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Demographic Study of Pleural Effusion in a Tertiary Care Centre

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Abstract: ***Background: Pleural effusion is a common problem encountered day in and day out of hospitals and clinics worldwide. Pleural effusion affects approximately one million people every year. It can present either as complication of diseases or as their presenting features. The pleural space normally contains between 7 to 16 ml of fluid. When the rate at which formation of pleural fluid is more than the rate of removal accumulation of pleural fluid takes place. The categorization of the same as exudates and transudates is crucial to make the right diagnosis and manage accordingly. The most commonly used method is Light's criteria. Objective: To study the demographic pattern of pleural effusion and common cause of transudate and exudate in study population. Methods: A total of 101 subjects presenting with pleural effusion in the outpatient and emergency departments of Atal Bihari Vajpayee Institute of Medical Sciences and Ram Manohar Lohia Hospital, New Delhi were taken in the observational study. Pleural fluid samples obtained by diagnostic thoracocentesis were subjected to biochemical studies, namely pleural fluid protein measured by Biuret method, lactate dehydrogenase (LDH) measured by Reflective Spectrophotometric assay, Adenosine Deaminase (ADA) by Adazyme kit. The classification of pleural fluid into transudates and exudates based on the lights criteria. Results: The study analyzed that 74.26 % were males and 25.74 % were females. According to the Light's criteria, 54.46% were found to be exudates and 45.54% transudates.Mean value for age in exudate group was 39.56 years & transudate group was 54.59 years. Maximum number of exudates fall in the age group of 20-30years whereas maximum transudates had fallen in the group of 51-60 years. Most common cause of transudate in our study was Chronic liver disease whereas most common cause of exudate turned out to be tuberculosis.***

Keywords: pleural effusion, transudates, exudates, chronic liver disease, tuberculosis

1. Introduction

Pleural effusion is a presentation of many systemic and localized diseases. It is a common clinical problem which we encounter daily in our OPD and emergency. Evaluation of pleural fluid gives clue to the diagnosis of many disorders. Differential diagnosis of pleural effusion is wide so it is often considered as a diagnostic dilemma [1].

Pleural effusion affects approximately one million people every year. It can present either as complication of diseases or as their presenting features. It can lead to worse prognosis, if not diagnosed and treated properly [2]. The pleural space normally contains between 7 to 16 ml of fluid. When the rate at which formation of pleural fluid is more than the rate of removal accumulation of pleural fluid takes place [3]. Pleural effusion as initial manifestation in a patient without accompanying symptoms is a major diagnostic problem. If the pleural effusion is exudative then finding the cause is major clinical problem rather than differentiating transudative from exudative effusion.

Many criteria's have been used till date to distinguish them, but none of them have been found to be satisfactory. Most commonly used method is Light's criteria for identifying transudates and exudates. The criteria developed by Lights include simultaneous measurement of the blood protein and LDH levels as well as pleural fluid protein and LDH levels. Pleural fluid is said to be an exudate if one of the subsequent 3 criteria is fulfilled:

i) Value of protein in pleural fluid divided by serum protein > 0.5
ii) Value of LDH in pleural fluid divided by serum LDH making ratio > 0.6.
iii) Value of pleural LDH > 2/3rd of the higher limit of normal serum LDH

Light et al. in 1972 found that the criteria developed by him have sensitivity and specificity of ninety nine percent and ninety eight percent respectively, to distinguish transudate and exudate. But the other researchers were only able to reproduce specificities of seventy (70%) to eighty six percent (86%) after applying Light's criteria. The criteria have more specificity than sensitivity and it typically identifies 98% of pleural exudates, but misclassifies 25 % of the transudates as exudates [4]. As a result of which patients who are labelled as exudate undergo irrelevant and risky invasive procedures, such as image-guided percutaneous pleural biopsy and thoracoscopic pleural biopsy[5]. It has been seen that even after extensive workup up to 20 % of the cases remain undiagnosed.

2. Material and Methods

The study was conducted in department of medicine in Atal Bihari Vajpayee Institute of Medical Sciences and Ram Manohar Lohia Hospital, New Delhi (ABVIMS and DR RML hospital). It is cross sectional observational study.

Study Period- November 2017 to March 2020

Sample Size-The study of Leers MPG observed sensitivity and specificity of pleural cholesterol for identifying exudates was 76% and 98%. Taking these values as reference, the minimum required sample size with desired precision of 12%, 80% power of study and 5% level of significance is 86 patients. To reduce margin of error, total sample size taken is 101.

Inclusion Criteria
- Age >18 years (male and female)
- Newly diagnosed cases of pleural effusion.

Exclusion Criteria

- Patients without definite clinical diagnosis.
- Patients with pulmonary embolism and renal insufficiency.

3. Methodology

The study was conducted on 101 patients with pleural effusion presenting to the Department of Medicine and fulfilling inclusion and exclusion criteria.

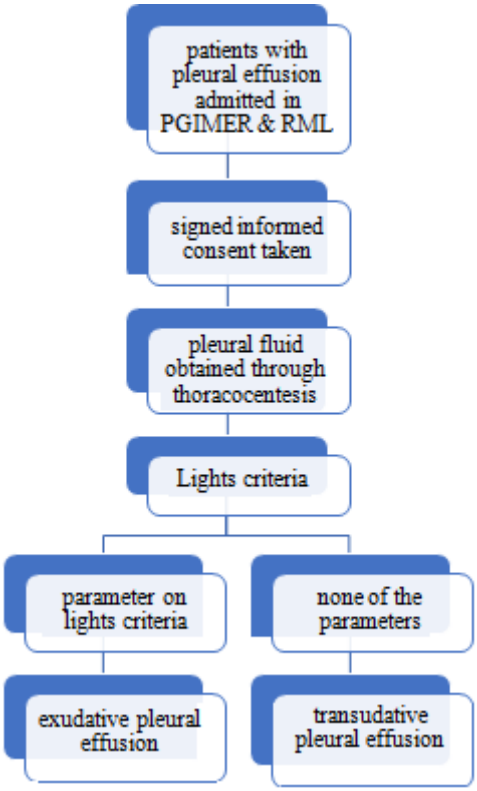
Patients after meeting inclusion and exclusion criteria and taken an informed consent underwent:

- Detailed history and clinical examination
- Blood investigations (complete haemogram, total protein, total cholesterol, LDH, renal function tests and liver function tests)
- Urine examination (routine and microscopy, culture sensitivity when needed)
- Chest radiograph postero-anterior view
- Electrocardiography
- Diagnostic Thoracocentesis under aseptic conditions in every case with the help of ultrasonography chest to localize the fluid wherever necessary.
- All pleural fluid samples will be tested for cell count, glucose, total protein, LDH, ,malignant cytology, ADA, gram stain.
- Ultrasonography chest and CT chest (when needed)
- FNAC to determine etiology whenever required
- Echocardiography when required

Effusions associated with congestive heart failure and liver cirrhosis are classified as transudates and the rest as exudates. Patients with renal disease and pulmonary embolism are excluded.

Table 1: Showing Methods of estimation of biochemical parameters of pleural fluid & serum

Parameter	Method of estimation
Protein	Biuret method
ADA	Adazyme kit
LDH	Reflective Spectrophotometric assay



4. Results

The aim of our study was to see demographic distribution of pleural effusion and classify the fluid as exudate or a transudate by lights criteria after parameters obtained by diagnostic thoracocentesis. It was an observational study and as per statistical analysis, a sample size of 101 was calculated.

Out of 101 patients included in the study, 74.26 % were males and 25.74 % were females [FIGURE 1].

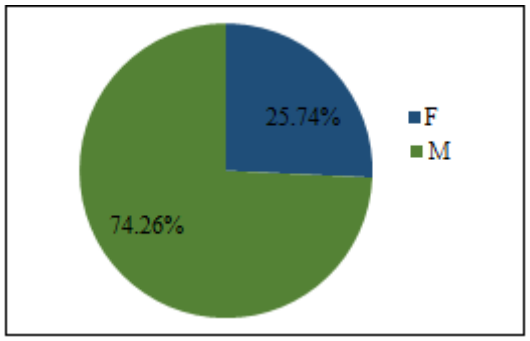


Figure 1: Depicting gender wise distribution of entire study population in which 74.26 % are males and 25.74 % are females

53.85% of females had an exudative effusion and 46.15% had a transudative effusion. Among the males, 54.67% and 45.33% had exudative and transudative effusions respectively. [FIGURE 2]

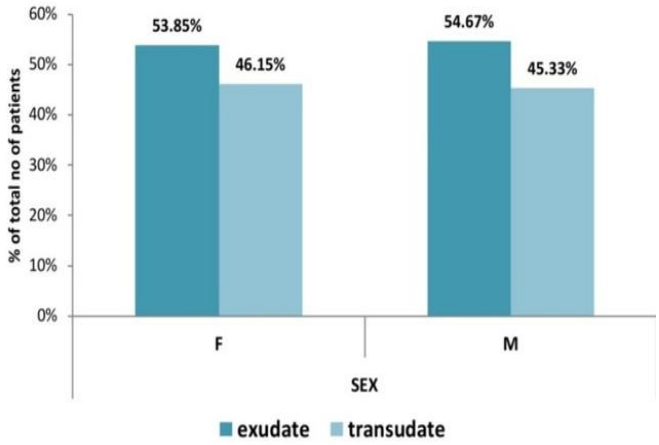


Figure 2: Showing Gender wise distribution of exudates & transudates

M- male
F- female

We studied the frequencies according to various cut offs for ages. For 20-30 years it was 20.79%, for 30-40 years 16.83%, for 41-50 years 23.76%, 51-60 years 24.76% and for 61-70 years 13.86%. The highest percentage of patients was found to be in the age group of 41-50 yrs.[FIGURE 3] Mean value for age in exudate group was 39.56 years &transudate group was 54.59 years.[FIGURE 4]

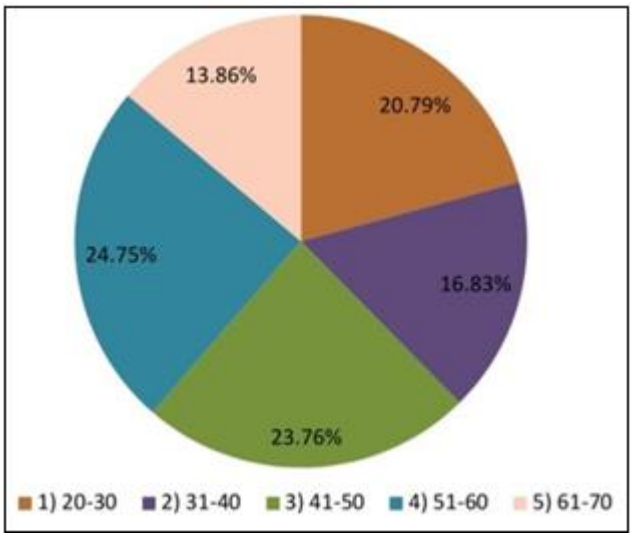


Figure 3: Percentage of total population at different age groups

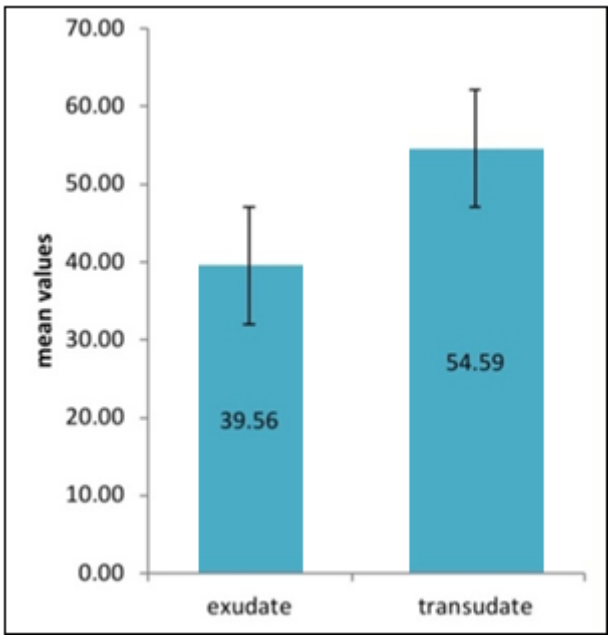


Figure 4: Showing mean values for age among exudate and transudate groups

Maximum number of exudates fall in the age group of 20-30years whereas maximum transudates had fallen in the group of 51-60 years. [Figure 5]

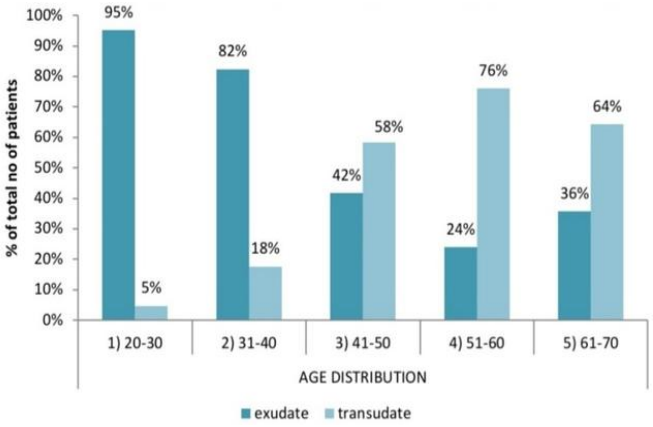


Figure 5: Percentage of transudates and exudates at different cut offs for various age groups

According to the Light's criteria, 54.46% were found to be exudates and 45.54% transudates. [FIGURE 6]. The most common cause of transudate was chronic liver disease whereas, the most common cause of exudate turned out to be tuberculosis. [Figure 7]

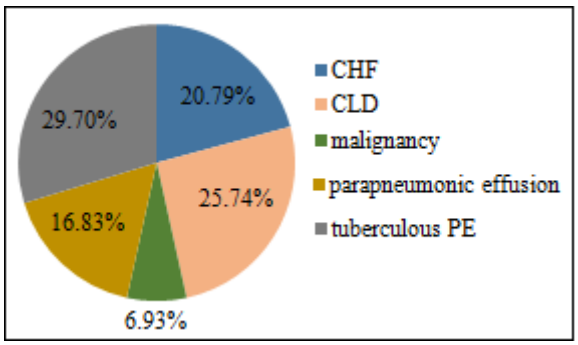


Figure 7: Showing percentages of etiological diagnosis of study population

5. Discussion

Pleural effusion is defined as the abnormal accumulation of pleural fluid within the pleural cavity. This is a potential space between the parietal pleura and the visceral pleura. It is the coupling system between the lung and the chest wall. The pleura is a type of serous membranous that lines the parenchyma of lungs, mediastinum, diaphragm, and the rib cage. Pleura are of two types parietal pleura and visceral pleura. The parietal pleura is the one which lines the thoracic cavities & is again divided on the basis of intrathoracic structures that it lines. The visceral pleura lines the parenchyma of lung, diaphragm, mediastinum and fissures in between lobes1.Pulmonary ligament is a double fold of pleura which is formed due to inferior extension of pleura, posterior to the lung root. Between the parietal and the visceral pleura a thin film of fluid exists normally which acts as lubricant and allows the visceral pleura to slide over the parietal pleura.

In parietal pleura there is a layer of loose irregular connective tissue consisting of mesothelial cells. Endothoracic fascia is a continuous band of irregular connective tissue .Within the pleura there are lymphatic lacunas & blood capillaries. The visceral pleura contain thick tissue, which is made of mesothelial cells, lymphatics and blood vessels. It contributes to the elastic recoil of the lung, which is important to expel air out of the lung. It also restricts the volume to which the lung can be inflated there by offering a protective mechanism. The mesothelial layer is a fragile mono layer with active pavement like mesothelial cells are lining the pleural surfaces that are sensitive to various stimuli. These cells can transform to macrophages and can express procoagulant activity due to tissue factor that binds the factor VII at their cell surface. Other functions of these cells include movement and transport of particulate matter and fluid across the pleural surfaces, migration of leukocytes in response to an inflammation, cytokines growth factors & EC (extracellular matrix protein) synthesis antigen presentation & transformation into myofibroblasts.

Pleural fluid formation takes place because of accelerated formation or shrivelled absorption or both.

1) Increased pleural fluid formation

Whenever there is increased pulmonary interstitial fluid or when one of the terms in Starling's equation is changed, there is increased pleural fluid formation.

A) Increased interstitial fluid formation

Whether the oedema is high protein or low protein pleural fluid starts to accumulate when the amount of fluid exceeds 5g per gram of ling dry weight. This occurs in

- Congestive cardiac failure
- Para pneumonic effusion
- Acute respiratory distress syndrome
- Lung transplantation

B) Increased hydrostatic pressure gradient

In the parietal pleura, the hydrostatic pressure is approximately 30 cm H2O, but the pleural pressure is approximately -5 cm H2O. The net hydrostatic pressure is therefore 30-(-5) = 35 cm H2O,due to this pressure movement of fluid occurs from the capillaries in the parietal pleura to the pleural space.

If there is an increased pressure gradient between the intravascular pressure and pleural pressure there will be an increase in the rate of pleural fluid formation. Seen in

- Right ventricular failure
- Left ventricular failure
- Pericardial effusion
- Superior vena caval obstruction

C) Increased capillary permeability

Inflammation of the pleural surface causes increase in capillary permeability leading to pleural fluid formation. VEGF is responsible for increased permeability of capillaries. Mesothelial cells have VEGF receptors on their surface and in exudative effusions the levels of VEGF are higher when compared to transudative pleural effusions.

D) Decreased oncotic pressure gradient

The hydrostatic pressure is opposed by oncotic pressure gradient. Plasma oncotic pressure is approximately 34 cm H2O. Normally the pleural fluid has oncotic pressure of approximately 5 cm H2O as it contains a small amount of protein. The net oncotic pressure gradient is 34-29 = 6cm H2O which favours the movement of fluid from the capillaries in the parietal pleura to the pleural space. It has been demonstrated that the net gradient for fluid movement across visceral pleura is zero in humans. The visceral pleural capillaries have less pressure than the parietal pleural capillaries because the visceral pleural capillaries are drained by pulmonary veins. Visceral pleural capillaries are far from the pleural space as compared to parietal pleura which leads to the low filtration coefficient of visceral pleura. Also fluid formation was more over the caudal ribs than over the cranial ribs. On the contrary, pleural fluid absorption was more in the parietal pleura adjacent to the intercostal space as compared to the parietal pleura overlying the ribs. The formation of pleural fluid was more, if the breathing frequency was increased. Decreased oncotic pressure gradient leads to the formation of pleural fluid through its influence on the Starling's equation. E.g. hypoproteinemic states.

E) Disruption of the thoracic duct or intrathoracic blood vessel

The free fluid in the peritoneal cavity traverse through the holes in the diaphragm and it will lead to pleural fluid accumulation and pleural effusion. There is collection of chyle in pleural space due to disruption of thoracic duct and blood after blood vessel disruption.

2) Decreased pleural fluid absorption

A) Lymphatic obstruction

In pleural space majority of absorption occurs through lymphatics. Normally the lymphatic flow from the pleural space is about 0.01ml / kg / hour or 15 ml / day, but the capacity of the lymphatics is about 0.20ml per kg per hour or 300 ml / day10. Obstruction to lymphatics is most commonly seen in malignant effusion where the lymphatics are obstructed by malignant cells.

B) Elevation of systemic venous pressure
When there is elevation of the pressures in the central veins, lymphatic flow will be decreased. This is because the lymphatics drain into the systemic venous circulation.

The pleural effusions develop because of (a) leakage of lymph out of the lymphatics that pass through the chest (these include the diaphragmatic and pulmonary lymphatics and thoracic duct) or (b) leakage of interstitial fluid into the pleural space due to obstruction of lung or chest wall lymphatics.

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