criteria to differentiate exudate and transudate. Pleural fluid cholesterol, pleural fluid cholesterol and LDH, bilirubin level in pleural fluid, soluble leukocyte selectin, cytokines, uric acid and pleural fluid to serum cholinesterase are the some new tests studied.

PLEURAL FLUID CHOLESTEROL AND LDH TO DIFFERENTIATE EXUDATE AND TRANSUDATE

Pleural fluid cholesterol and LDH is studied to separate exudates and transudate. Marina Costa and M.Quironga⁴³ worked on finding out simple set of indicators to separate exudate and transudate. They worked on pleural fluid cholesterol and other parameters with cholesterol, to determine whether similar results can be attained with Light et al. They found that pleural fluid cholesterol of >45mg/dl and LDH of >200 IU/L is simple and presence of any one or both of these parameters is diagnostic of exudate. This test is comparable to light's criteria.

Rohit Rungta⁴⁴ also studied about pleural fluid biochemical parameters and analysed them with pleural fluid cholesterol. He found that pleural fluid analysis of cholesterol and LDH with cut off value of > 45mg/dl and > 200 IU/L respectively has higher sensitivity and specificity than any other combinations in differentiating exudate and transudate.

So this study 'PLEURAL FLUID CHOLESTEROL AND LACTATE DEHYDROGENASE TO DIFFERENTIATE EXUDATES AND TRANSUDATE AND COMPARING IT WITH LIGHT'S CRITERIA' is taken to assess the diagnostic utility of this test, in separation of pleural effusion to exudate and transudate in comparison with Light's criteria.

MATERIALS AND METHODS

MATERIALS AND METHODS

SETTING

This study is conducted in the patients admitted in Government Royapettah hospital.

STUDY POPULATION

Patients admitted as in-patients in medical wards and diagnosed to have pleural effusion clinically and radiologically.

TYPE OF THE STUDY - Cross sectional study

HAZARDS OF THE STUDY - Nil

INCLUSION CRITERIA

- Newly diagnosed cases of pleural effusion

EXCLUSION CRITERIA

- Traumatic pleural effusion
- Patients already started on treatment for pleural effusion

SAMPLE SIZE

50 patients admitted and diagnosed to have pleural effusion in the medical ward.

STUDY DURATION

From January 2015 to August 2015

METHODS AND DATA COLLECTION

After taking detailed history from the patient, they are subjected to complete clinical examination. All the patients with suspected pleural effusion clinically are further subjected to radiological investigations, mainly chest x ray and then ultrasonogram chest if needed, to confirm the presence of pleural effusion.

The thoracentesis procedure was explained to the patient. After getting consent from the patient, under strict aseptic precaution, thoracentesis was done and pleural fluid was sent for analysis.

Pleural fluid analysis comprising of glucose, protein, LDH, cholesterol, cytology, AFB, gram stain was done. Complete blood count, blood urea, serum creatinine, liver function tests, serum protein and serum LDH, serum cholesterol was done. Sputum gram stain and AFB was done.

Light's criteria was used to classify the patients into exudate and transudate. Transudate group were further subjected to find the cause of transudate by subjecting them to ultrasound abdomen, echocardiogram etc.

Pleural fluid cholesterol of more than 45mg/dl and LDH of more than 200IU/L is taken as cut off value and presence of any one or both parameter is diagnostic of exudate, as per previous study references.

STATISTICAL ANALYSIS

All the observations in this study analysed statistically using software. Sensitivity, Specificity, Positive predictive value and Negative predictive value of Pleural fluid cholesterol and LDH test is analysed with SPSS 20.0 software and CHI-SQUARE test is used. Measure of agreement between two test and kappa value is calculated. P value of <0.05 is taken as significant.

OBSERVATION

AND

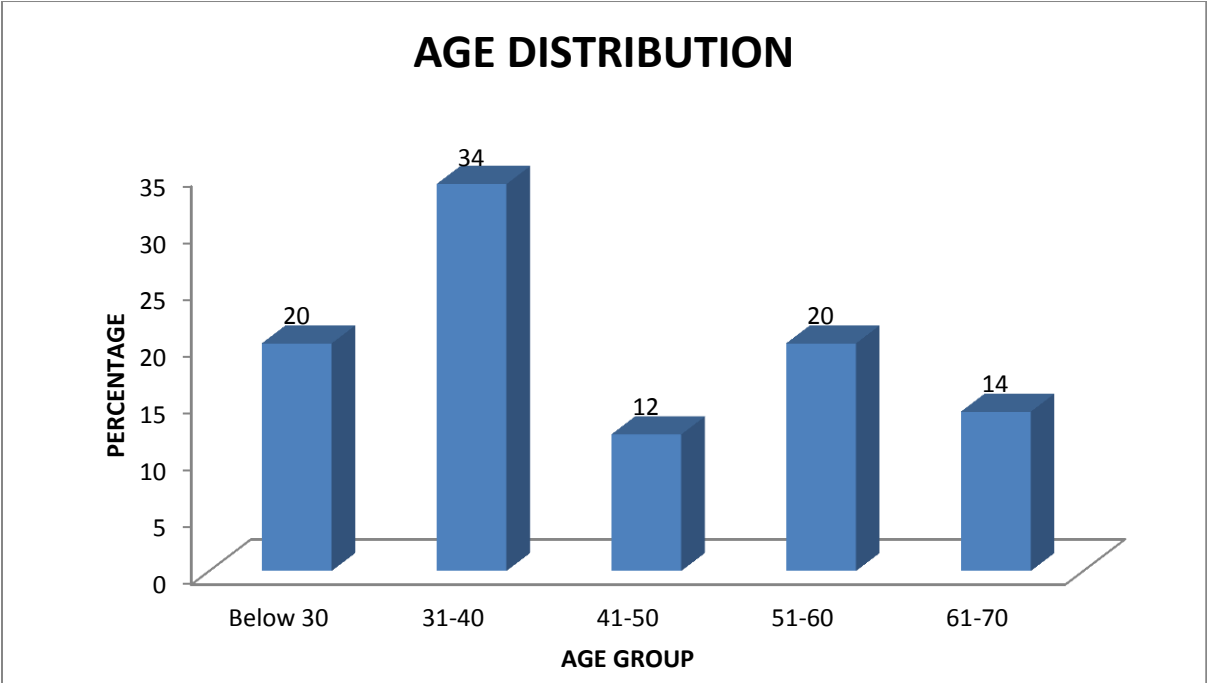
ANALYSIS

OBSERVATION AND ANALYSIS

AGE DISTRIBUTION

10 cases out of fifty cases in the study are below the age of thirty years comprising 20% of total cases 17 cases are between 31-40 yrs comprising 34% of total cases 6 cases are between 41-50yrs comprising 12% of total cases. 10 cases are between 50-60 yrs comprising 20% of total cases. 7 cases are above 60 yrs comprising 14% of total cases.

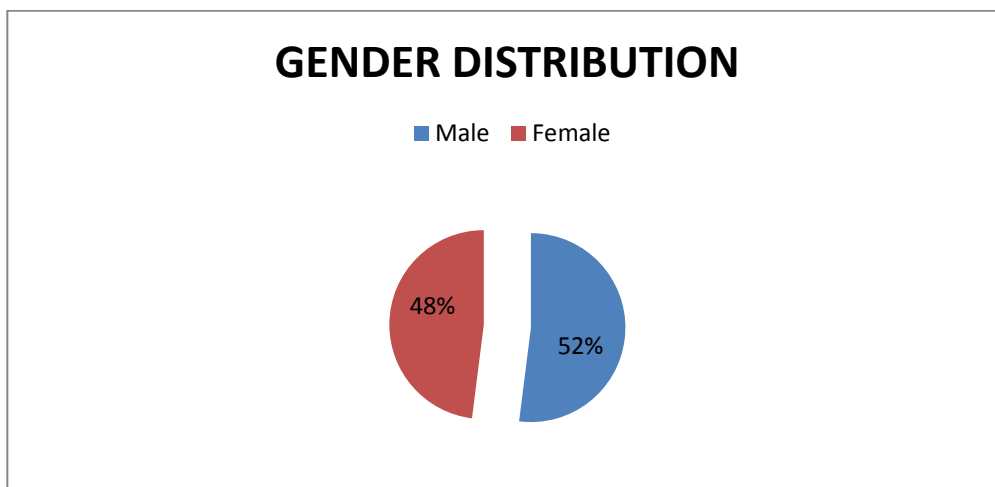
AGE IN YEARS	FREQUENCY	PERCENT
Below 30	10	20.0
31-40	17	34.0
41-50	6	12.0
51-60	10	20.0
61-70	7	14.0



SEX

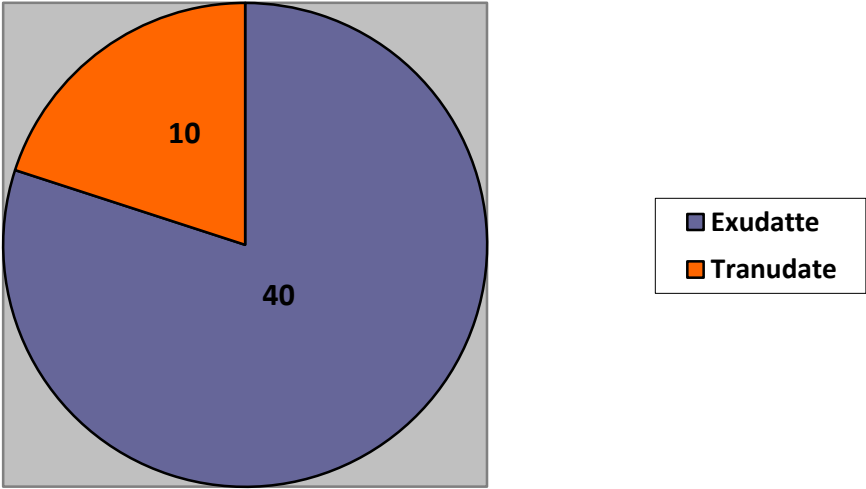
Among 50 patients in the study males are 26 and females are 24 cases comprising 52% and 48% respectively.

GENDER	FREQUENCY	PERCENT
Male	26	52.0
Female	24	48.0



EXUDATE VS TRANSUDATE

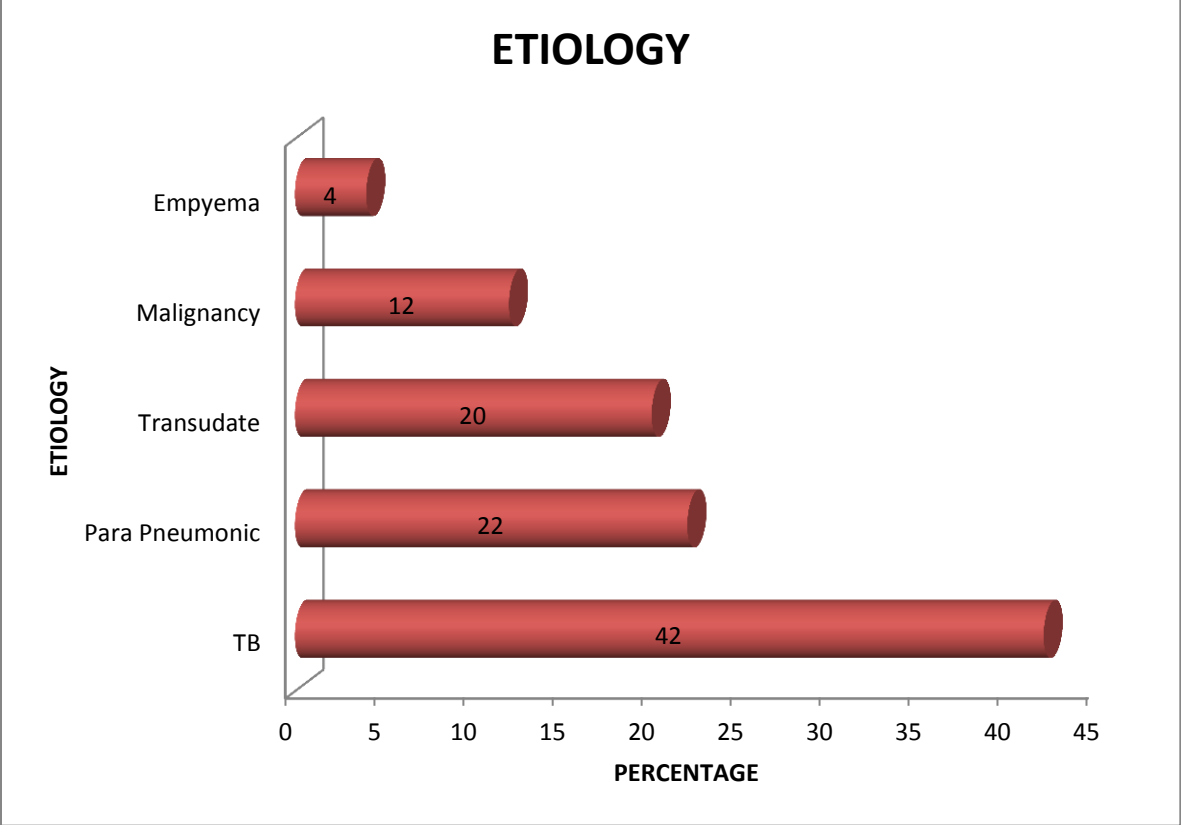
TYPE	Frequency	Percent
Exudate	40	80.0
Transudate	10	20.0



ETIOLOGY

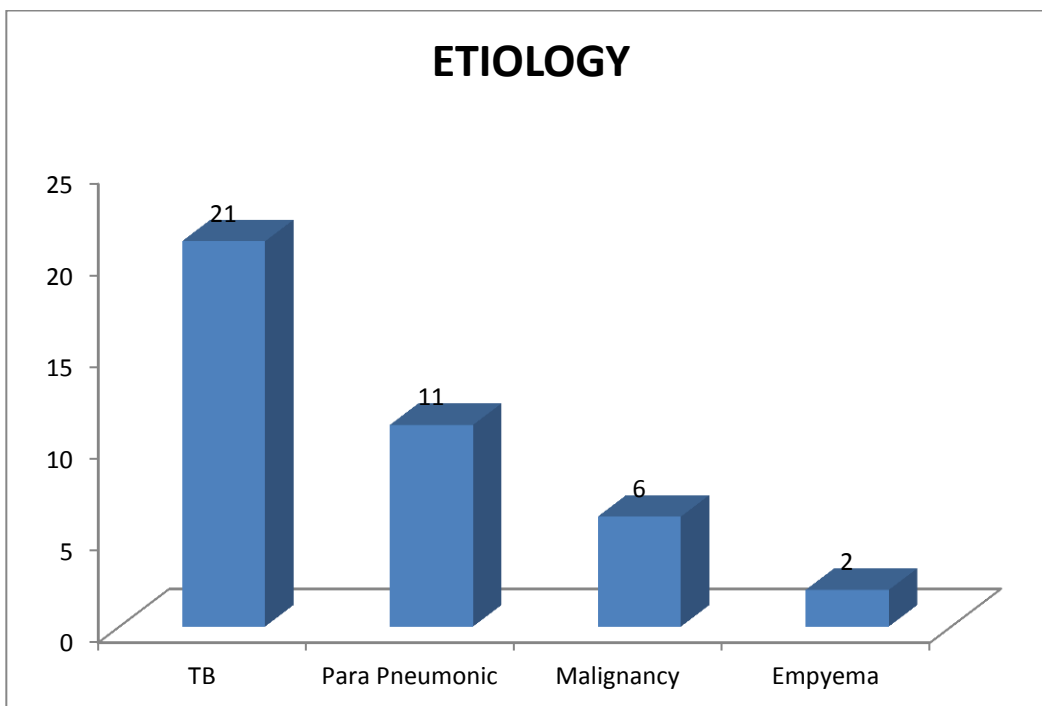
- Out of 50 cases tuberculosis comprises 21 cases with 42%
- Parapneumonic effusion cases are 11 with 22%
- Transudates are 10 cases with 20%
- Malignancy are 6 cases with 12%
- Empyema comprises 2 cases with 4%.

TYPE	FREQUENCY	PERCENT
Tuberculosis	21	42.0
Para Pneumonic	11	22.0
Transudate	10	20.0
Malignancy	6	12.0
Empyema	2	4.0



ETIOLOGY IN EXUDATE

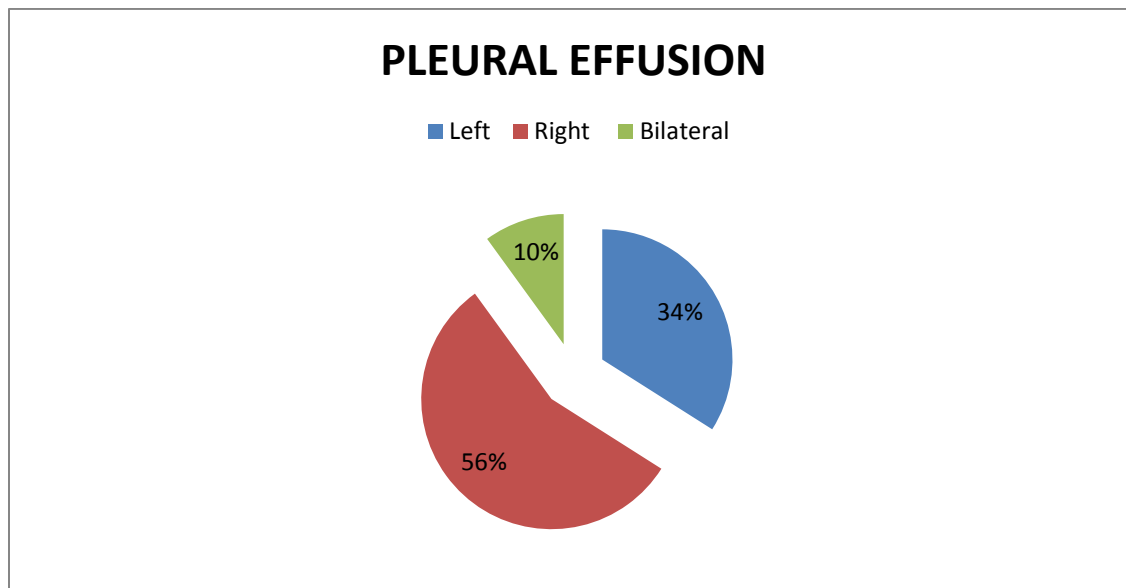
ETIOLOGY	Frequency
TB	21
Para Pneumonic	11
Malignancy	6
Empyema	2



DISTRIBUTION OF CHEST XRAY FINDING

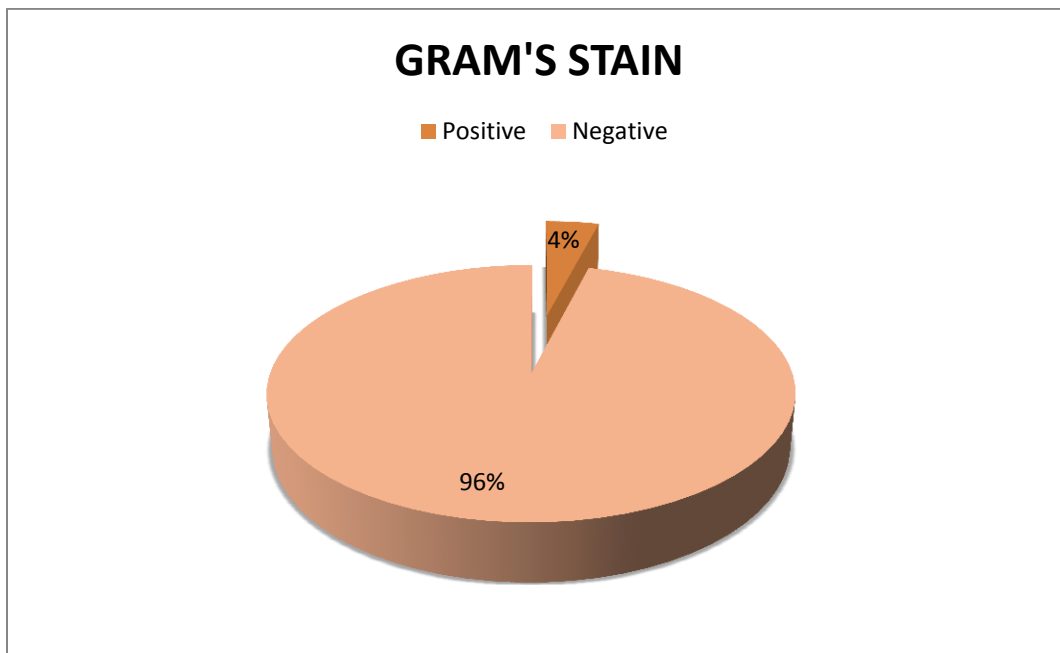
Out of 50 cases chest x ray showed right sided pleural effusion in 28 cases comprising 56%, 17 cases of left sided pleural effusion with 34%, 5 cases of bilateral effusion which comprises 10%.

SIDE	FREQUENCY	PERCENT
Left	17	34.0
Right	28	56.0
Bilateral	5	10.0



PLEURAL FLUID GRAM STAIN

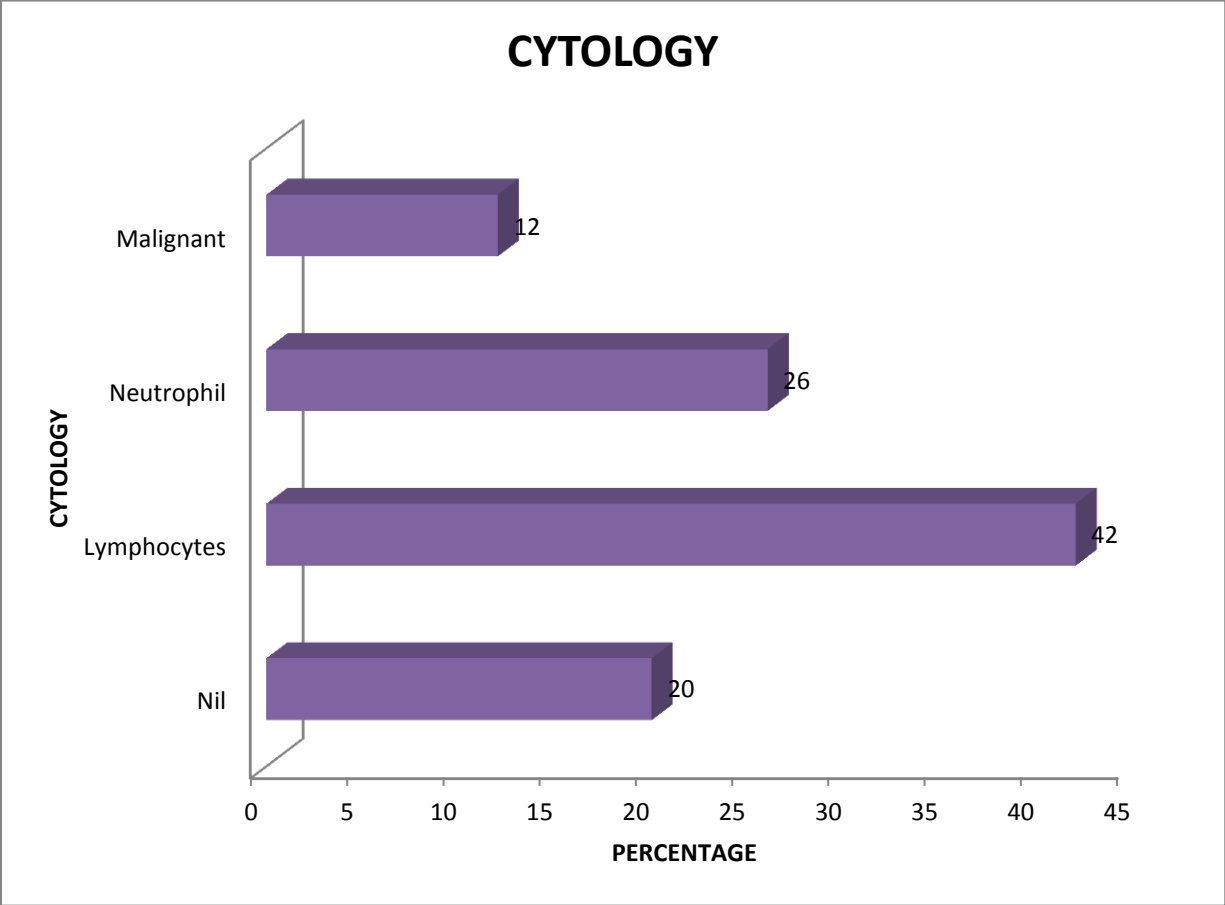
GRAM STAIN	FREQUENCY	PERCENT
Positive	2	4.0
Negative	48	96.0



PLEURAL FLUID CYTOLOGY

Lymphocytic pleural effusion is 21 cases out of 50 cases comprising 42%. All the lymphocytic cases are tuberculous etiology. 13 pleural effusions are neutrophilic in nature which comprises 26%. 11 neutrophilic effusions are parapneumonic and 2 cases of empyema. 6 cases are malignant pleural effusion with 12% of total. 10 pleural effusions are acellular with 20% of total. All the acellular pleural effusions are transudate in nature.

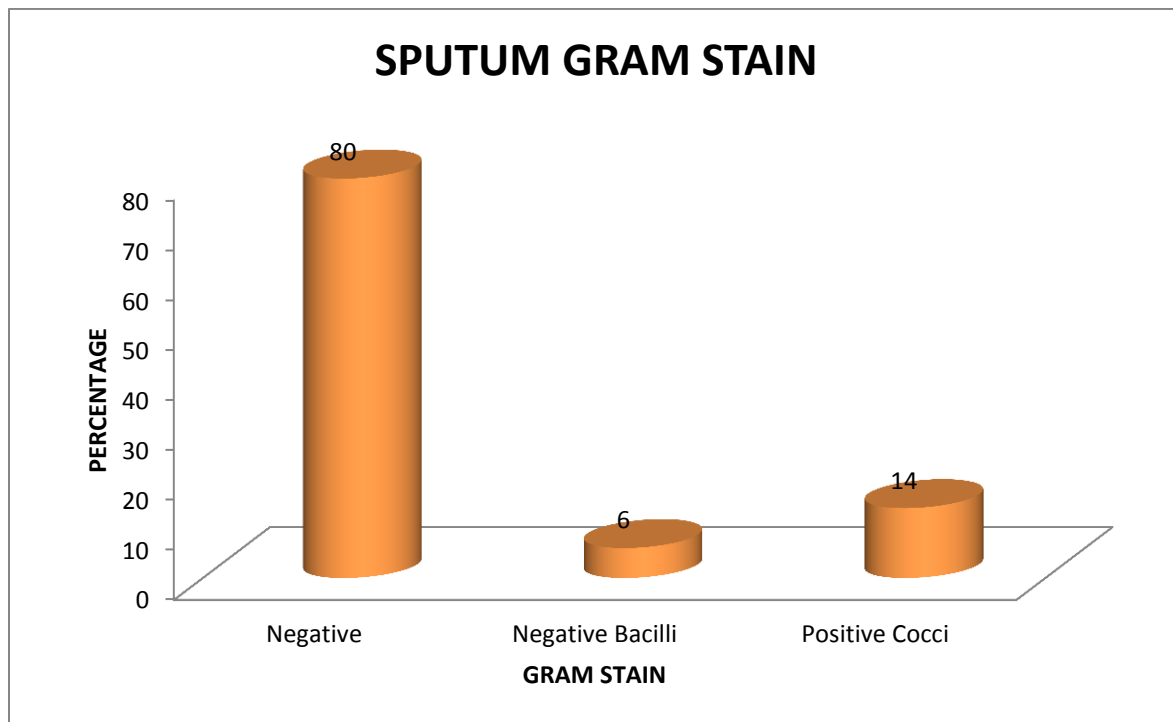
CYTOLOGY	FREQUENCY	PERCENT
Nil	10	20.0
Lymphocytes	21	42.0
Neutrophil	13	26.0
Malignant	6	12.0



SPUTUM GRAM STAINS:

Only 10 cases are sputum gram stain positive in which three are gram negative bacilli and seven cases are gram positive cocci.

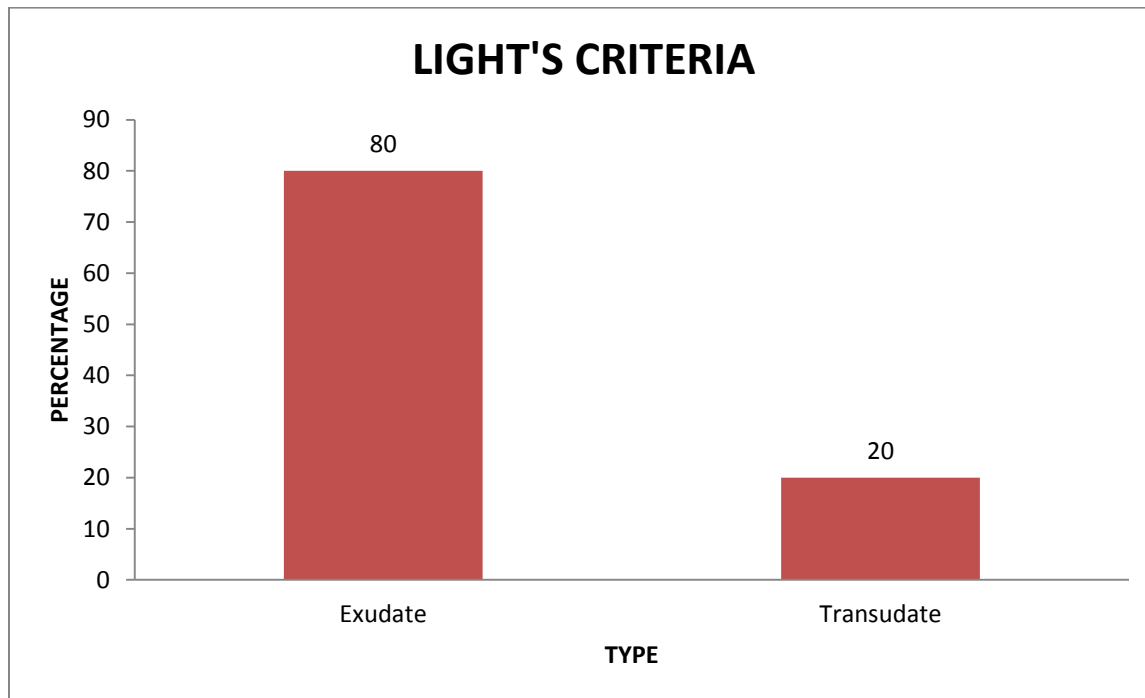
GRAM STAIN	FREQUENCY	PERCENT
No organism	40	80.0
Negative Bacilli	3	6.0
Positive Cocci	7	14.0



LIGHTS CRITERIA

Out of 50 cases, Light's criteria diagnosed 40 cases as exudates which is 80% of total cases and 10 cases as transudates which is 20% of total cases.

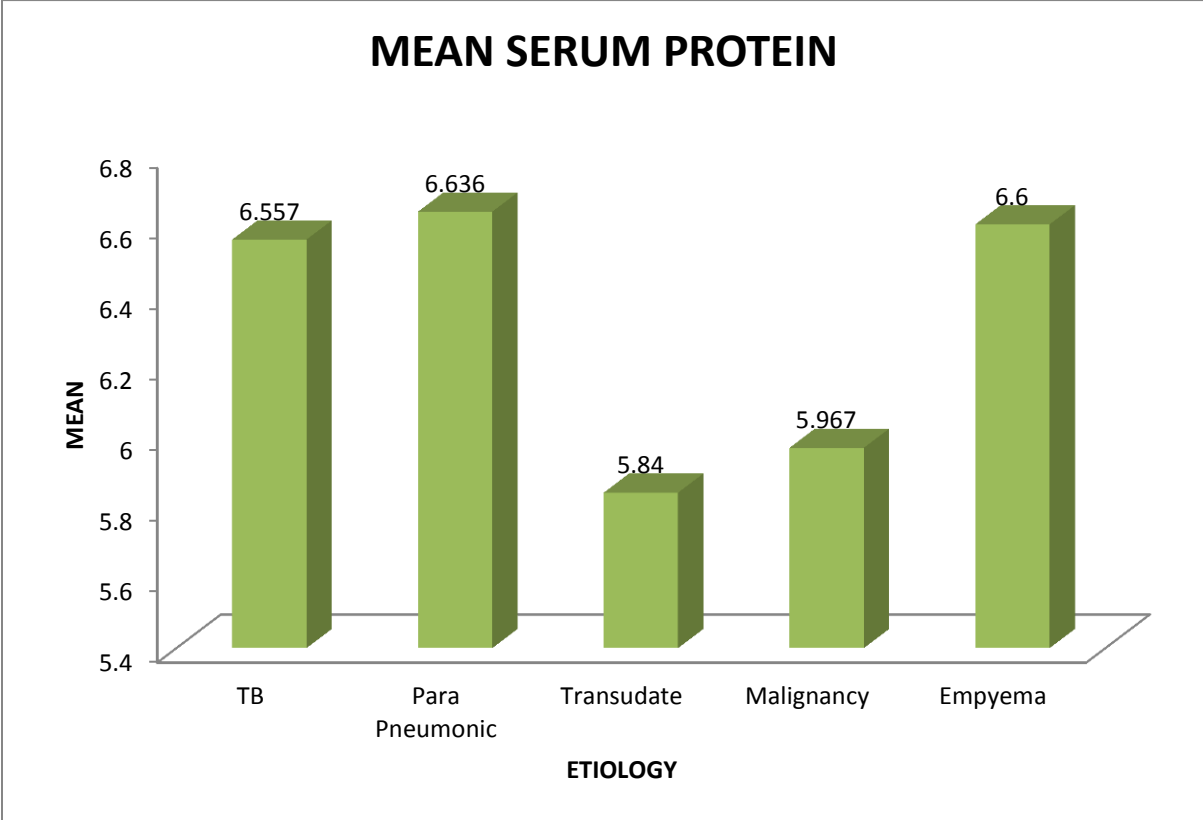
TYPE	FREQUENCY	PERCENT
Exudate	40	80.0
Transudate	10	20.0



MEAN SERUM PROTEIN LEVEL

Mean serum protein level in tuberculous effusion patients is 6.5gm/dl. In Para pneumonic effusion cases mean serum protein level is 6.6gm/dl. Mean serum protein level in transudative effusion cases is 5gm/dl. In patients with malignant effusion, the mean serum protein level is 5.9 gm. /dl. In empyema patients, mean serum protein level is 6.6gm/dl.

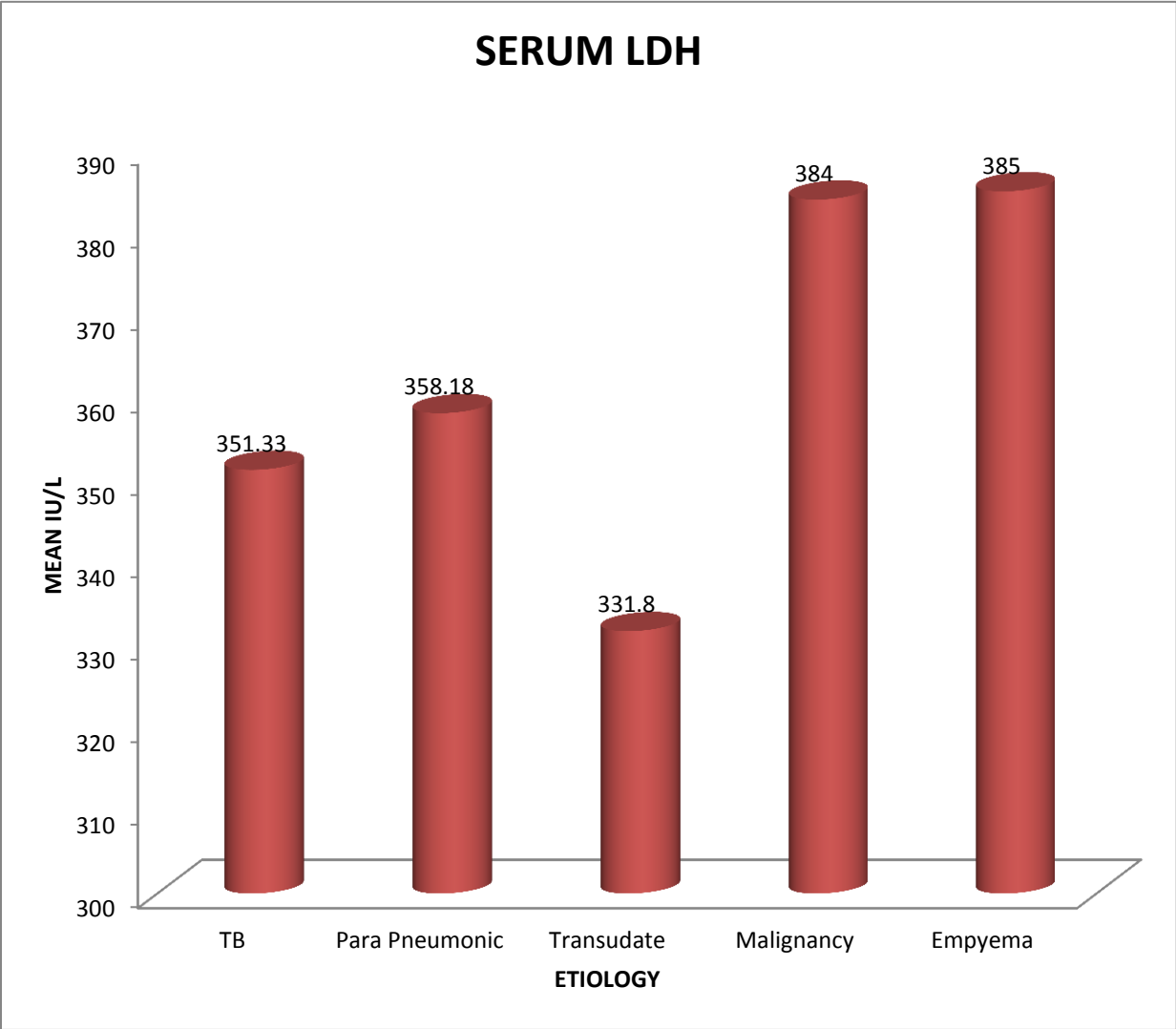
ETIOLOGY	N	MEAN (gm./dl)	SD
TB	21	6.557	0.263
Para Pneumonic	11	6.636	0.366
Transudate	10	5.840	0.636
Malignancy	6	5.967	0.196
Empyema	2	6.600	0.282



MEAN SERUM LDH LEVEL

In patients with tuberculous pleural effusion the mean serum LDH level is 351IU/L. In patients with parapneumonic effusion, the mean serum LDH value is 358 IU/L. In transudative effusion patients the mean serum LDH value is 331IU/L. In malignant effusion cases, the mean serum LDH is 384 IU/L. In empyema cases, the mean serum LDH level is 385 IU/L.

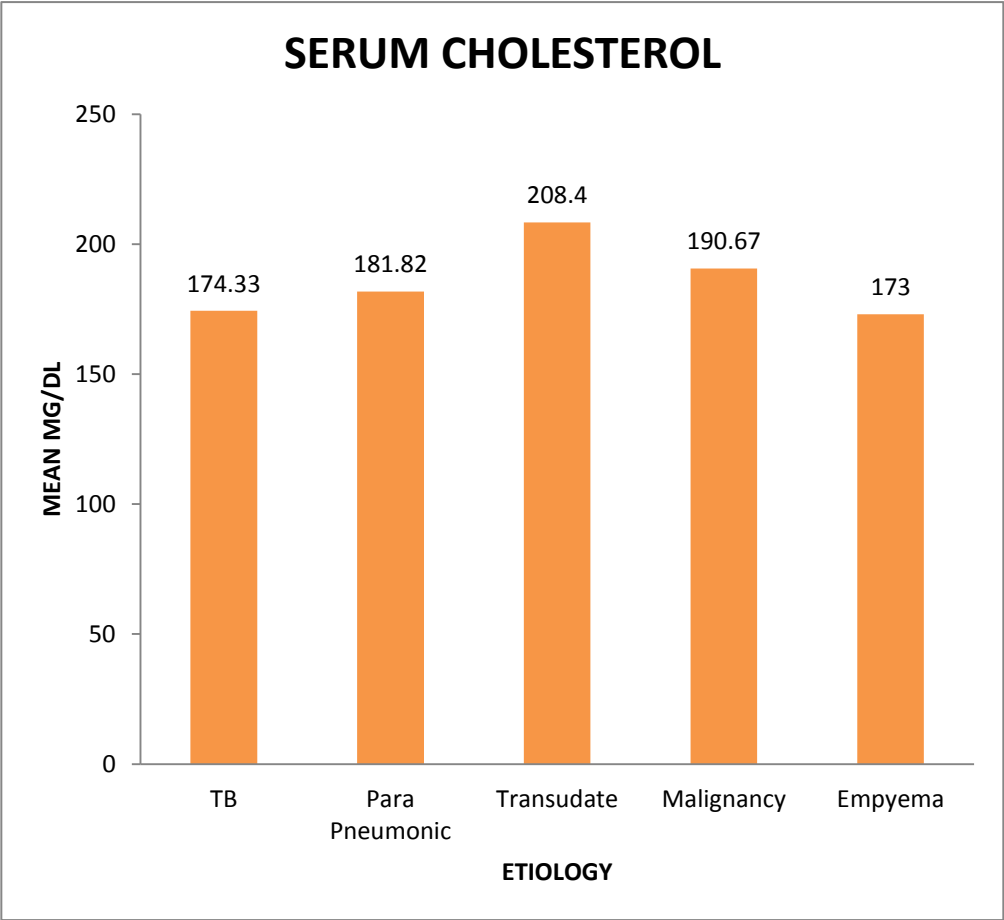
ETIOLOGY	N	MEAN (IU/L)	SD
TB	21	351.33	30.622
Para Pneumonic	11	358.18	28.375
Transudate	10	331.80	16.612
Malignancy	6	384.00	11.524
Empyema	2	385.00	15.556



MEAN SERUM CHOLESTEROL LEVEL

In tuberculous pleural effusion patients, the mean value of serum cholesterol is 174mg/dl. In parapneumonic effusion patients, the mean level is 181mg/dl. In transudative effusion cases, the mean serum cholesterol is found to be 208mg/dl. In malignant cases, the mean serum cholesterol level is 190mg/dl. In cases of empyema, the mean level is found to be 173 mg /dl.

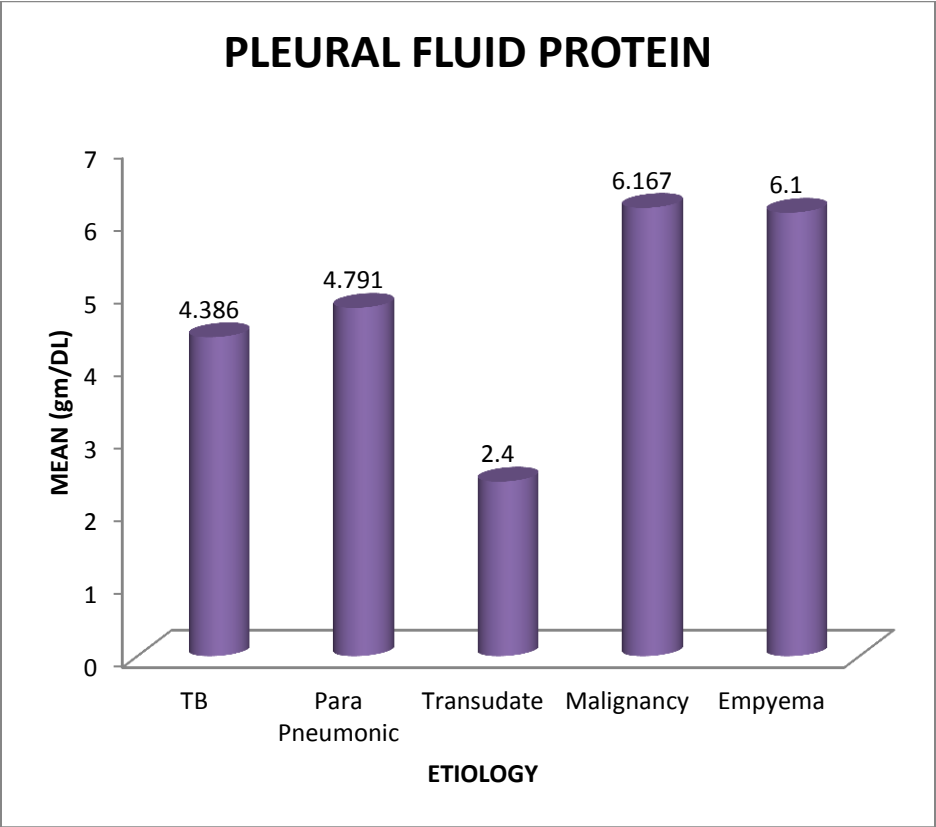
ETIOLOGY	N	MEAN (Mg/dl)	SD
TB	21	174.33	11.521
Para Pneumonic	11	181.82	10.638
Transudate	10	208.40	31.690
Malignancy	6	190.67	7.866
Empyema	2	173.00	18.385



MEAN PLEURAL FLUID PROTEIN LEVEL

Mean pleural fluid protein level in tuberculous effusion patients is 4.3gm/dl. In parapneumonic effusion patients, the mean pleural fluid protein level is 4.7 gm/dl. Trasudative pleural effusion patients showed pleural fluid mean protein level of 2.4gm/dl. Maliganat cases showed pleural fluid protein value of 6.1gm/dl. In empyema patients, the mean value is 6.1gm/dl.

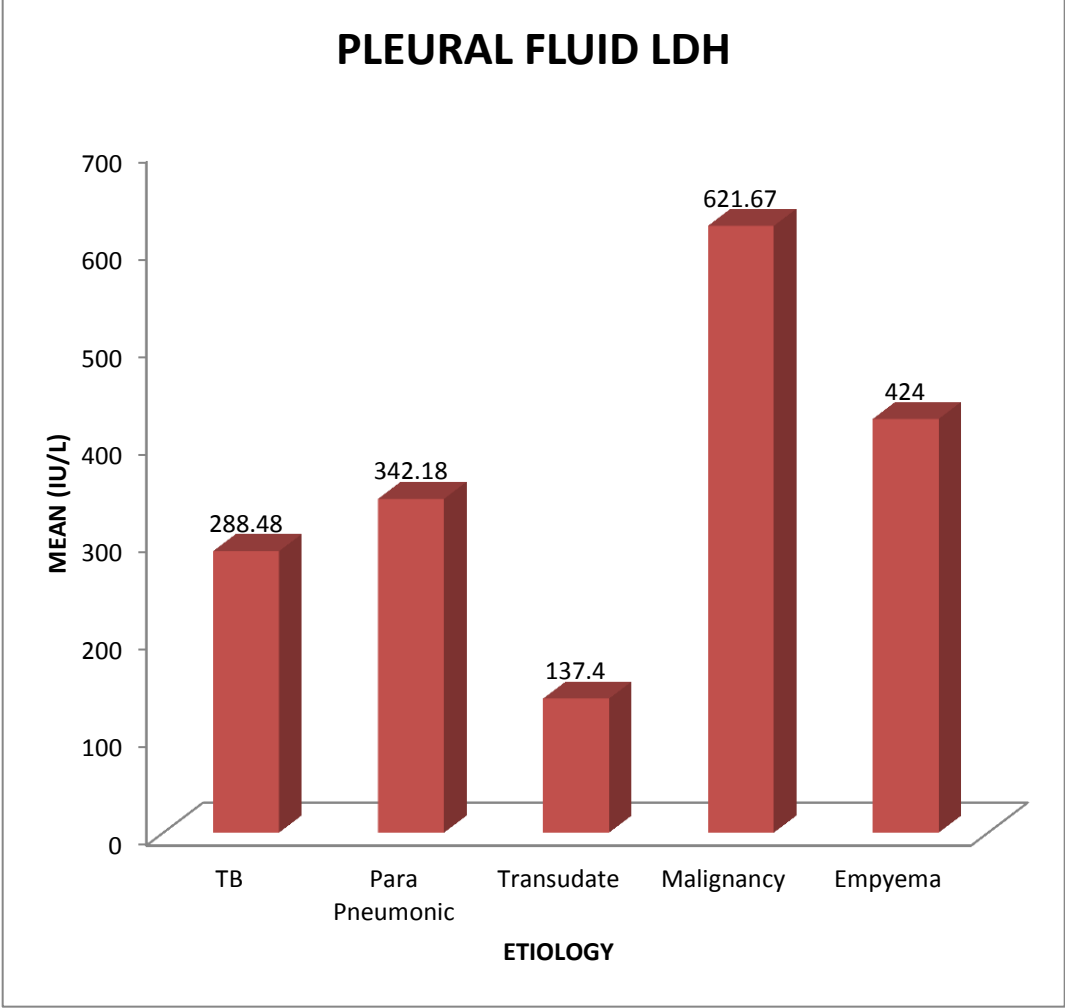
ETIOLOGY	N	MEAN (gm./dl)	SD
TB	21	4.386	0.377
Para Pneumonic	11	4.791	0.270
Transudate	10	2.400	0.722
Malignancy	6	6.167	0.367
Empyema	2	6.100	0.424



MEAN PLEURAL FLUID LDH LEVEL

288 IU/L is the mean pleural fluid LDH value in tuberculous effusion cases. In parapneumonic effusion patients, the mean pleural fluid LDH value is 342 IU/L. In transudates the mean pleural fluid LDH is 137 IU/L. In empyema, the mean pleural fluid LDH is 424 IU/L. In malignant effusion, the mean pleural fluid LDH is found to be 621 IU/L.

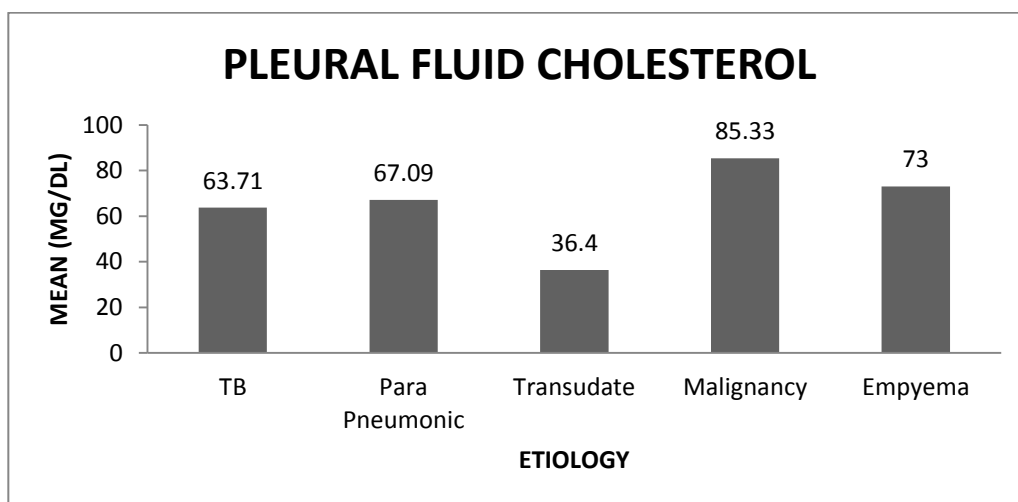
ETIOLOGY	N	MEAN (IU/L)	SD
TB	21	288.48	11.677
Para Pneumonic	11	342.18	35.535
Transudate	10	137.40	26.298
Malignancy	6	621.67	44.279
Empyema	2	424.00	.000



MEAN PLEURAL FLUID CHOLESTEROL

In pleural effusion due to tuberculosis, the mean pleural fluid cholesterol is 63mg/dl. In parapneumonic effusion patients, the mean value of pleural fluid cholesterol is 67mg/dl. In transudates, the mean pleural fluid cholesterol is found to be 36mg /dl. In malignant pleural effusion cases, the mean pleural fluid cholesterol value is 85mg/dl. In empyema cases, the mean value is 73mg/dl.

ETIOLOGY	N	MEAN	SD
TB	21	63.71	4.529
Para Pneumonic	11	67.09	4.323
Transudate	10	36.40	3.502
Malignancy	6	85.33	6.154
Empyema	2	73.00	4.243



ACTUAL ETIOLOGY VS LIGHT'S CRITERIA VS PLEURAL FLUID

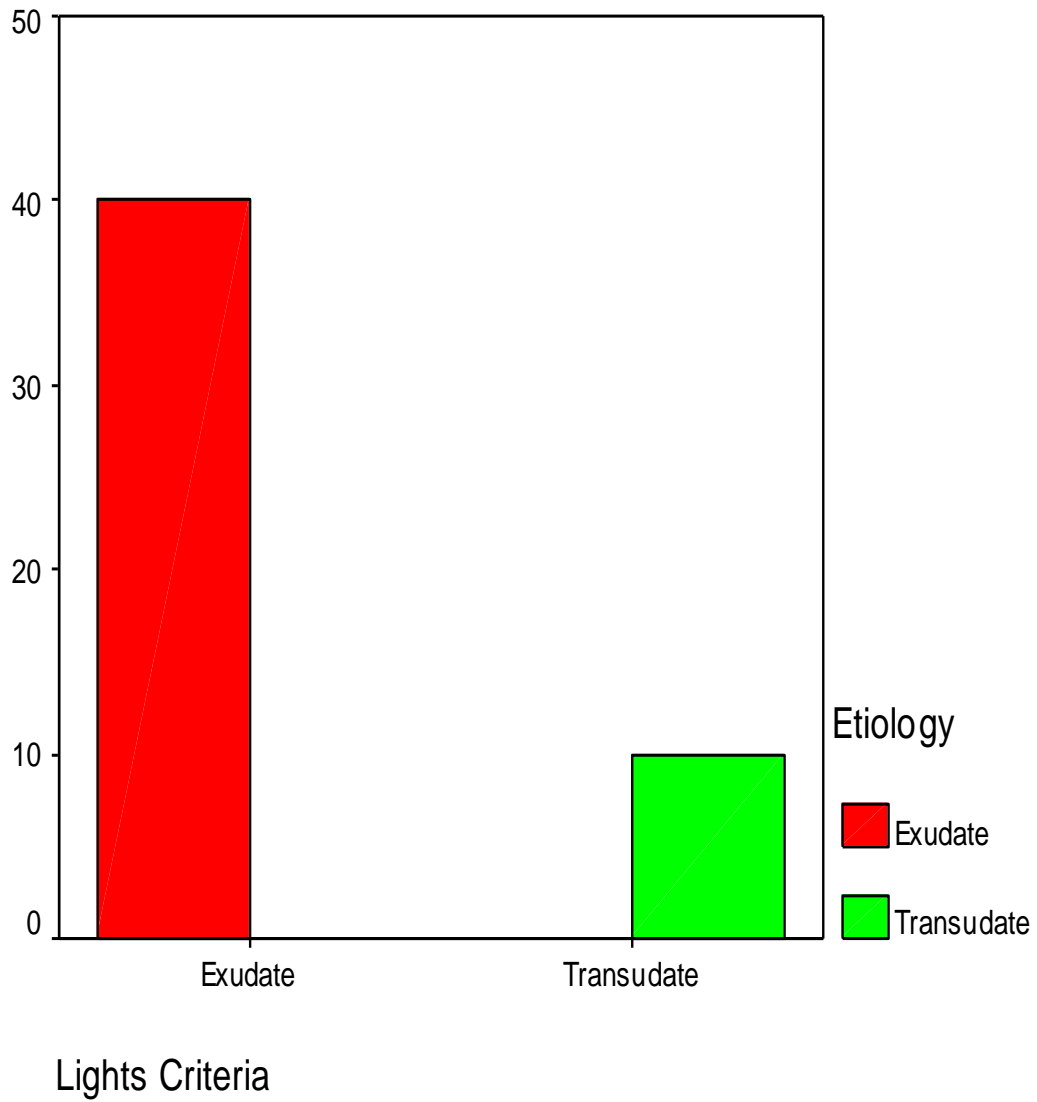
CHOLESTEROL AND LDH TEST

Out of 50 total cases, as per etiology, 40 cases were exudates and light's criteria diagnosed all the 40 cases correctly as exudates. The newer test pleural fluid cholesterol and LDH, diagnosed 38 cases correctly as exudates and two cases were misclassified as transudates. Out of 10 transudate cases by etiology, Light's diagnosed all the 10 cases as transudates correctly. Pleural fluid cholesterol and LDH test diagnosed 8 cases correctly as transudates and misclassified two transudates as exudates.

LIGHT'S CRITERIA VS ACTUAL ETIOLOGY

		ACTUAL ETIOLOGY		Total	P VALUE
		EXUDATE	TRANSUDATE		
LIGHTS CRITERIA	EXUDATE	40	0	40	0.001**
	TRANSUDATE	0	10	10	
Total		40	10	50	

LIGHT'S CRITERIA VS ACTUAL ETIOLOGY

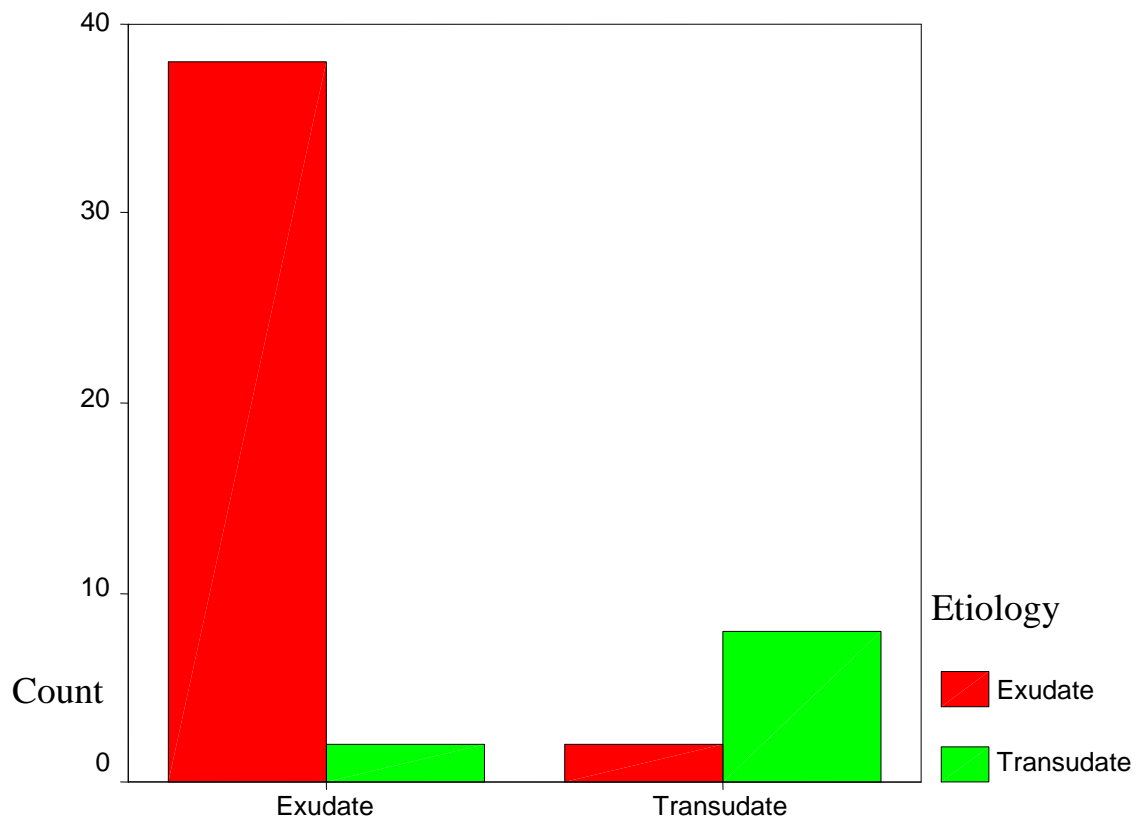


PLEURAL FLUID CHOLESTEROL AND LDH TEST

VS

ACTUAL ETIOLOGY

		ACTUAL ETIOLOGY		Total	P VALUE
		EXUDATE	TRANSUDATE		
PLEURAL FLUID CHOLESTEROL + LDH	EXUDATE	38	2	40	0.001**
	TRANSUDATE	2	8	10	
Total		40	10	50	



Pleural fluid cholesterol and ldh

LIGHT'S CRITERIA

VS

PLEURAL FLUID CHOLESTEROL AND LDH TEST

As already seen, the routinely used light's criteria diagnosed 40 cases as exudates. The pleural fluid cholesterol and LDH test diagnosed 38 cases as exudates out of 40 cases diagnosed by Light's criteria as exudates. Two exudates were only misclassified as transudates.

Similarly pleural fluid cholesterol and LDH test misclassified only two transudates as exudates, which were diagnosed as transudates by Light's criteria.

		PLEURAL FLUID		Total	P VALUE
		CHOLESTEROL+LDH			
		EXUDATE	TRANSUDATE		
LIGHTS CRITERIA	EXUDATE	38	2	40	0.001**
	TRANSUDATE	2	8	10	
Total		40	10	50	

By this 2*2 table pleural fluid cholesterol and LDH was compared with the standard LIGHTS criteria and the results showed

- Sensitivity to be 95%
- Specificity to be 80%
- Positive predictive value 95%
- Negative predictive value 80%.
- P value of <0.001** was significant
- In measure of agreement, kappa value is 0.75 which shows good correlation.

DISCUSSION

DISCUSSION

Out of 50 cases taken into the study, 40 cases were exudates and 10 cases were transudates etiologically. 26 patients are male and 24 patients are female out of fifty patients. Most of the patients are between 30 to 60 years. Only 7 cases were above 60 years and 10 cases below 30 years.

In this study, it was found that tuberculosis is found to be the most common cause of pleural effusion and parapneumonic effusion came next. Transudative effusion comprises only 20%. X ray chest showed right sided pleural effusion in 56% of cases and left sided effusion in 34% of cases. Bilateral effusion was seen in 10% of cases, all of which were due to transudative etiology.

In this study, it is found that lymphocytic effusion predominates and all are due to tuberculosis etiology. The next one is neutrophilic effusion which is due to parapneumonic etiology and empyema. All the transudative effusions were acellular.

Sputum AFB was negative in all the patients in the study. Sputum gram stain is positive in ten cases, in which three are gram negative bacilli and seven are gram positive cocci. All were seen with parapneumonic effusion.

Mean serum protein level is found to be high in effusion due to parapneumonic cause and empyema with 6.6gm/dl. Mean serum LDH is higher in malignant pleural effusion and empyema with 384 IU/L and 385 IU/L respectively. Mean serum cholesterol is higher in transudative effusion patients with value of 208mg/dl.

Mean pleural fluid protein is 2.4gm/dl in transudative effusion. In exudative effusion, mean pleural fluid protein is found to be highest in malignancy and empyema with value of 6.1gm/dl. In tuberculosis it is 4.3gm/dl and in parapneumonic 4.7gm/dl.

Mean Pleural fluid LDH in transudate is 137 IU/L. In malignancy, it is highest with 621 IU/L and in empyema, it is 424 IU/L. In tuberculosis, it is 288 IU/L and in parapneumonic effusion, it is 342 IU/L.

Mean pleural fluid cholesterol is highest in malignant pleural effusion with 85mg/dl and next comes empyema with 73mg/dl. In transudates, it is 36mg/dl. In tuberculosis cases, it is 63mg/dl and in parapneumonic effusion, it is 67mg/dl.

Light's criteria diagnosed 40 cases as exudates which correctly matched with etiological diagnosis. In the same way, it diagnosed ten cases as transudates which also correctly matched with etiological diagnosis. So light's criteria showed 100% sensitivity and 100% specificity according to this study. The point to be noted is pleural fluid analysis was done even before starting diuretics.

The pleural fluid cholesterol and LDH test diagnosed 38 cases as exudates when compared with Light's criteria and two exudates were only misclassified as transudates. In the same way, this test diagnosed 8 cases as transudates correctly when compared with Light's criteria and 2 transudates were misclassified as exudates.

COMPARITIVE ANALYSIS OF LIGHT'S CRITERIA AND PLEURAL FLUID CHOLESTEROL AND LDH TEST

When pleural fluid cholesterol and LDH test is compared with routinely used Light's criteria in a 2 * 2 table, the pleural fluid cholesterol and LDH test showed sensitivity of 95% and specificity of 80%, positive predictive value of 95% and negative predictive value of 80%.

Study conducted by Marina costa MD; Teresa Quiroga MD, showed 99% sensitivity and 98% specificity for pleural fluid cholesterol and LDH combination in differentiating exudate and transudate. Study by Rohit Rungta also showed sensitivity of 99% and specificity of 98%. Our present study showed 95% sensitivity and 80% specificity.

**MEASURE OF AGREEMENT BETWEEN LIGHT'S CRITERIA
AND
PLEURAL FLUID CHOLESTEROL AND LDH TEST**

		VALUE	P VALUE
Measure of Agreement	Kappa	0.750	<0.01**
N of Valid Cases		50	

Measure of agreement between these two tests is calculated using kappa value which also showed significant agreement between these two tests with kappa factor of 0.75 and P value of 0.01** which is highly significant.

CONCLUSION

- Light's criteria is the most accepted criteria for differentiating between exudates and transudate in pleural effusion.
- It uses four parameters serum protein, serum LDH, pleural fluid protein and pleural fluid LDH.
- Pleural fluid cholesterol and LDH test in pleural fluid with cholesterol $>45\text{mg/dl}$ and LDH $>200\text{ IU/L}$ showed 95% sensitivity and 80% specificity in diagnosing exudates, which is comparable with Light's criteria, with significant measure of agreement between the two, with significant P value of $<0.01^{**}$.
- Pleural fluid cholesterol and LDH test is useful to differentiate pleural fluid exudate and transudate with the advantage of requiring only two laboratory parameters and no simultaneous blood sample especially in countries like India, where financial and technical constraints are immense.

LIMITATIONS OF THE STUDY

- This study comprises only 50 patients which is a very limited number.
- Pleural fluid analysis was done in newly diagnosed patients who were not started on any diuretics. Therefore the sensitivity and specificity of light's criteria is very high in this study.
- The sensitivity and specificity of the new test should further be evaluated by involving a larger number of subjects.

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ANNEXURE

PROFORMA 1

NAME:

AGE:

SEX:

OCCUPATION:

ADDRESS:

I.COMPLAINTS& PRESENTING ILLNESS

II.PAST HISTORY:

H/o DM,SH1,COPD,PULMONARY TB

H/O trauma

III.PERSONAL HISTORY

H/O Alcoholism&SMOKING

IV.TREATMENT HISTORY

V.GENERAL EXAMINATION

PALLOR /ICTERUS /CLUBBING/CYANOSIS/PEDAL EDEMA

PULSE BP RR JVP

VI.SYSTEM EXAMINATION CVS RS ABDOMEN
CNS

PROFOMA 2

VII. INVESTIGATIONS

Hb TC DC ESR

RBS UREA CREATININE

SERUM PROTEIN

SERUM CHOLESTEROL

SERUM LDH

CXR PA VIEW :

PLURAL FLUID ANALYSIS:

i) protein

ii) ldh

iii) cholesterol

iv) glucose

iv) cytology

v) gram stain, afb stain

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Protocol ID No.06/01/2015 Dt. 20. 01.2015
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A study titled Pleural fluid cholesterol and lactatedehydrogenase to Differentiate exudates and transudate and comparing it with light's criteria. -For Project Work-submitted by Dr.R.B.S.Manian, Post Graduate in General Medicine, KMC, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.



Yannu
20/1/15

CHAIRMAN,
Ethical Committee
Govt. Kilpauk Medical College, Chennai

[Signature]
19/1/2015

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PLEURAL FLUID CHOLESTEROL AND LACTATE DEHYDROGENASE TO

INTRODUCTION

Pleural effusion is one of the common clinical disorders encountered in the medical wards. In a patient with pleural effusion diagnosis can be arrived by history, clinical examination and radiological techniques. Pleural effusion occurs due to various etiologies.

Pleural effusion is of two different types depending on pathophysiology. They are Transudate and Exudate. To find out etiology, first step is to differentiate whether the pleural effusion is of exudate or transudate type. Etiology of pleural effusions differs in different parts of the world.

In Indian scenario, infectious causes of pleural effusion particularly tuberculosis and pneumonic consolidation leading on to parapneumonic effusion will **be** leading cause of pleural effusion.

Though worldwide the incidence of empyema, has come down due to early initiation of antibiotic therapy, in India still many cases of empyema and loculated pleural effusion are encountered in tertiary care hospitals.

After diagnosing pleural effusion in a patient, thoracentesis should be done under strict aseptic precautions and after getting concurrence from the patient. Pleural fluid aspirate is analysed for various parameters like protein,

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INTRODUCTION

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In Indian scenario, infectious causes of pleural effusion particularly tuberculosis and pneumonic consolidation leading on to parapneumonic effusion will be the leading cause of pleural effusion.

Though worldwide the incidence of empyema has come down due to early initiation of antibiotic therapy, in India still many cases of empyema and localized pleural effusion are encountered in tertiary care hospitals.

After diagnosing pleural effusion in a patient, diagnostics should be done under strict aseptic precautions and after getting concurrence from the patient. Pleural fluid aspirate is analyzed for various parameters like protein, glucose, LDH etc.

AGE	SEX	COMPLAIN	TC	ESR	SR.PRO	SR.LDH	SR.CHO	RBS	PLEF.PRO
25	M	F,CP-RT	4,500	40	6.6	386	165	140	4.2
32	M	F,CP-RT	4,700	44	6.5	374	170	130	4.0
60	F	F,SP,D	13,400	42	7.0	380	200	176	4.4
50	M	D,PE	3,800	24	5.2	300	210	182	2.1
18	F	CP.LT	5,600	38	6.8	360	154	140	4.3
55	F	D,CP.LT	3,800	30	6.0	384	206	208	5.8
33	F	F,CP,LOW	6,400	52	6.7	368	172	120	4.2
65	M	D,PE	4,400	18	6.0	388	246	184	2.2
22	F	F,SP,D	16,400	22	6.8	346	166	132	5.1
32	M	CP.RT	4,700	30	6.6	340	150	166	3.8
64	M	D,LOW	5,100	28	6.2	400	190	212	6.6
20	F	D,F	6,800	32	6.2	346	166	126	3.6
43	F	F,SP,D	13,800	30	6.0	390	198	264	4.8
64	M	D,AD,PE	4,600	18	6.1	326	224	186	2.4
38	F	F,CP	6,400	46	6.4	346	182	166	4.0
42	M	D,PE	3,800	20	5.0	386	144	110	2.1
40	F	F,CP.RT,SP	20,600	22	6.8	374	160	242	6.4
65	M	D,AD,PE	6,200	18	6.5	356	240	176	3.2
57	F	F,SP,CP.RT	18,200	18	6.8	378	172	196	4.8
35	M	CP.RT,F	7,200	54	7.0	388	178	166	4.8
65	F	D,	5,600	20	6.0	376	186	146	6.6
28	F	F,D	4,500	46	6.8	384	164	124	5.2
55	M	D,AD,PE	3,600	16	4.8	346	178	106	2.2
34	M	D,F	5,200	18	7.0	374	184	134	4.8
60	F	F,CP.LT,SP,C	18,600	20	6.4	396	186	244	5.8
34	M	D,LOW	12,000	26	6.0	374	184	148	6.0
27	F	F,CP	4,400	36	6.4	346	174	176	4.6
45	M	F,CPLT,SP	12,600	22	7.2	376	186	166	5.2
33	M	F,LOW	3,800	14	6.7	354	186	126	4.4
55	F	D,PE	4,200	14	6.6	346	220	144	3.1
60	M	F,CP,SP	14,600	18	7.0	364	190	134	5.0
18	F	F,CP.RT,SP	4,200	44	6.8	358	166	116	4.8
38	M	F,C,CP,SP	14,400	16	6.6	374	176	138	4.4
40	M	F,LOW	4,200	18	6.8	384	180	160	4.4
55	F	D,LOW	7,400	22	5.6	374	190	182	6.2
36	M	D,F	5,500	34	6.4	326	186	134	4.8
40	F	D,F,C,CP	17,600	22	6.2	302	180	112	5.0
55	M	D,AD,PE	3,200	12	5.8	324	180	96	2.2
24	F	D,F,CP	3,800	34	6.4	320	184	122	4.2
50	F	D,CP.LT	7,400	44	6.4	326	176	164	4.4
56	M	D,LOW,CPLT	5,500	18	6.0	396	188	264	5.8
35	M	F,D	5,200	32	6.2	284	198	134	4.4
28	F	F,LOW	3,400	24	6.6	296	176	112	4.6
70	M	D,PE	4,400	14	6.6	346	226	156	1.8
38	M	F,C,CP,SP	14,200	16	6.4	326	176	166	4.8
32	F	F	3,800	22	6.4	376	180	116	4.6
65	F	D,AD,PE	4,400	12	6.0	326	216	226	1.6
36	M	F,CP	5,400	14	6.0	324	184	144	4.2
45	M	F,C,CP,SP	6,600	18	6.6	328	176	156	4.6
30	F	F,CP.RT	4,800	38	6.4	394	166	146	4.4

PLEF.LDH	PLEF.CHO	PLEF.GLU	PLEF.GRA	PLEF.AFB	PLEF.CYT	SP.GRAM
284	62	66	Negative	Negative	Lymphocytes	Negative
278	66	58	Negative	Negative	Lymphocytes	Negative
368	74	48	Negative	Negative	Neutrophil	Positive Cocci
96	38	66	Negative	Negative	Nil	Negative
284	68	54	Negative	Negative	Lymphocytes	Negative
612	82	40	Negative	Negative	Malignant	Negative
288	72	48	Negative	Negative	Lymphocytes	Negative
212	52	64	Negative	Negative	Nil	Negative
384	70	40	Negative	Negative	Neutrophil	Positive Cocci
292	62	50	Negative	Negative	Lymphocytes	Negative
602	92	42	Negative	Negative	Malignant	Negative
178	40	52	Negative	Negative	Lymphocytes	Negative
374	72	42	Negative	Negative	Neutrophil	Negative Bacilli
110	30	62	Negative	Negative	Nil	Negative
284	66	54	Negative	Negative	Lymphocytes	Negative
206	54	52	Negative	Negative	Nil	Negative
424	70	38	Positive	Negative	Neutrophil	Positive Cocci
154	40	60	Negative	Negative	Nil	Negative
386	70	42	Negative	Negative	Neutrophil	Negative Bacilli
316	68	56	Negative	Negative	Lymphocytes	Negative
614	92	44	Negative	Negative	Malignant	Negative
324	58	52	Negative	Negative	Lymphocytes	Negative
166	40	64	Negative	Negative	Nil	Negative
284	54	60	Negative	Negative	Lymphocytes	Negative
424	76	48	Positive	Negative	Neutrophil	Positive Cocci
696	88	40	Negative	Negative	Malignant	Negative
288	56	52	Negative	Negative	Lymphocytes	Negative
334	68	38	Negative	Negative	Neutrophil	Negative
280	60	54	Negative	Negative	Lymphocytes	Negative
166	38	64	Negative	Negative	Nil	Negative
324	68	38	Negative	Negative	Neutrophil	Negative Bacilli
198	42	54	Negative	Negative	Lymphocytes	Negative
312	66	48	Negative	Negative	Neutrophil	Positive Cocci
286	68	52	Negative	Negative	Lymphocytes	Negative
642	80	36	Negative	Negative	Malignant	Negative
286	66	56	Negative	Negative	Lymphocytes	Negative
374	64	48	Negative	Negative	Neutrophil	Positive Cocci
146	40	56	Negative	Negative	Nil	Negative
280	68	54	Negative	Negative	Lymphocytes	Negative
288	66	52	Negative	Negative	Lymphocytes	Negative
564	78	40	Negative	Negative	Malignant	Negative
292	64	56	Negative	Negative	Lymphocytes	Negative
278	66	58	Negative	Negative	Lymphocytes	Negative
146	36	66	Negative	Negative	Nil	Negative
298	64	56	Negative	Negative	Neutrophil	Positive Cocci
294	62	58	Negative	Negative	Neutrophil	Negative
146	36	68	Negative	Negative	Nil	Negative
286	64	52	Negative	Negative	Lymphocytes	Negative
316	60	54	Negative	Negative	Neutrophil	Negative
284	62	50	Negative	Negative	Lymphocytes	Negative

SP.AFB	CXR	LIG_CRI	LDH_CHOL	ETIOLOGY
Negative	Right Plef	Exudate	exudate	TB
Negative	Right Plef	Exudate	exudate	TB
Negative	Left Plef	Exudate	Exudate	Para Pneumonic
Negative	Right Plef	Transudate	Transudate	CCF
Negative	Right Plef	Exudate	Exudate	TB
Negative	Left Plef	Exudate	Exudate	Malignancy
Negative	Right Plef	Exudate	Exudate	TB
Negative	B/L Plef	Transudate	Exudate	CCF
Negative	Left Plef	Exudate	Exudate	Para Pneumonic
Negative	Right Plef	Exudate	Exudate	TB
Negative	Left Plef	Exudate	Exudate	Malignancy
Negative	Right Plef	Exudate	transudate	TB
Negative	Left Plef	Exudate	Exudate	Para Pneumonic
Negative	Right Plef	Transudate	Transudate	CIRRHOSIS
Negative	Left Plef	Exudate	Exudate	TB
Negative	B/L Plef	Transudate	Exudate	CCF
Negative	Right Plef	Exudate	Exudate	Empyema
Negative	B/L Plef	Transudate	Transudate	CIRRHOSIS
Negative	Right Plef	Exudate	Exudate	Para Pneumonic
Negative	Right Plef	Exudate	Exudate	TB
Negative	Left Plef	Exudate	Exudate	Malignancy
Negative	Right Plef	Exudate	Exudate	TB
Negative	B/L Plef	Transudate	Transudate	CIRRHOSIS
Negative	Right Plef	Exudate	Exudate	TB
Negative	Right Plef	Exudate	Exudate	Empyema
Negative	Right Plef	Exudate	Exudate	Malignancy
Negative	Left Plef	Exudate	Exudate	TB
Negative	Right Plef	Exudate	Exudate	Para Pneumonic
Negative	Left Plef	Exudate	Exudate	TB
Negative	Right Plef	Transudate	Transudate	CCF
Negative	Left Plef	Exudate	Exudate	Para Pneumonic
Negative	Right Plef	Exudate	transudate	TB
Negative	Right Plef	Exudate	Exudate	Para Pneumonic
Negative	Left Plef	Exudate	Exudate	TB
Negative	Left Plef	Exudate	Exudate	Malignancy
Negative	Right Plef	Exudate	Exudate	TB
Negative	Right Plef	Exudate	Exudate	Para Pneumonic
Negative	Right Plef	Transudate	Transudate	CIRRHOSIS
Negative	Right Plef	Exudate	Exudate	TB
Negative	Left Plef	Exudate	Exudate	TB
Negative	Left Plef	Exudate	Exudate	Malignancy
Negative	Right Plef	Exudate	Exudate	TB
Negative	Right Plef	Exudate	Exudate	TB
Negative	B/L Plef	Transudate	Transudate	CCF
Negative	Left Plef	Exudate	Exudate	Para Pneumonic
Negative	Right Plef	Exudate	Exudate	Para Pneumonic
Negative	Right Plef	Transudate	Transudate	CIRRHOSIS
Negative	Left Plef	Exudate	Exudate	TB
Negative	Left Plef	Exudate	Exudate	Para Pneumonic
Negative	Right Plef	Exudate	Exudate	TB