“CLINICAL AND ETIOLOGICAL STUDY OF PLEURAL EFFUSION, ONE YEAR LONGTUDINAL STUDY”

By

REG NO. BG0116012

Dissertation

Submitted to the
KLE Academy of Higher Education and Research,
Belagavi, Karnataka
In partial fulfillment
of the requirements for the degree of

M. D.
in
GENERAL MEDICINE

DEPARTMENT OF MEDICINE,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
BELAGAVI, KARNATAKA

APRIL – 2019

KLE Academy of Higher Education and Research, Belagavi, Karnataka

ENDORSEMENT

This is to certify that the dissertation entitled **"CLINICAL AND ETIOLOGICAL STUDY OF PLEURAL EFFUSION, ONE YEAR LONGTUDINAL STUDY"** is a bonafide research work done by **(REG NO. BG0116012)**

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LIST OF ABBREVIATIONS USED

PE	:	Pleural Effusion
TB	:	Tuberculosis
HTN	:	Hypertension
DM	:	Diabetes Mellitus
IHD	:	Ischemic Heart Disease
RHD	:	Rheumatic Heart Disease
PEM	:	Protein Energy Malnutrition
RVD	:	Retroviral Disease
CXR PA	:	Chest X ray Postero-Anterior View
LDH	:	Lactate Dehydrogenase
ADA	:	Adenosine Deaminase
AFB	:	Acid Fast Bacilli
CLD	:	Chronic Liver disease
CKD	:	Chronic Kidney Disease
CCF	:	Congestive Cardiac Failure

ABSTRACT

Introduction:

In the present study of 100 patients with Pleural Effusion admitted in Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre in the study period from January 2017 to December 2017 was undertaken to find the aetiology of Pleural Effusion.

Materials and Methods:

All Patients admitted to wards of Department of General Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi with Pleural Effusion, were subjected for Pleural Fluid Analysis.

Observations and Conclusion:

The results observed on analysing the pleural fluid were majority of patients with exudative effusions in which most of the patients were tubercular followed by Parapneumonic effusions. In our study there was a male preponderance with a male to female ratio of 2.125 to 1 (68 males, 32 females).

We applied Light's criteria to find out type of effusion. Maximum number of patients had exudative effusion of which commonest etiology was found to be Tuberculosis in 49 Patients (49%) There was a positive correlation between Pleural LDH and Pleural ADA, and Pleural ADA with advancing age.

Keywords:

Pleural Effusion, Light's Criteria

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INTRODUCTION

Accumulation of fluid in the pleural cavity is known as pleural effusion and also sometimes referred as water in the lung. Pleural cavity is the narrow space filled with fluid between the pulmonary pleurae. The intrapleural pressure is mostly subatmospheric, and it varies at different levels of the pleural cavity[1]. Pleural effusion is a common clinical condition which is more often a manifestation of various primary or secondary disorders.

The estimated prevalence of pleural effusion is 320 cases per 100,000 people in industrialized countries, with a distribution of etiologies related to the prevalence of underlying diseases[2]. Leading cause of pleural effusion in the US is CHF, with an estimated incidence of 500,000 followed by Pneumonia is second with an incidence of 300,000[3]. In Southwest Nigeria, pleural diseases accounted for 7.7% of respiratory morbidity with the majority between the ages of 15 and 44 years[4]. Substantial pleural effusion is more often reported as the result of malignant disease in 67% of cases, most commonly lung or breast carcinoma[5]. Approximately 40% of the hospitalized patients with pneumonia have an associated parapneumonic effusion[6]. Pulmonary embolus, viral disease, coronary artery bypass surgery, and cirrhosis are also common causes of effusion. Small pleural effusions are present in up to 40% of patients with pulmonary embolism. Of all patients with cirrhosis, 5% have an associated pleural effusion[7]. Additional to all the causes, TB is also an important cause, especially in the low and middle income countries. Previous studies have reported associations between mortality and pleural effusion in few specified populations. Bilateral pleural effusions were identified as the strongest independent predictors of mortality for patients who were admitted with community acquired pneumonia (CAP)[8]. Shaw P et

al., have reported that half of all patients with metastatic malignancies develop pleural effusions[9].

Causes actually vary with age groups, among adult' sheart failure, malignancy, pneumonia, tuberculosis, and pulmonary embolism, whereas pneumonia is the leading etiology among children across the globe[3]. Accumulation of pleural fluid might not be due to specific disease, but rather due to presence of various underlying causes.

Clinical presentation of pleural effusion is generally dependent on the quantity of the fluid present and majority of the patients have no typical symptoms at the time of diagnosis. Most commonly reported symptoms include pleuritic dyspnea, chest pain and on productive cough. The chest painduring pleural effusion is usually due to inflammation of the pleura which is caused due to friction between the surfaces[10].

The history and physical examination are critical in guiding the evaluation of pleural effusion. Taking a careful history and conducting a complete physical examination would yield information not only about the presence of the pleural effusion but also about its underlying cause. Chest radiographs are usually sufficient to confirm the effusion, but ultrasound or computed tomography (CT) scans are absolute techniques for detecting small effusions and for differentiating pleural fluid from pleural thickening.

Pleural fluid analysis is considered to be an important aspect in the diagnosis, as the effusions might be transudates or exudates based, which would be help in assessing the physiology of the formation. Exudates occur due to alterations in the factors which are important role in pleural fluid accumulation. Measurement of protein content of pleural effusion is essential in distinguishing between transudate and exudate[11]. Exudates are usually differentiated from transudates based on the

Light`s criteria. Other diagnostic procedures like bronchoscopy, percutaneous biopsy and thoracoscopy are frequently used.

Managament of pleura effusion majorly involves, draining of the excess accumulated fluid, surgical and therapeutic management and pleurodesis. Treatment of the underlying cause is always beneficial in treating most of the transudative effusions. Through the present longitudinal study, an attempt is made to assess the clinical and etiological features of pleural effusion.

AIMS AND OBJECTIVES

- To assess the clinical and etiological profile of patients presenting with pleural effusion to a tertiary care teaching hospital

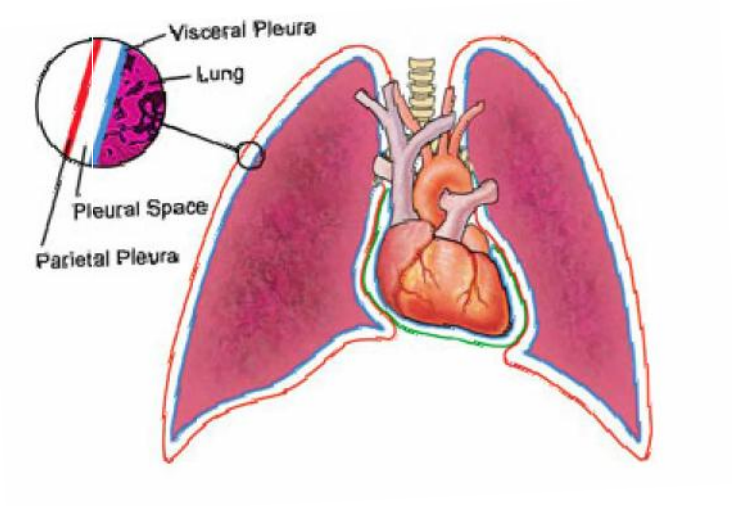
- Clinical Study and Etiology of Patients with Pleural Effusion at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre.

REVIEW OF LITERATURE:

Anatomy and physiological role of pleura

The thoracic cavity is divided into right pleural cavity, mediastinum and left pleural cavity. The pleural cavity is the space lined by a serous membrane called the pleural membrane. The membrane covers both the lungs and the thoracic wall and the space between the two membranes is the pleural cavity. The pleura cavity is filled with the pleural fluid which is acts as a lubricant between the layers. Membrane which is attached to the lobes of lungs is called as visceral pleura and the membrane covering wall of the outer cavity is called as parietal pleura.

Fig: Anatomy of the pleural cavity



The visceral pleura receives its blood supply from both bronchial and pulmonary artery branches, whereas the parietal pleura is supplied by the systemic circulation only. The visceral pleura is drained by the pulmonary veins, and the parietal pleura is drained by intercostal and bronchial veins. Lymphatic drainage of the visceral pleura is toward the hilar nodes, and that of the parietal pleura is toward the mediastinal glands[11].

The pleural cavity plays an important role in optimal functioning of the lungs especially during heavy breathing. It transmits movements of the chest wall to the lungs. Chest wall transmits pressures to the visceral pleural surface and to the lungs[12, 13]. The visceral pleura receives its blood supply from the bronchial circulation and the parietal pleura receives its blood supply from the intercostal arteries.

Common etiologies of pleural effusion

Infective conditions

Anie, Y et al[14]., have carried out a study, with an objective to develop non-bacteriologic methods and to assess their potential utilities for the rapid diagnosis. A total of one hundred forty patients with pleural effusion were investigated for tuberculous etiology by bacteriologic methods. Mycobacterium tuberculosis in the pleural fluid specimens was isolated in 11 patients. Estimation of TBGL antigen by ELISA showed 100% specificity and overall 85.5% sensitivity. Immunocytochemistry could be applied only in those samples with adequate number of macrophages. PCR carried sensitivity and specificity of 87% and 93%, respectively. From the results, authors have stated that. Estimation of TBGL antigen in pleural fluid specimens by ELISA has a definite role in establishing tuberculous etiology, particularly in those patients in whom bacteriologic methods did not demonstrate M. tuberculosis.

To determine the morbidity and mortality related to respiratory diseases among adults attending a tertiary-care hospital in Nigeria, Desalu OO et al[4]., carried out a retrospective study among183 adult patients diagnosed with respiratory diseases. Results showed that, pulmonary TB, asthma, pneumonia and pleural pathologies to be the most common in women, whereas COPD was more common in men. The most common comorbidity was HIV infection. Mortality was higher in women and in the

25-44 year age bracket. From the findings, authors have come to a conclusion that, pulmonary TB, asthma and pneumonia were the leading causes of respiratory disease-related morbidity.

Tumors Malignant conditions

Agrawal A et al[15]., through their study, have made an attempt to find out the incidence of malignant pleural effusion, its aetiology and clinical course in patients attending a tertiary care teaching hospital. A total of 308 patients were included in this study. The major primary cancers were found to be, lung cancer (135), lymphoma (40), breast cancer (36), female genital tract (30) gastrointestinal (21), and others (8). The yields of pleural fluid cytology, blind pleural biopsy, CT/USG guided pleural biopsy and thoracoscopy were 60%, 49%, 76% and 91% respectively. Authors have concluded that, malignant pleural effusion is a commonly misdiagnosed medical entity and lung cancer is the commonest cause. Even after all the interventional measures, in about 15% of the cases, primary remains undiagnosed.

Pleural effusions are rarely associated with PCM and most often signify a concurrent disease process. Malignant myelomatous pleural effusions are even more unusual and carry a poor prognosis. Babu KA et al[16]., have reported a unique case of unsuspected PCM with thoracic involvement in the form of massive left side pleural effusionin 48-year-old man from North East India. Pleural fluid cytology revealed numerous atypical plasma cells. Subsequently on further workup, urine Bence Jones protein was positive. Bone marrow aspiration and biopsy and computed tomography of the chest and abdomen revealed features consistent with multiple myeloma. Through this case, authors have concluded that, presence of atypical plasma cells in the pleura may provide useful evidence for the diagnosis of PCM. Myelomatous

pleural effusion is a rare manifestation of the disease, as by itself it indicates an advanced stage of the disease and carries a poor prognosis.

Gadewad N, et al[17]., have studied the clinical profile of100 patients presenting with malignant pleural effusion, their cytological and histopathological features and the efficacy of pleurodesis in preventing recurrence. Most common presenting symptoms were breathlessness (86%) and cough (86%). All (100%) of the malignant pleural effusions were exudative. Pleural fluid cytology was positive in 86% while pleural biopsy was positive only in 44%. Pleural biopsy was positive only in 17% of patients with negative cytology. Adenocarcinoma (59%) was found to be the most common type of cytological diagnosis. Based on the study findings, authors have concluded that,most common presenting symptoms were breathlessness and cough and they were exudative, lymphocytic predominant with low ADA levels. Adenocarcinoma of the lung was the most common cause of malignant pleural effusion.

Trauma

Pleural effusion is a potential complication following blunt splenic injury. Kulaylat AN et al[18]., have performed a ten-year retrospective review on children with blunt splenic injury. Of 274 evaluable non-operatively managed pediatric blunt splenic injures, 12 patients (4.4%) developed left-sided pleuraleffusions. Seven (58%) of 12 patients required left-sided tube thoracostomy for worsening pleural effusion and respiratory insufficiency.. Median length of stay was 4days for those without and 7.5days for those with pleural effusions ($p<0.001$) and 6 and 8days for those pleural effusions managed medically or with tube thoracostomy ($p=0.006$), respectively. In multivariate analysis, high-grade splenic injury (IV-V) (OR 16.5,

p=0.001) was associated with higher odds of developing a pleural effusion compared to low-grade splenic injury (I-III).From the study findings, authors have come to a conclusion that pleural effusion following pediatric blunt splenic injury has an incidence of 4.4% and is associated with high-grade splenic injuries and longer lengths of stay.

Unusual presentations

Imatinib is a tyrosine kinase inhibitor and has rarely been reported as a potential causal factor for pleural effusion Banka R, Udwadia Z[19] have presented a case of an 88-year-old male, known case of gastrointestinal stromal tumour on treatment with imatinib, who presented with a 2-week history of cough and dyspnea. He was diagnosed to have a right-sided pleural effusion and thoracentesis of the fluid revealed an exudate with low adenosine deaminase and negative cytology. Withdrawal of the drug lead to resolution of symptoms. Through this case study, authors have made an attempt to highlight the side effect profile of imatinib and warn physicians regarding this potential adverse effect which may be mistaken for metastasis or infection.

The occurrence of microfilaria in pleural fluid is rare. Chaturvedi A and Kumar A.[20], have reported a case of 74-year-old male patient, non-smoker who was admitted to our hospital with breathlessness and chest discomfort of two weeks duration. He had, eosinophilia and deranged renal function. X-ray chest revealed massive left sided pleural effusion. Pleural fluid analysis revealed atypical cells and pleural fluid cytology showed microfilaria (Wuchereria bancrofti), which were also found on peripheral smear.

Arora VKand Gowrinath K[21], have reported a case of pleural effusion is reported. A 60-year-old illiterate female with malaise, vague abdominal symptoms since 7 months and dry cough with low grade intermittent fever. Chest x-ray revealed a moderate effusion on left side. The pleural biopsy showed presence of microfilariae suggestive of WuchereriaBancrofti. Treatment with diethyl carbamazine have yielded excellent results. Authors have also suggested that, filarial aetiology should be included in the differential diagnosis of idiopathic pleural effusions, especially in endemic areas.

Sahlu Gupta et al[22]., have reported a case of late onset ovarian hyperstimulation with bilateral pleural effusion and respiratory distress as the sole manifestation after embryo transfer. A 21 year-old female married at the age of 18 years attended the Institute of Reproductive Medicine out patient clinic with secondary subfertility of 2 years, irregular cycles and polycystic ovaries. Ultrasonography (both transvaginal and transabdominal) of the abdomen and chest showed bilateral enlarged ovaries (90 × 85 mm and 85 × 54 mm) with no fluid collection in the pelvis. However, there was evidence of bilateral pleural effusion. From the findings, authors have stated that, the frequency of OHSS presenting with an isolated pleural effusion is more often underestimated. In any patient diagnosed to have pleural effusion following controlled ovarian stimulation, OHSS should be considered as the most likely cause.

Fine needle aspiration and biopsy forceps are the tools mostly used by pulmonary physicians and radiologists. During these procedures pneumothorax can occur and immediate treatment is necessary. Jaggi S et al[23]., in their stud have stated that, in order to reduce the complications such as pneumothorax, every operator should know more about anatomic structure, characteristics in the specific situation,

skills of selected tools or methods. While choosing the right patients with suitable instruments at the right place and time for proper operation, the operators would really improve the positive rate and security.

Sarcoidosis is a multisystem granulomatous disease of unknown etiology. Pleural involvement is relatively rare. Development of pleural effusion in sarcoidosis needs to be evaluated for other causes, especially tuberculosis in endemic countries. Sarcoid pleural effusion responds to systemic corticosteroids. Joshi S et al[24], have presented a case of 42 year old male patient of sarcoidosis who developed massive pleural effusion while on treatment with steroids, which was attributed to disease per se. Sarcoidosis as a cause of massive pleural effusion has not been mentioned before in published literature. From the case findings, authors have suggested that, pleural effusion in all sarcoidosis patients must be appropriately confirmed with pleural biopsies and culture studies to distinguish between pleural involvement due to sarcoid or tuberculosis.

Subpleural and mediastinal lipomatosis are benign intrathoracic conditions discovered on plain chest radiographs. However, diagnosis is usually made by Computed Tomography (CT). Kaur N et al[25] presented a case of both mediastinal and pleural lipomatosis associated with use of steroids in a 58-year-old male patient presented with complaints of breathlessness and dry cough. Initially, pleural effusion and bilateral upper lobe patchy opacities were suspected as a cause of breathlessness on the basis of chest x-ray findings. For which, CT scan of chest was advised.Pleural and mediastinal lipomatosis occurring simultaneously in same patient is a rare entity and pleural lipomatosis more often acts a mimicker of pleural effusion. Through this case study, authors have suggested that, lipoma should be considered as one of the common aetiologies before confirming it as a case of idiopathic effusion.

Kumar J et al[26]., have reported a case of contralateral pleural effusion secondary to malposition of PICC line in an extremely preterm neonate .A preterm neonate born at 27 weeks, with a birth weight of 555 g, was on continuous positive airway pressure (CPAP) for apnoea of prematurity and initially received total parenteral nutrition (TPN) through the umbilical venous catheter. Peripherally inserted central catheter (PICC) was inserted in the left basilica vein on day 8 to continue TPN. The baby developed respiratory distress with persistent hypoxia after TPN was initiated through the PICC line. The baby required mechanical ventilation due to worsening of respiratory distress, and chest X-ray, as well as ultrasound conducted 12 hours, postinfusion of TPN revealed right-sided pleural effusion. On careful observation, PICC was traced in the right lung area. The PICC line was removed immediately and the baby improved over the next 18 hours and was extubated to CPAP within the next 48 hours.

Kumar S et al[27], have reported a rare case due to the rare manifestation in pulmonary sarcoidosis presenting clinically as hemorrhagic pleural effusion.A 53-year-old female bidi smoker was admitted to our department with complaints of loss of appetite and left-sided chest pain for 5 months. On examination, the patient was alert and well oriented. She was not in obvious distress. Her vital signs were stable. The only significant finding on examination of the chest was reduced breath sound at the left lower axillary area with bi basilar crept in the infrascapular area. Sarcoid pleural effusions may resolve spontaneously or require corticosteroids for resolution. The time of spontaneous resolution is variable, but most resolve in 1-3 months. In the present case, corticosteroid therapy resulted in marked improvement of the pleurisy as well as the parenchymal infiltrates

Light RW[6]., have stated in their study that, The mortality rate in patients with a parapneumonic effusion is higher than that in patients with pneumonia without a parapneumonic effusion. Parapneumonic effusions occur in 20 to 40% of patients who are hospitalized with pneumonia. Some of the excess mortality is due to mismanagement of the parapneumonic effusion. If the fluid cannot be removed with a therapeutic thoracentesis, a chest tube should be inserted and consideration be given to the intrapleural instillation of fibrinolytics. If the loculated effusion persists, the patient should be subjected to video-assisted thoracoscopic surgery, and if the lung cannot be expanded with this procedure, a full thoracotomy with decortication should be performed.

Mitra S et al[28]., in their case report, have presented a case of a 35-year-old female patient admitted with the chief complaints of a cough and shortness of breath for the past 4 months and heaviness and left-sided chest pain for 20 days. A cough was mostly dry without any associated expectoration or hemoptysis. Shortness of breath was insidious in onset and gradually increased to modified medical research council Grade 3. A black pleural effusion with squamous cells in the pleural fluid caused a suspicion of an unusual etiology. A careful evaluation of CT revealed some fat densities in the multiloculated mass associated with the effusion enabling us to make a tentative diagnosis of a mature cystic teratoma before referring her for an urgentthoracotomy.Authors have reiterated the CT findings of soft tissue, fat, and calcification which helped in the management of this patient.

Diagnosis of pleural effusion role of various modalities

Chinchkar NJ, et al[29]., have conducted prospective study on 50 patients, diagnosed with pleural effusion to establish an etiologic diagnosis in a series of such patients before starting treatment. Etiologic diagnosis of pleural effusion was established in 44 (88%) Metastases (24%), para-pneumonia (22%), congestive cardiac failure (18%), tuberculosis (14%, hemothorax (4%), trapped lung, renal failure, and liver cirrhosis (2% each).Out of the 50 patients, 10 died in the hospital, 2 left against medical advice, and 2 were referred to oncology center for further treatment. The remaining 36

patients were clinically stabilized and discharged. During a 3-month follow-up, eight of them were re-hospitalized, of which four died. From the study findings, authors have concluded that, pleural effusion in RICU carries a high risk of mortality.

Imaging modalities

Sharma M et al[30]., have stated that, the most efficient and cost-effective approach for the diagnosis of pleural exudates remains uncertain and is a subject of controversy. Essential factors to be considered include the respective diagnostic yields of thoracocentesis, closed pleural biopsy, and thoracoscopy. The role of endoscopic ultrasound (EUS) of the oesophagus as a modality for the evaluation of pleural exudates has not yet been evaluated. The applied anatomy of the pleura has been discussed. The techniques involved in the EUS imaging of different aspects of the pleura in normal cases and in cases with pleural effusion are elaborated. The practical application of this knowledge can be useful in EUS-guided sampling of the pleural wall, pleural nodules, and in cases of pleural effusion.

Hasley PB et al[8]., have carried out a prospective cohort study to determine whether pulmonary radiographic findings at presentation are independently associated with 30-day mortality in patients with suspected CAP.Out of the total 2287 patients included in the study, 1906 patients (83.3%) had a pulmonary radiographic infiltrate confirmed by the radiology panel. Overall, 30-day mortality in this cohort was 4.9%. Following radiographic characteristics were found to significantly associated with 30-day mortality, 1) bilateral pleural effusions (RR, 7.0, 95% CI, 3.9-12.6), 2) a pleural effusion of moderate or greater size (RR, 3.4, 95% CI, 1.4-8.4), 3) 2 or more lobes involved with infiltrate (RR, 2.5, 95% CI, 1.6-3.8), 4) bilateral infiltrate (RR, 2.8, 95% CI, 1.9-4.2), 5) bronchopneumonia (RR, 1.6, 95% CI, 1.0-2.7), and 6) air bronchograms (RR 0.5, 95% CI, 0.2-0.9). Multivariate analysis of radiographic

features and other clinical characteristics showed the presence of bilateral pleural effusions was independently associated with mortality.From the findings, authors have concluded that, in patients with CAP, the presence of bilateral pleural effusions is an independent predictor of short-term mortality.

Pleural fluid analysis (Biochemical, microbiological and immunological tests)

Sharma SK and Banga A[31] have carried out a study on 101 patients with pleural effusion, to assess the diagnostic utility of IFN-gamma level estimation in patients with TB pleural effusion and to define the best cut off of IFN-gamma for diagnosis TB pleural effusion. Measurement of pleural fluid IFN-gamma levels was done by ELISA technique. The median value of pleural fluid IFN-gamma levels in patients with TB (1480 pg/ml, range 3-14,000 pg/ml) was significantly higher ($p < 0.001$) compared with the non-TB group (3 pg/ml, range 0-900 pg/ml). The receiver operator characteristic (ROC) curve for IFN-gamma showed an area under the curve (AUC) value of 0.954, and the best cut off was computed to be 138 pg/ml. Using this cut off for IFN-gamma levels in pleural fluid for the diagnosis of TB, sensitivity, specificity, negative predictive value, and positive predictive value were found to be 90.2%, 97.3%, 85.7%, and 98.3%, respectively. Through this study, authors have suggested that, estimation of IFN-gamma levels in pleural fluid is a useful diagnostic modality for TB pleural effusion. A cut off of 138 pg/ml provides the best sensitivity and specificity for diagnosis of TB.

Wu H et al[32]., have conducted a quality improvement initiative using 74 samples from 60 individual patients, to determine the volume of fluid sufficient for a diagnosis of malignant pleural effusion. Thirty-six patients (60%) had a previous diagnosis of malignancy. Of the 74 specimens, 26 (35.1%) were positive for malignancy. The detection rate for malignant pleural effusion by cytology for 25 mL,

50 mL, and 150 mL were 88.5%, 96.2%, and 100.0%, respectively (P = 0.16). Two specimens that were negative in the 25 mL samples turned out to be positive in the 50 mL and 150 mL samples. One specimen was negative in the 25 mL and 50 mL samples but positive in the 150 mL sample. Authors have stated that, no statistically significant difference was observed in the detection of malignant effusion in the 25 mL, 50 mL, and 150 mL group.

Bansal A et al[33]., conducted a study in 30 patients of malignant pleural effusion and 30 patients of non-malignant pleural effusion. Sialic acid levels were estimated in serum and pleural fluid by Warren's TBA method. Serum sialic acid levels were found to be higher in group II as compared to group I. Smokers in group II had higher serum sialic acid as compared to group 1 (P < 0.05). The sensitivity and specificity of pleural fluid/serum sialic acid ratio with cut off value of 0.7 were 76.67% and 20% respectively, while taking the cut off value of 70 mg/dL for pleural fluid sialic acid in malignant pleural effusions, the sensitivity was 63.33%, specificity 60% and positive predictive value 46.34%. From the study findings, authors have concluded that, determination of sialic acid levels in pleural fluid has diagnostic value as a cheap, simple and reliable marker for malignant pleural effusion.

Pleural biopsy

Welagedara S et al[34]., have reported a case of an elderly Asian man where a medical error and diagnostic delays obscured the diagnosis of pleural tuberculosis (TB). The patient was hospitalized for evaluation of a unilateral pleural effusion. Initially, the patient was subjected to a pleural aspiration on the wrong side due to a lack of bedside ultrasound guidance. Subsequently, the patient underwent several investigations but not a blind closed pleural biopsy (BCPB) due to a lack of equipment. Furthermore, the patient was deemed to be too sick to undergo a

thoracoscopic pleural procedure. Eventually, a bronchoscopy was performed, and washings from the right upper lobe were cultured, which established the diagnosis of TB. Through this case, authors have made an attempt to highlight the need to use bedside ultrasound in the investigation of pleural effusions, the role of BCPB especially in frail patients and finally the utility of bronchoscopy in establishing a diagnosis of pleural TB.

Biswas B et al[35]., carried out a prospective study to evaluate the role of pleural fluid analysis in diagnosing pleural diseases and to study the advantages and disadvantages of thoracocentasis and pleural biopsy. A total of 66 indoor patients over followed a duration of 1 year. Pleural fluid was collected and cytological smears were made from the fluid. Plural biopsy was done in the same patient by Cope needle. Tuberculosis was the commonest nonneoplastic lesion followed by chronic nonspecific pleuritis comprising 60% and 33.3% of the nonneoplastic cases respectively and tuberculosis was predominantly diagnosed in the younger age group. Majority (70.8%) of malignancy cases were in the age group of >50-70. Adenocarcinoma was found to be the commonest (66.7%) malignant neoplasm in the pleurae followed by small-cell carcinoma (20.8%). Based on the study findings, authors have concluded that, pleural biopsy is a useful and minimally invasive procedure.

To assess the diagnostic yield and safety of closed pleural biopsy in patients with pleural effusion, James P et al[36]., have evaluated 48 consecutive cases of pleural effusion. In all these 48 cases of pleural effusion closed pleural biopsy was done with tru-cut biopsy needle and biopsy samples were sent for histopathology and mycobacterial culture. Out of 48 cases, main causes of pleural effusion were tuberculosis in 21(43.8%) cases, malignancy in 14 (29.2%) cases, paramalignant

effusion in six (12.5%) cases, empyema in three (6.3%) cases, transudative effusion in three (6.3%) cases and parapneumonic effusion in one (1.9%) case. Diagnostic yield of closed pleural biopsy was 62.2% in cases of all exudative pleural effusion, 76.2% in cases of tubercular pleural effusion and 85.7% in cases of malignant pleural effusion. Authors have come to a conclusion that, closed pleural biopsy provides the highest diagnostic yield in cases of pleural tuberculosis and malignancy, the two most important causes of exudative pleural effusion.

Nattusamy L et al[37]., have performed systematic review of studies reporting the utility of semi-rigid thoracoscopy from India. A total of 48 patients underwent semi-rigid thoracoscopy. Pre-procedure clinico-radiological diagnoses were malignant pleural effusion (36 patients (75%)), tuberculosis (TB) (10 (20.83%) patients), and empyema (2 patients (4.17%)). Fourteen (29.17%) patients were diagnosed with non-specific pleuritis and normal pleura was diagnosed on a pleural biopsy in 2 (4.17%) patients. Overall, a definitive diagnosis of either pleural malignancy or TB was obtained in 32 (66.7%) patients. Combined overall sensitivity, specificity, positive predictive value and negative predictive value of thoracoscopic pleural biopsy for malignant pleural effusion were 96.77%, 100%, 100% and 66.67%, respectively. There was no procedure-related mortality. Based on the study findings, authors have concluded that, semi-rigid thoracoscopy is a safe and efficacious procedure in patients with undiagnosed exudative pleural effusions.

Rajawat GS et al[38]., have carried out a cross-sectional study to analyze the diagnostic yield and safety of closed needle pleural biopsy in exudative pleural effusion and assessment of patients' characteristics with the yield of pleural biopsy. The main outcome measure was diagnostic yield in the form of confirming diagnosis. Out of the 191 patients with exudative lymphocytic pleural effusion, 123 (64.40%)

were diagnosed on the first pleural biopsy. Among the remaining 68 patients, 22 patients had repeat pleural biopsy with a diagnostic yield of 59.9%. The overall pleural biopsy could establish the diagnosis in 136 (71.20%) patients with pleural effusion. The most common diagnosis on pleural biopsy was malignancy followed by tuberculosis. Authors have concluded that, closed pleural biopsy provides diagnostic yield nearly comparative to thoracoscopy in properly selected patients of pleural effusions

Other biochemical investigations

Matthai SM and Kini U[39]., have examined 26 eosinophilic pleural effusions from among 444 consecutive pleural effusions to assessl the diagnostic and prognostic significance of these eosinophilic effusions and assess their clinical implications. Koss and Light's criteria were applied in the analysis, Out of the 26 EPFs studied, five were associated with tuberculosis and three with metastatic disease. Nineteen patients had significant associated lymphocytosis. Twenty-four patients have been followed up and are in good health to date and have had no recurrence of effusion. Through this, authors have concluded that, EPF could be associated with inflammatory, benign, and malignant conditions.

Sehgal IS et al[40]., performed a systematic review investigating the role of Xpert MTB/RIF in the diagnosis of tuberculous pleural effusion (TPE) was conducted. The pooled sensitivities and specificities of Xpert MTB/RIF were 51.4% and 98.6%, respectively, with culture used as a reference standard and 22.7% and 99.8%, respectively, with a composite reference standard (CRS) used as the benchmark. Xpert MTB/RIF has low sensitivity but excellent specificity in the diagnosis of TPE.

Most relevant studies

Rahul Gupta et al[41]., have carried out a study, to evaluate the new-onset cases of pleural effusion with respect to etiology/causation. A total of 1000 patients were included in the study aged between 18 and 70 years. Out of total 1000 patients, 69.5% had tuberculosis followed by malignancy (16%) with the systemic causes forming about 15% bulk of the patients with pleural effusion. It was found more in males, associated with smoking, and majority of patients had unilateral effusion. Eighty-nine percent of patients had exudative effusion. The results of the study revealed that tuberculosis is still the most common cause of pleural effusion and efforts need to be stepped up to control tuberculosis. Based on the study findings, authors have suggested that, the national programs for control of tuberculosis need to be revisited to assess the magnitude of the problem, and the patients need to be counseled for the compliance of the therapy.

To investigate the etiology of pleural effusions (PE) in adults and the accuracy of pleural fluid (PF) cytology and cultures in malignant and infectious PE, respectively, José M. Porce et al[42]., have carried out a retrospective analysis of all patients with PE undergoing diagnostic thoracentesis. The leading causes of PE among the 3077 patients were found to be cancer (27%), heart failure (21%), pneumonia (19%), tuberculosis (9%), abdominal surgery (4%), pericardial diseases (4%) and cirrhosis (3%). Tuberculosis was the most common etiology in patients <34 years of age (52%), whereas heart failure predominated in octogenarians (45%). The most common primary tumors in malignant PE were lung (37%) and breast (16%) tumors. The overall accuracy of PF cytology was 59%.Viridans streptococci were the most commonly isolated pathogens (25.5%).From the study findings, authors have cconcluded that, three quarters of patients with PE in whom a diagnostic

thoracocentesis was indicated had cancer, heart failure, pneumonia or tuberculosis and on the other hand. PF cytology and cultures give false negative results in a significant number of cases.

Pujan Parikh et al[43]., have carried out a study to arrive at the etiological diagnosis of pleural effusion by analysis of history, clinical presentation, biochemical, radiological, cytological and bacteriological methods. Most common symptom was chest pain (72%) followed by fever (62%). Most of cases were tuberculous (62%) followed by malignant (18%). There were 15 patients with undiagnosed pleural effusion, in which, thoracoscopic pleural biopsy was done, among them 9 patients had malignancy and 5 patients had tuberculous pleural effusion. Most of etiologies for pleural effusion were tuberculosis among young's and malignancy in older age. Right sided pleural effusion was more common in exudative effusion while bilateral pleural effusion was more common in patients with transudative. From the results, authors have come to a conclusion that thoracocentesis followed by pleural fluid analysis is the best method to diagnose the underlying etiology.

To evaluate the common causes of pleural effusion in children. Saleh AB Memon and, Shajeel J Shaikh[44], have carried out a retrospective study on 50 patients. Results have showed that, the boys were 30 (60%) and 20 (40%) were girls. Age range was three years to 14 years. The common symptoms were fever and cough. X-Ray chest showed large pleural effusion. Forty (80%) patients were anemic with less than 8gm Hb level. In 39 patients the ESR was less than 40mm in the first hour. The pleural fluid specimen was taken for culture and sensitivity. Sputum was sent for acid fast bacilli. In two patients, the puss was thick; these patients underwent thoracotomy and decortications. From the results, authors have concluded that, tuberculous pleural

effusion was the most commonly encountered. It was found in 35 (66%) cases. The second most common cause was paraneumonic pleural effusion.

METHODOLOGY

Study site: This study was conducted in the Department of General Medicine at KLES Dr. Prabhakar Kore Hospital, Belagavi

Study population: All the patients admitted in the wards Of General Medicine at KLES Dr. Prabhakar Kore Hospital, Belagavi with Pleural Effusion were considered as study population.

Study design: The current study was a longitudinal study

Sample size: 100

Sampling method: All the eligible subjects were recruited into the study consecutively by convenient sampling till the sample size is reached.

Study duration: The data collection for the study was done between January 2017 to December 2017 for a period of 1 year.

Inclusion Criteria:

- Patients above 18 years of age with symptoms and signs of pleural effusion
- Chest X- ray showing pleural effusion

Exclusion criteria:

- Patients below 18 years
- Known case of pleural effusion previously tapped for etiological basis
- Acute respiratory distress syndrome
- Minimal pleural effusion

Ethical considerations: Study was approved by institutional human ethics committee. Informed written consent was obtained from all the study participants and only those participants willing to sign the informed consent were included in the study. The risks and benefits involved in the study and voluntary nature of participation were explained to the participants before obtaining consent. Confidentiality of the study participants was maintained.

Data collection tools: All the relevant parameters were documented in a structured study proforma.

Methodology:

Inpatients with pleural effusion fulfilling the Inclusion & Exclusion criteria were taken in to study after obtaining written informed consent in their own vernacular language. Demographic data, History, Clinical examination and details of investigations (routine blood investigations, sputum examination, chest x ray pa view and pleural fluid analysis. The pleural fluid was analyzed for cell count, cell type, protein and sugar content, ADA. The pleural fluid PH, LDH, Ultrasound thorax was done as per case basis.

After these Pleural effusions were classified into exudative and transudative according to

- **LIGHT'S CRITERIA**

Fluid is exudate if one of the following Light's criteria is present

- Effusion protein/serum protein ratio greater than 0.5
- Effusion lactate dehydrogenase (LDH)/serum LDH ratio greater than 0.6
- Effusion LDH level greater than two-thirds the upper limit of the laboratory's reference range of serum LDH

INVESTIGATIONS:

- CBC
- SPUTUM EXAMINATION
- CHEST XRAY PA VIEW
- PLEURAL FLUID ANALYSIS-CELL COUNT CELL TYPE ,PROTEIN ,SUGAR,ADA LDH
- SERUM LDH
- SERUM PROTEIN

STATISTICAL METHODS:

Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency and proportion for categorical variables. Data was also represented using appropriate diagrams like bar diagram, pie diagram and box plots.

The association between age groups, LDH level and different categories of ADA was assessed by cross tabulation and comparison of percentages. Chis square test was sued to assess the statistical significance of the association. P value < 0.05was considered statistically significant. IBM SPSS statistical software version 21 was used for data analysis.

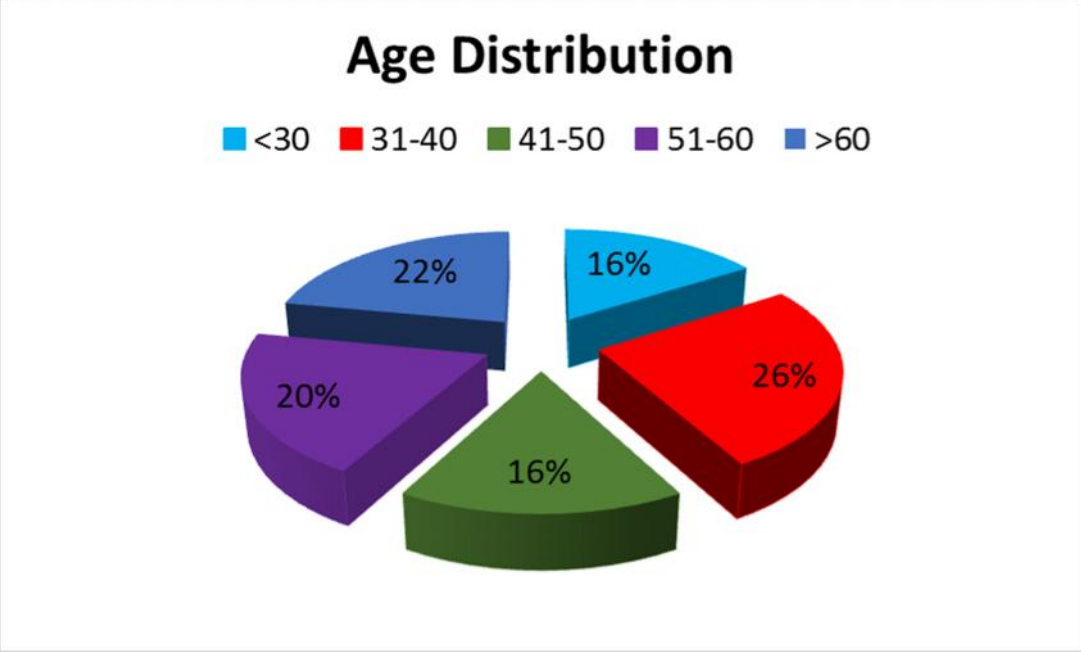
RESULTS

The Present one year longitudinal study titled "Clinical and Etiological Study of Pleural Effusion" was carried out in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. During the study period from January 2017 to December 2017, a total of 100 patients of Pleural Effusion were studied. The findings/observations and final results are tabulated as below.

Table 1. Age Distribution

Age Group (Years)	Distribution (n=100) Number	Distribution Percentage
<30	16	16
31 to 40	26	26
41 to 50	16	16
51 to 60	20	20
>60	22	22
Total	100	100.00

Graph 1.

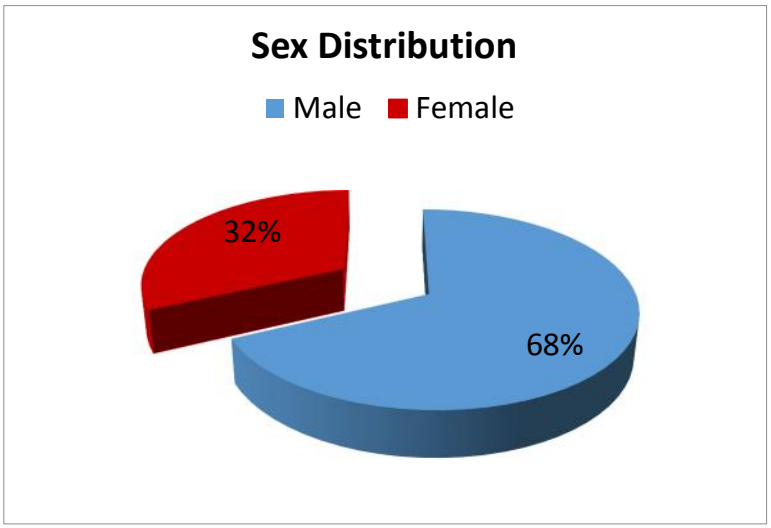


Patients age ranged from 18 to 84 years, maximum number of cases were in the age group of 31 to 40 years i.e. 26 patients (26%), 22 patients in >60 years, between 51 to 60 years 20 patients(20%), between 41 to 50 years 16 patients(16%) and 16 patients (16%) in <30 years.

Table 2. Sex Distribution

Sex	Distribution(n=100) Number	Distribution Percentage
Male	68	68%
Female	32	32%
Total	100	100.00

Graph 2.



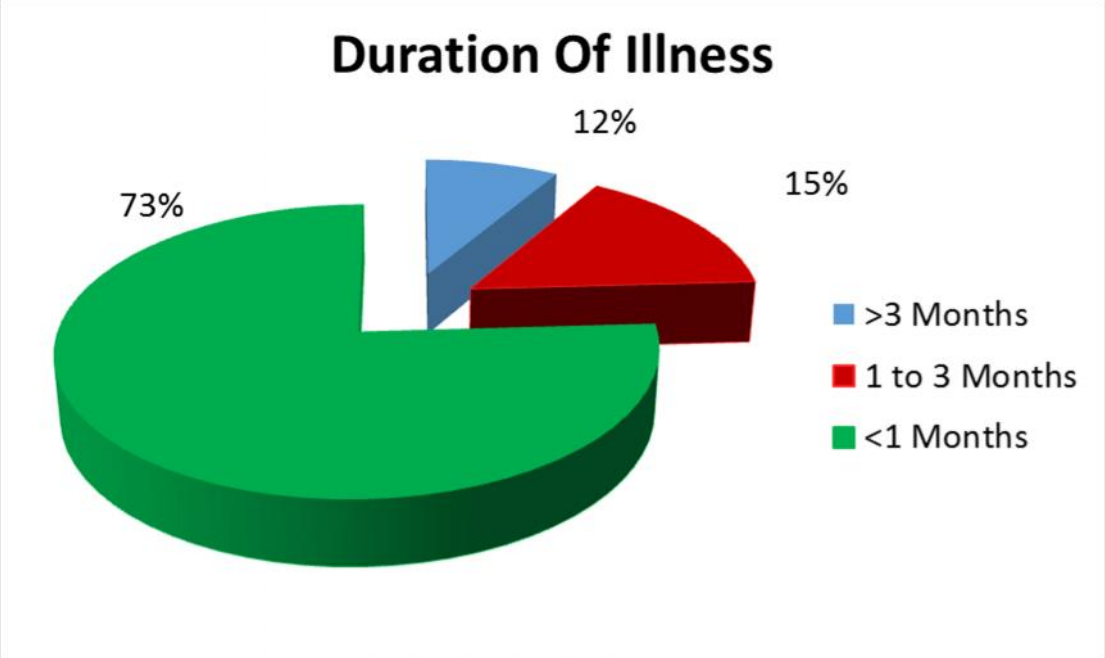
Out of 100 patients 68(68%) were males and 32 patients (32%) were females, accounting a ratio of M:F:: 2.125:1

Inference: Male Preponderance was observed

Table 3. Duration of Illness

Duration(months)	Distribution(=100) Number	Distribution Percentage
>3	12	12
1 to 3	15	15
<1	73	73
Total	100	100.00

Graph 3.

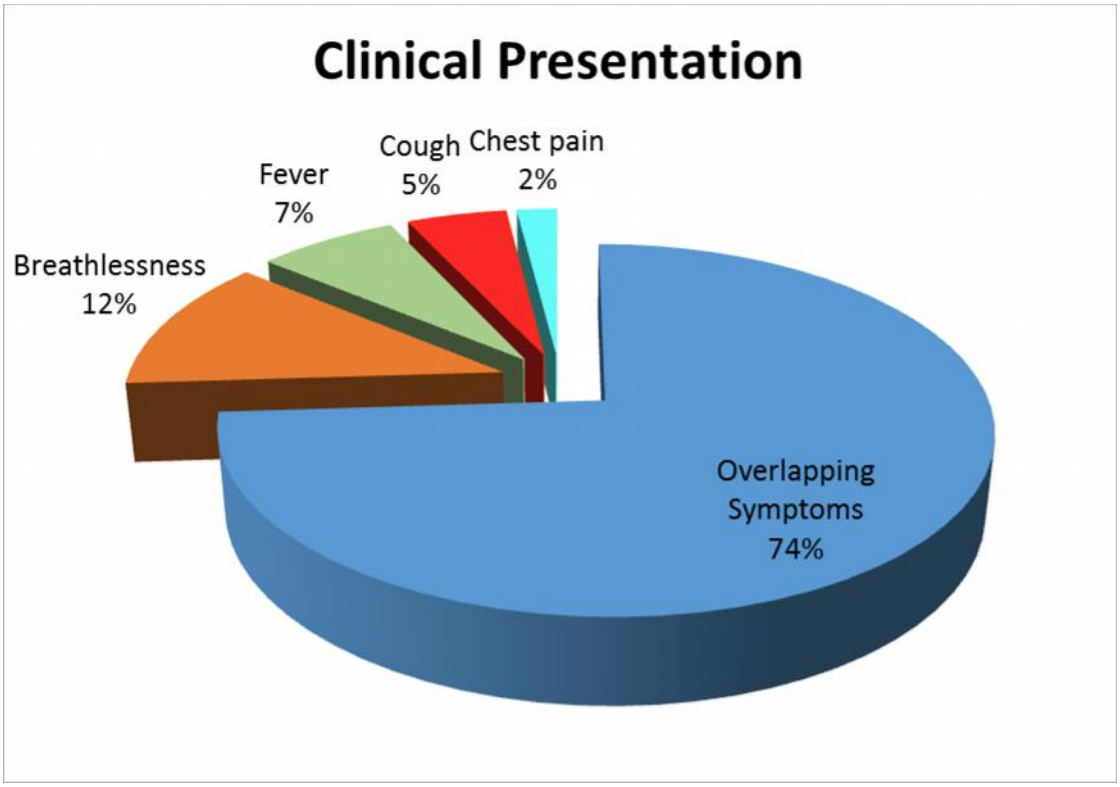


In the present study we observed the duration of illness varied from less than 1month to being more than 3 months. In 73 patients (73%) duration of illness was <1 month, in 15 patients (15%) was between 1 to 3 months and in 12 patients (12%) was >3 months.

Table 4. Clinical Presentation

Complaints	Distribution(n=100) Number	Distribution Percentage
Overlapping Symptoms	74	74%
Breathlessness	12	12%
Fever	7	7%
Cough	5	5%
Chest Pain	2	2%
Total	100	100.00

Graph 4.

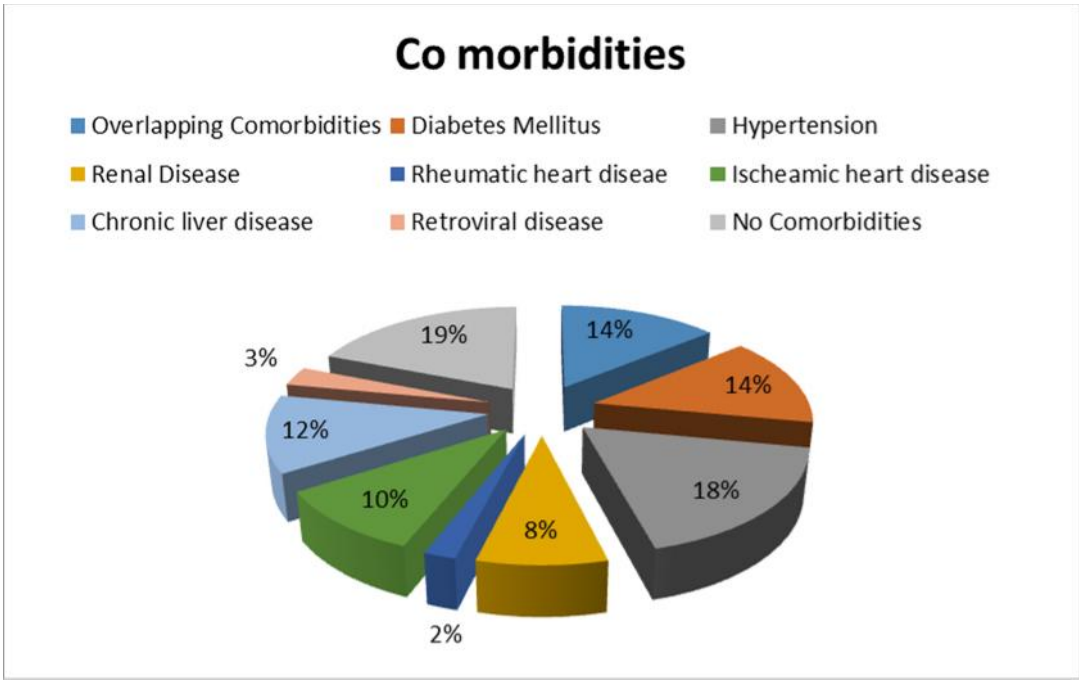


In our study majority of the patients ie.74(74%) presented with overlapping symptoms (Like breathlessness, chest pain, cough and fever), 12 patients (12%) presented with breathlessness followed by 7 patients (7%) with Fever, 5 patients (5%) with cough and 2 patients(2%) with Pleuritic chest pain.

Table 5. Co-morbid Conditions

Comorbidities	Distribution(n=100) Number	Distribution Percentage
Overlapping Co morbidities	14	14
Chronic liver disease	12	12
Retroviral disease	3	3
Renal Disease	8	8
Rheumatic Heart Disease	2	2
Ischemic Heart Disease	10	10
Diabetes Mellitus	14	14
Hypertension	18	18
No co morbidities	19	19
Total	100	100.00

Graph 4.

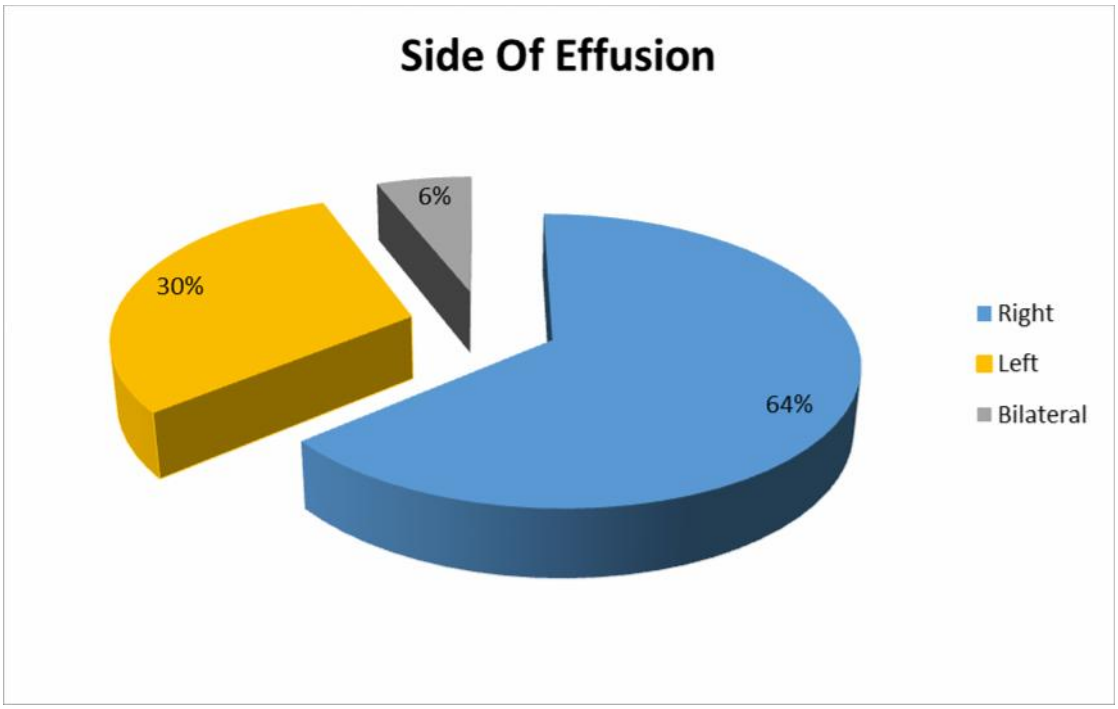


In our study, 14 patients (14%) had overlapping Co-morbidities(DM,HTN,IHD, Chronic Liver Disease and Chronic Kidney Disease),other Co morbidities are depicted in the above table.

Table 6. Side of Effusion

Side	Distribution(n=100) Number	Distribution Percentage (%)
Right	64	64
Left	30	30
Bilateral	6	6

Graph 6.

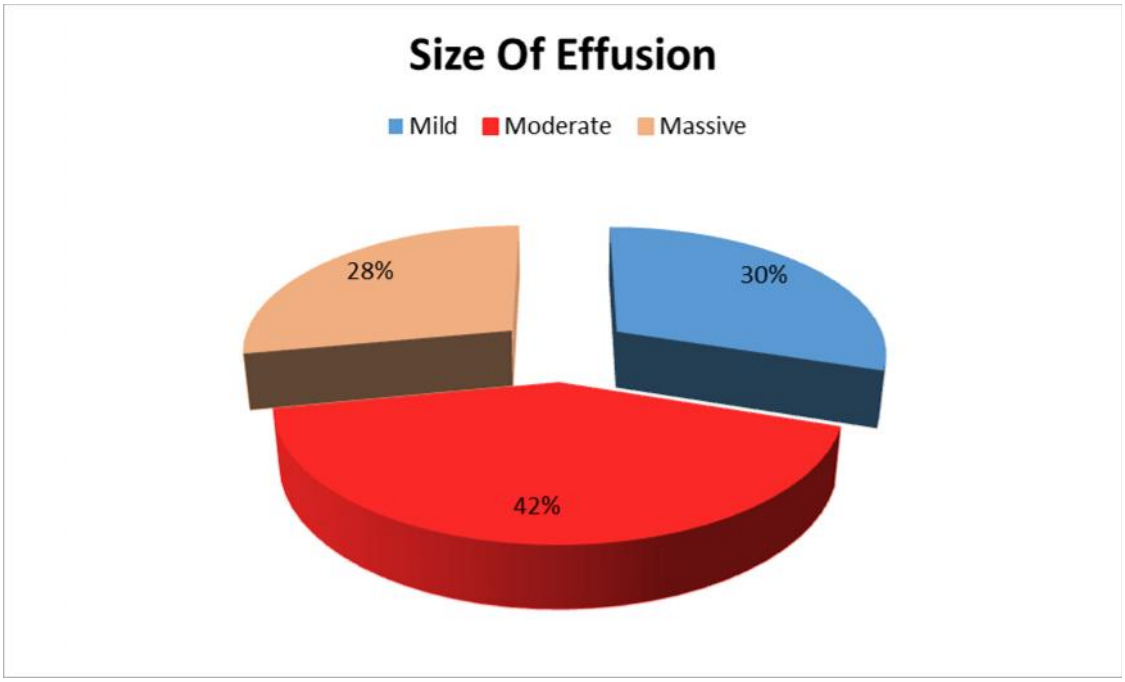


In our Present study, we observed 64 patients (64%) had Right Sided Pleural Effusion, 30 patients (30%) had Left Sided Pleural Effusion and only 6 patients (6%) had Bilateral Pleural Effusion.

Table 7. Size of Effusion

Size(Chest X-ray)	Distribution(n=100) Number	Distribution Percentage (%)
Mild	30	30
Moderate	42	42
Massive	28	28
Total	100	100.00

Graph 7.



In our Present Study of Pleural Effusion 42 patients (42%) had moderate Pleural Effusion ,30 patients(30%) had mild Pleural Effusion and 28 patients (28%) had massive Pleural Effusion. Division of mild, moderate and massive effusion was made on Chest X ray.

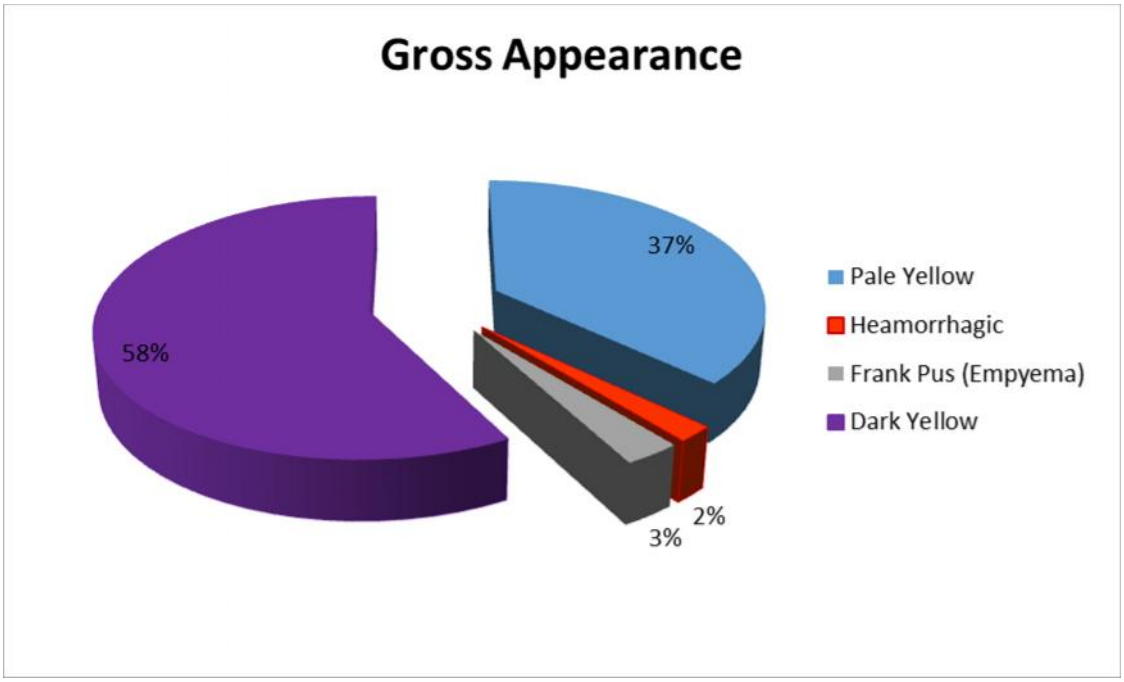
Lab Parameters

Pleural Fluid Analysis

Table 8. Gross Appearance

Appearance	Distribution(n=100) Number	Percentage (%)
Dark Yellow	58	58
Pale Yellow	37	37
Haemorrhagic	2	2
Frank Pus(Empyema)	3	3
Total	100	100.00

Graph 8.

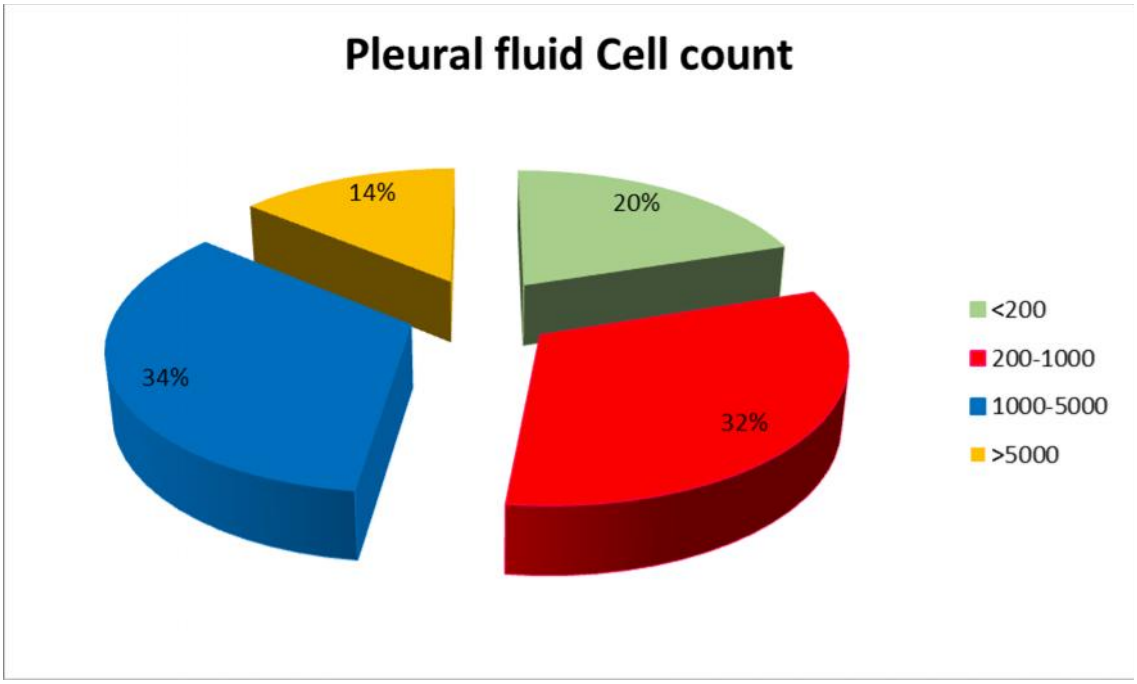


In our present study of 100 patients, majority (95%) had gross appearance of yellow colour (dark yellow 58% and pale yellow 37%), remaining 3 had frank pus suggestive of Empyema and 2 had haemorrhagic appearance (both were mesotheliomas).

Table 9. Pleural fluid Total Cell Count

Total Cell Count	Distribution(n=100) Number	Distribution Percentage (%)
<200	20	20
200-1000	32	32
1000-5000	34	34
>5000	14	14
Total	100	100.00

Graph 9.

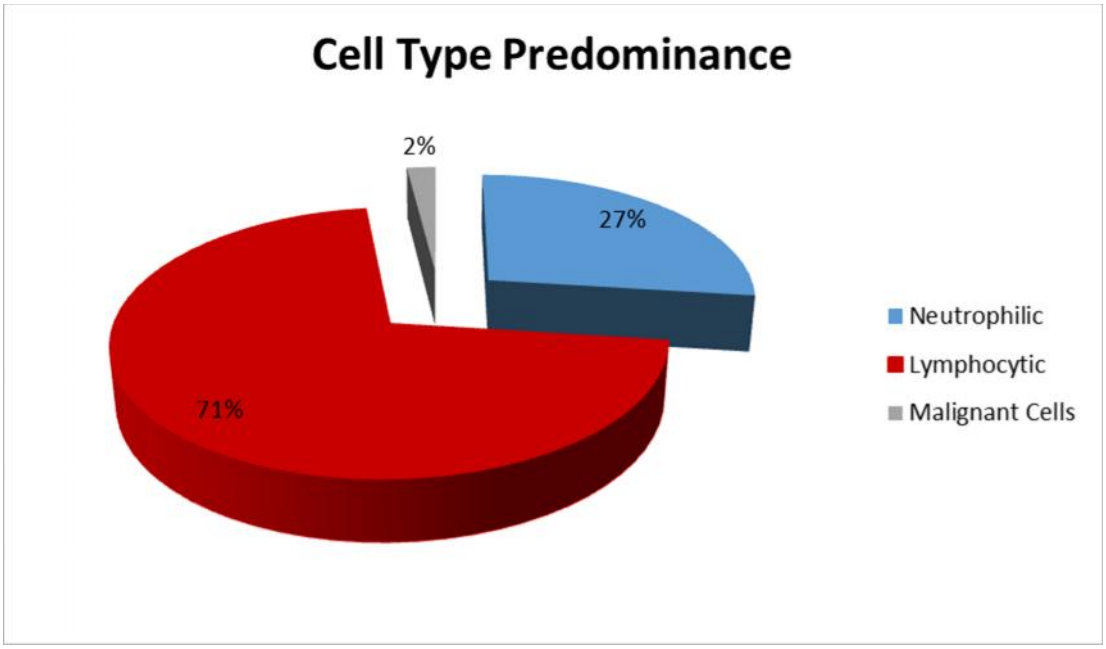


In our study we observed the cell count ranged from <200 to >5000. 20 patients (20%) had <200 cell count, 32 patients (32%) were in the range of >200 to 1000, 34 patients (34%) were in the range of 1000 to 5000 and 14 patients (14%) were having >5000 cell count.

Table 10.Pleural Fluid Cell Type

Cell Type Predominance	Distribution (n=100) Number	Distribution Percentage (%)
Neutrophilic	27	27
Lymphocytic	71	71
Malignant Cells	2	2
Total	100	100.00

Graph 10.

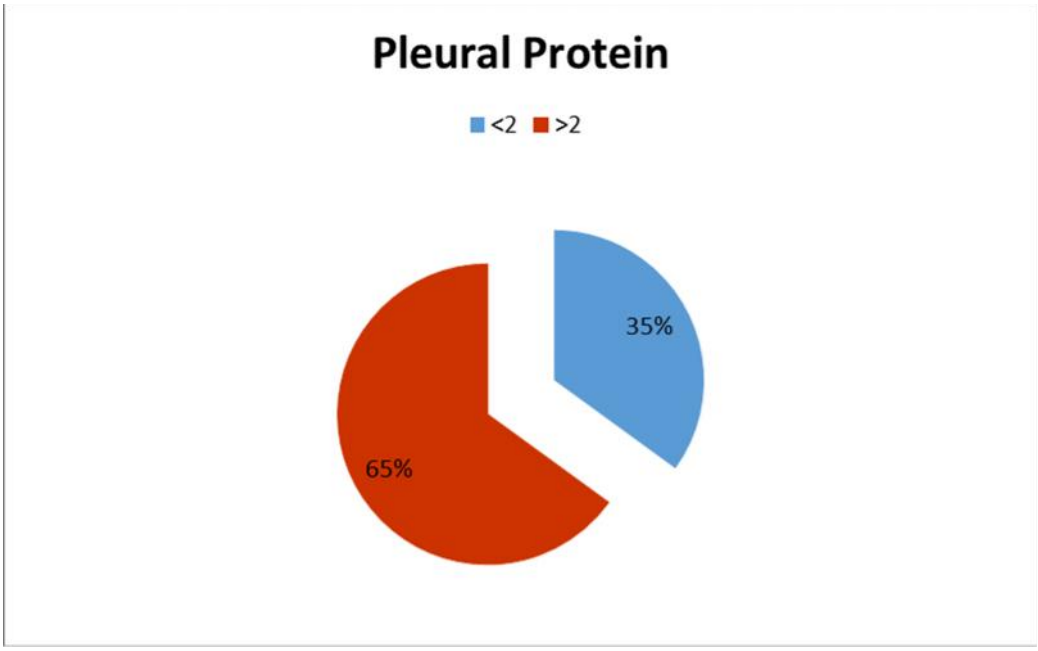


In our present study of 100 patients majority of patients i.e. 71 patients (71%) had lymphocytic predominance, 27 patients (27%) had neutrophilic predominance and 2 patients (2%) had malignant cells (both were mesotheliomas).

Table 11.Pleural Fluid Protein

Pleural Protein (g/dl)	Distribution (n=100) Number	Distribution Percentage (%)
<2	35	35
>2	65	65
Total	100	100.00

Graph 11.



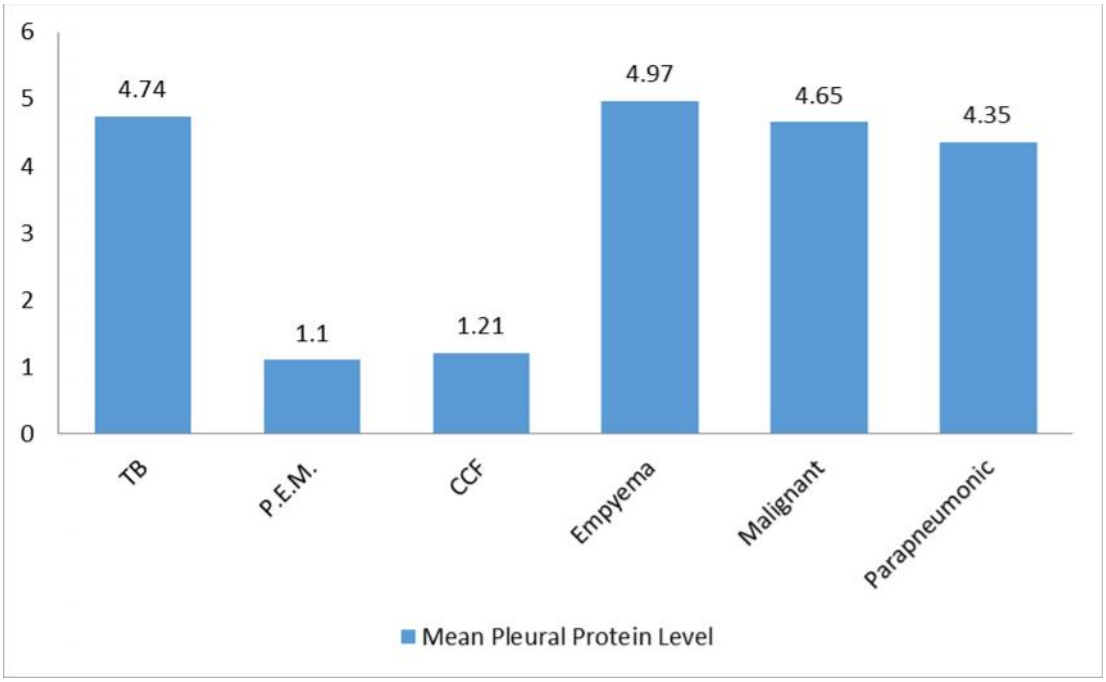
In our present study of 100 patients, 65 patients (65%) had >2g/dl pleural protein and 35 patients (35%) had <2g/dl.

The Mean Pleural Fluid Protein was 3.337g/dl.

Table 12. Comparison of Mean Pleural Protein with Etiology

Etiology	Mean Protein Level
TB	4.74
P.E.M.	1.1
CCF	1.21
Empyema	4.97
Malignant	4.65
Parapneumonic	4.35

Graph 12.

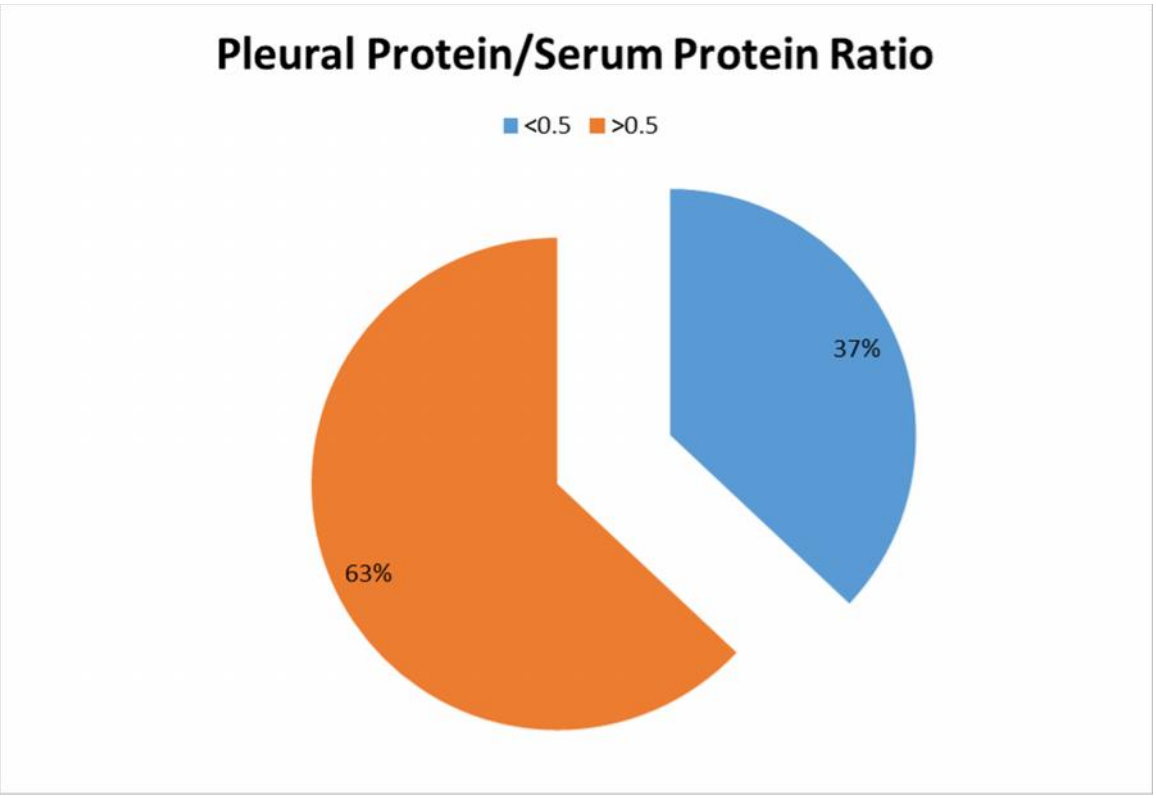


In our study of 100 patients Mean Pleural Protein with different etiologies is depicted in the above table.

Table 13.Pleural Protein to Serum Protein Ratio

Pleural Protein/Serum Protein Ratio	Distribution (n=100) Number	Distribution Percentage (%)
<0.5	37	37
>0.5	63	63
Total	100	100.00

Graph 13.

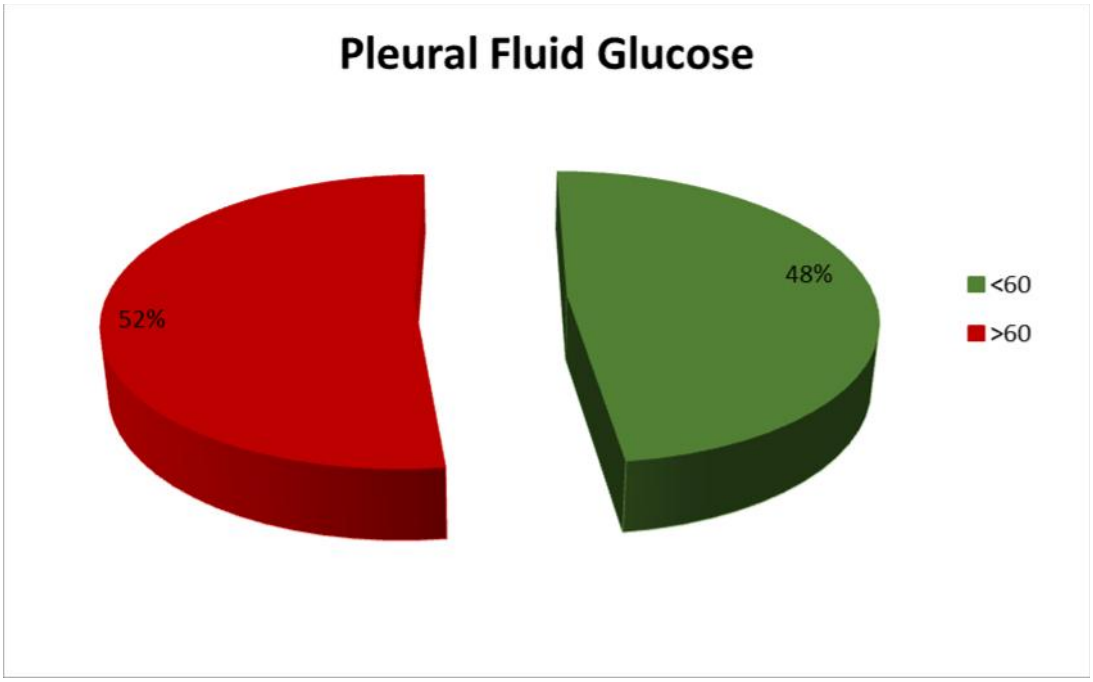


Comparison of Pleural Protein to Serum Protein was done to distinguish whether fluid is transudate or exudate by Light's criteria, revealed 63 patients (63%) had >0.5and 37 patients (37%) had <0.5

Table 14. Pleural Fluid Glucose

Pleural Fluid Glucose (mg/dl)	Distribution (n=100) Number	Distribution Percentage (%)
<60	48	48
>60	52	52
Total	100	100.00

Graph 14.

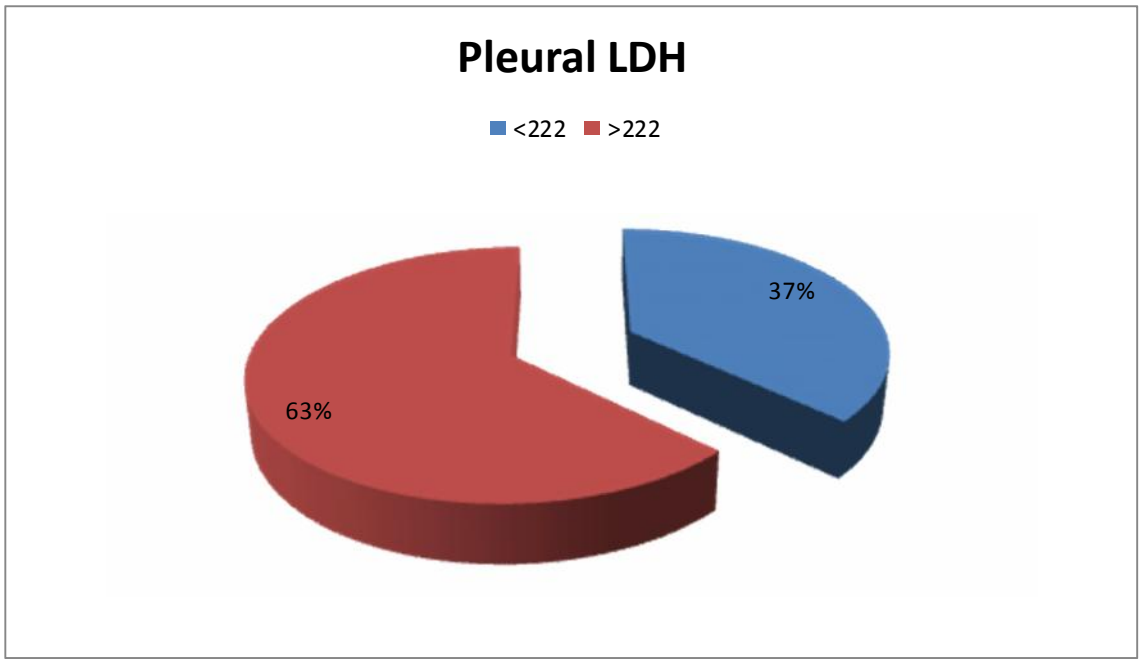


Pleural Fluid Glucose levels revealed 48 patients (48%) had <60 g/dl and 52 patients (52%) had >60 g/dl.

Table 15. Pleural Fluid LDH

Pleural LDH	Distribution(n=100) Number	Distribution Percentage (%)
<222	37	37
>222	63	63
Total	100	100.00

Graph 15.



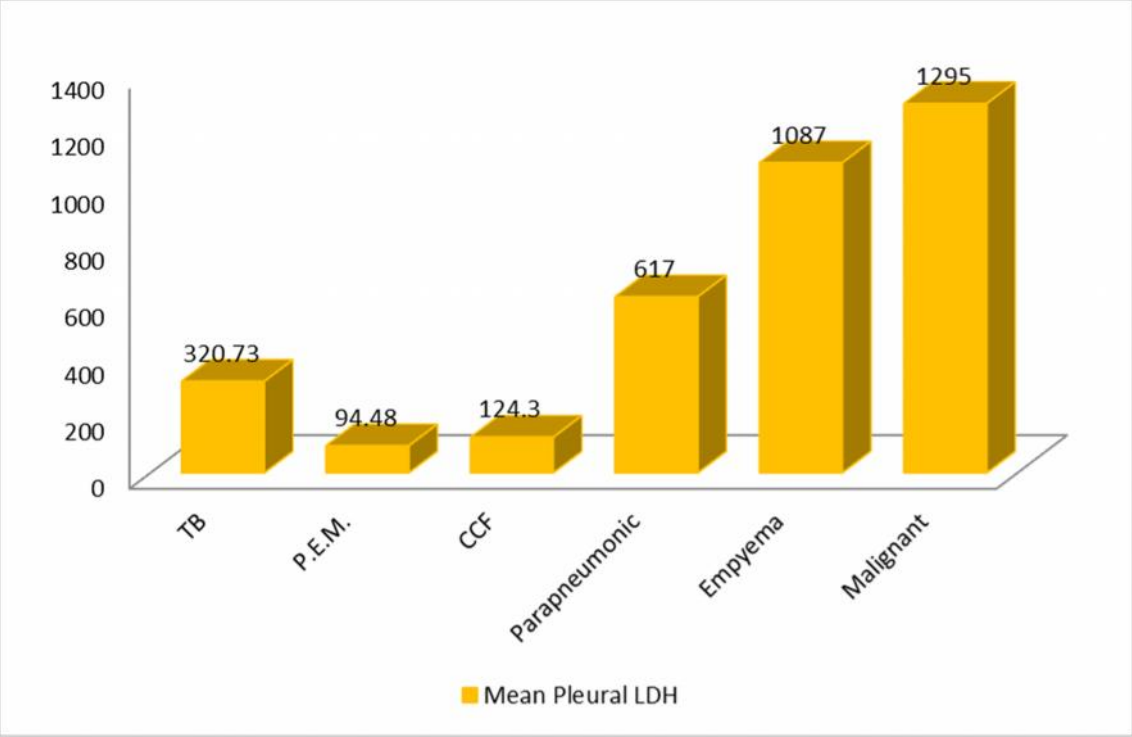
In our study of 100 patients we observed 63 patients (63%) had >222 Pleural LDH and 37 patients (37%) had <222 Pleural LDH.

The Mean Pleural LDH was 309.75

Table 16. Comparison of Mean Pleural LDH with Etiology

Etiology	Mean Pleural LDH
TB	320.73
P.E.M.	94.48
CCF	124.30
Parapneumonic	617.00
Empyema	1087.00
Malignant	1295.00

Graph16.

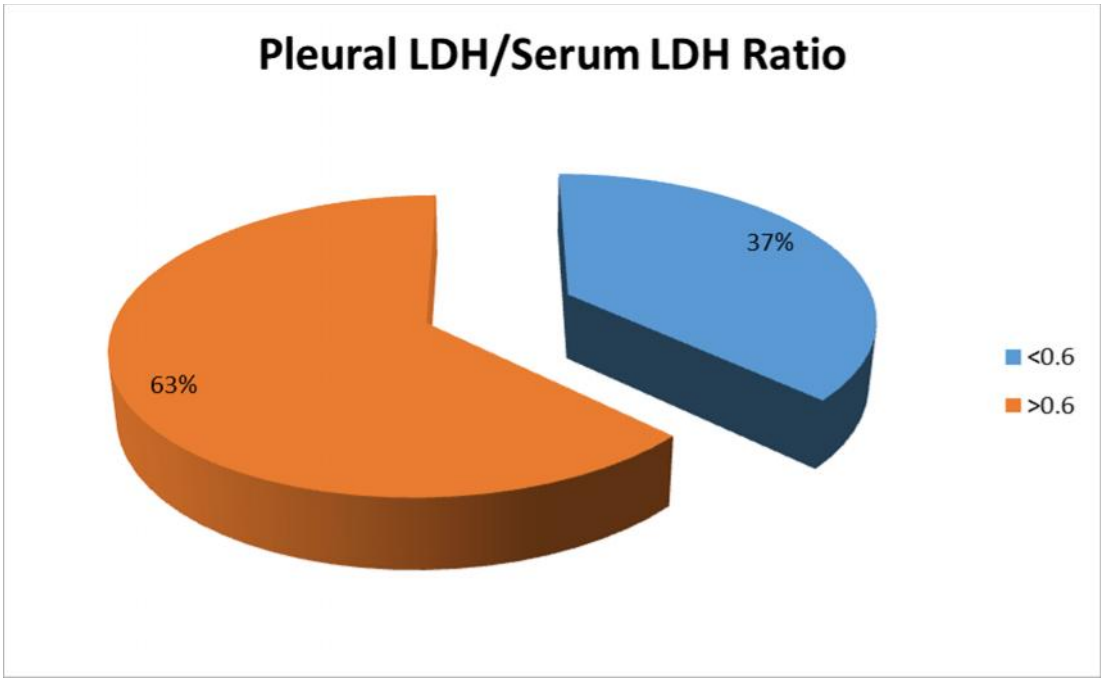


Mean Pleural LDH with different etiologies is depicted in the above table.

Table 17. Pleural LDH/ Serum LDH

Pleural fluid LDH/serum LDH Ratio	Distribution (n=100) Number	Distribution Percentage
<0.6	37	37
>0.6	63	63
Total	100	100.00

Graph 17.

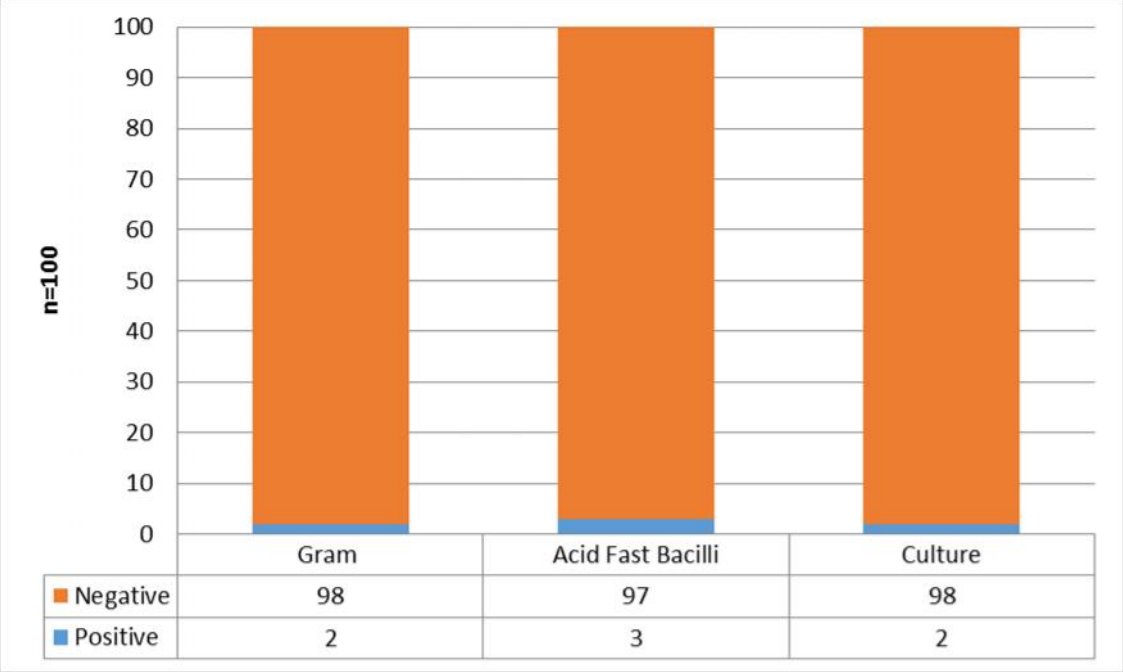


Ratio of Pleural LDH to Serum LDH was done , which revealed 63 patients(63%) with >0.6 and 37 patients (37%) with <0.6.

Table 18. Pleural Fluid Gram Stain, Culture and AFB

	Positive	Negative	Total
Gram Stain	2	98	100
Culture	2	98	100
AFB	3	97	100

Graph 18.

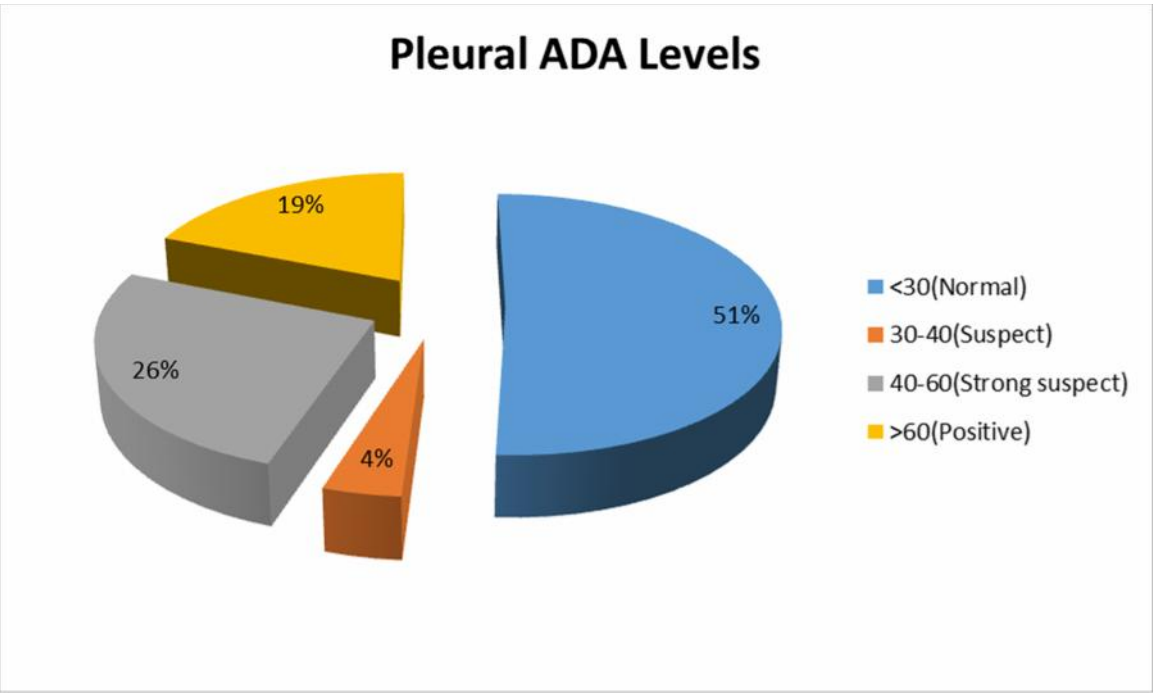


Pleural fluid gram stain, culture and Acid Fast Bacilli staining revealed, 2 patients (2%) were positive for gram staining (gram positive cocci), 2 patients (2%) were culture positive revealing *acinetobacter* and *klebsiella* and in only 3 patients (3%) Acid Fast Bacilli was positive.

Table 19. Pleural Fluid Adenosine Deaminase (ADA)

Pleural ADA (U/L) Level	Distribution (n=100) Number	Distribution Percentage
<30	51	51
30-40	4	4
40-60	26	26
>60	19	19
Total	100	100.00

Graph 19.

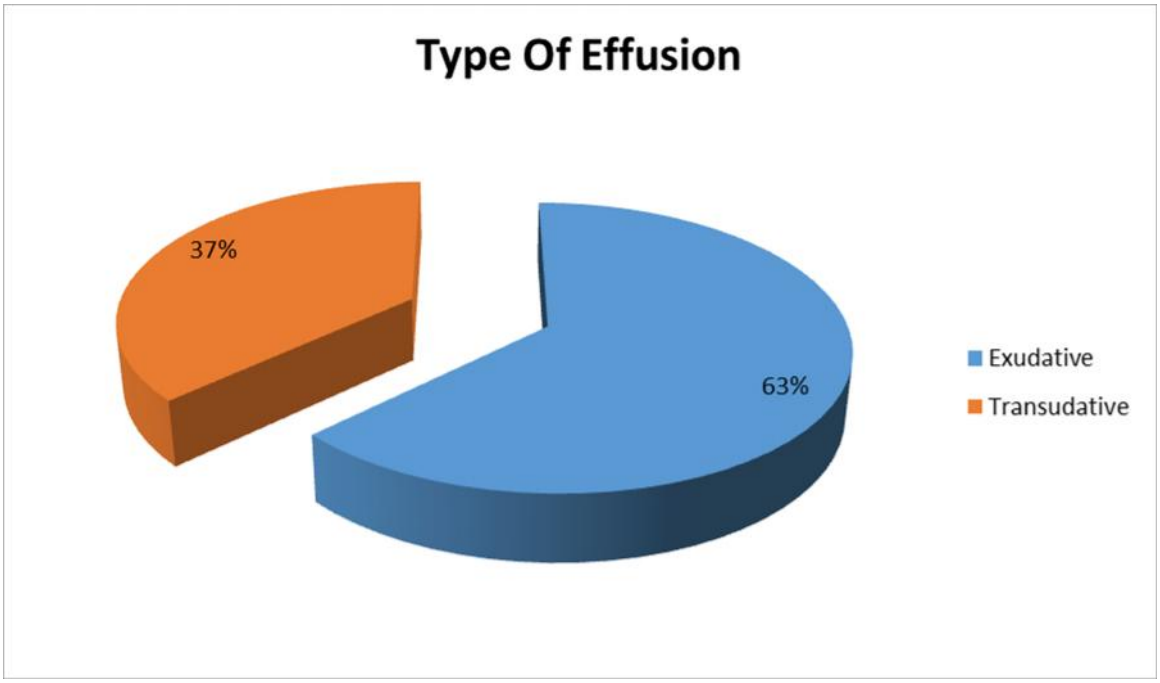


Pleural Fluid ADA estimation to suspect TB vs Non TB was done. Which revealed 51 patients (51%) had normal ranges of ADA, 4 patients (4%) had suspicion , 26 patients (26%) had strong suspicion and 19 patients (19%) were suggestive of Tuberculosis, as depicted in the above table.

Table 20. Type of Effusion

Type of Effusion	Distribution Number (n=100)	Distribution Percentage (%)
Exudative	63	63
Transudative	37	37
Total	100	100.00

Graph 20.

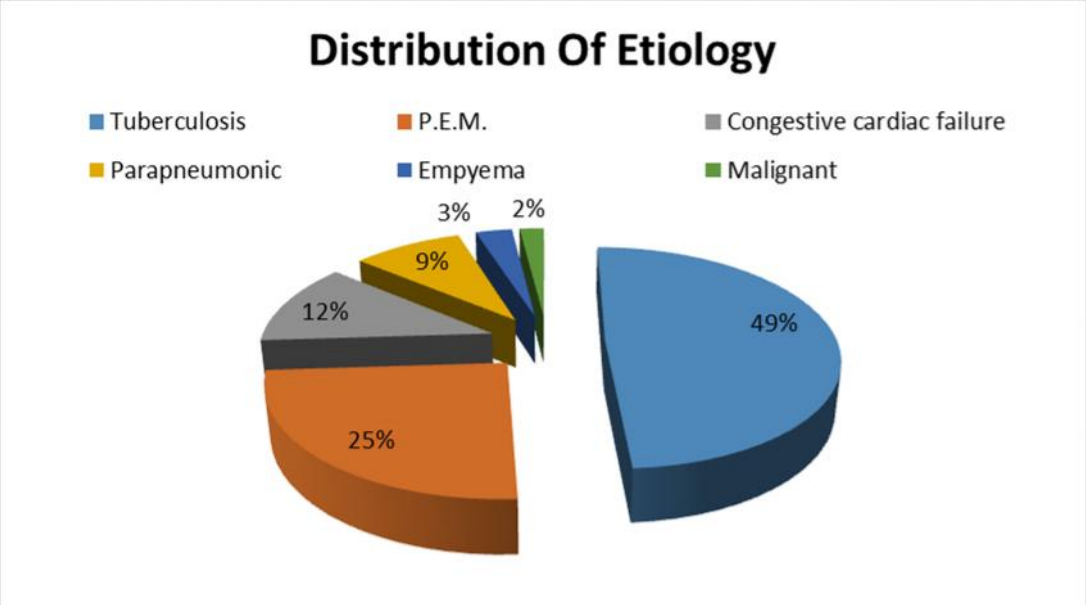


In our present study of 100 patients, 37 patients (37%) had Transudative Pleural Effusion and 63 patients (63%) had Exudative Pleural Effusion.

Table 21. Etiology of Pleural Effusion

Etiology	Distribution number (n=100)	Distribution Percentage (%)
Tubercular	49	49
P.E.M.	25	25
Congestive Cardiac Failure	12	12
Parapneumonic	9	9
Empyema	3	3
Malignant	2	2
Total	100	100.00

Graph 21.



In our present study of 100 patients of Pleural Effusion, 63 were exudative effusions of which revealed 49 patients (49%) were Tubercular Effusion, 9 patients (9%) were Para pneumonic Effusion, 3 patients (3%) were Empyema and only 2 patients (2%) with malignant pleural effusion.

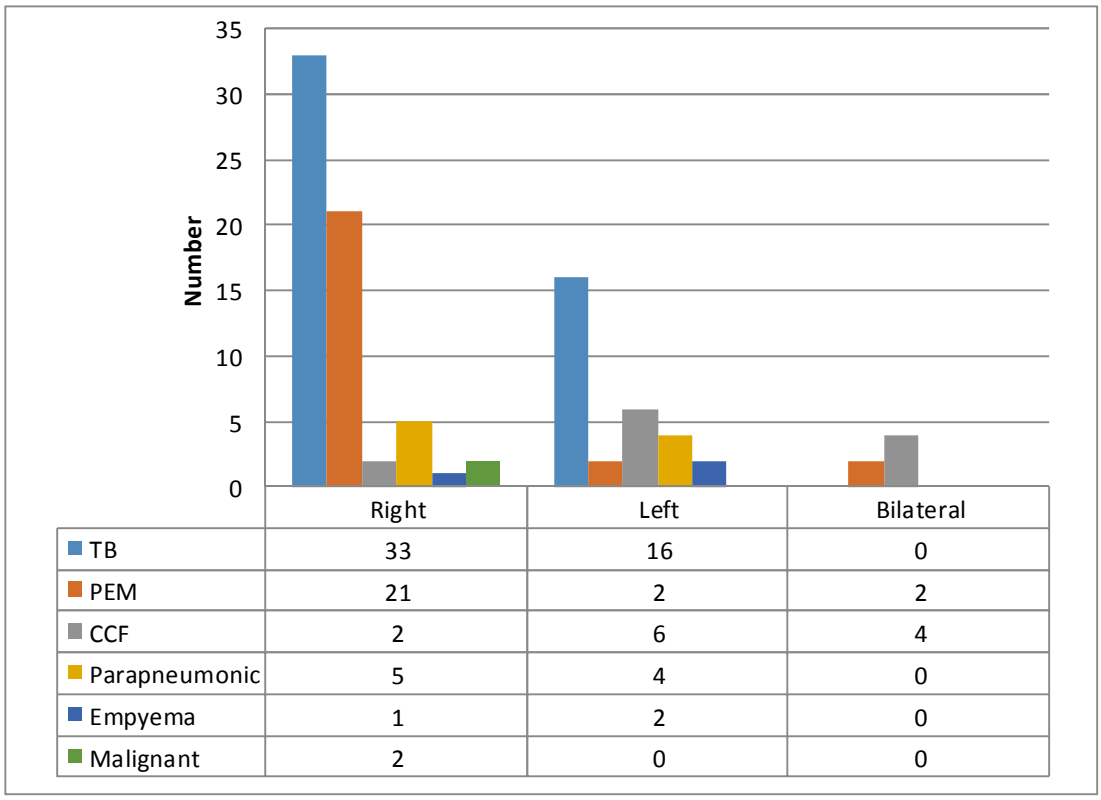
Remaining 37 patients had Transudative Effusion, causes are depicted in the above table.

Table 22: Comparison of Side of Pleural Effusion with Etiology

Etiology	Number (n=100)	Right Sided (n=64)	Left Sided (n=30)	Bilateral (n=6)	P value
TB	(n=49)	33 (51.56%)	16 (53.33%)	0 (0%)	0.002
P.E.M.	(n=25)	21 (32.81%)	2 (6.67%)	2 (33.33%)	
CCF	(n=12)	2 (3.12%)	6 (20%)	4 (66.67%)	
Parapneumonic	(n=9)	5 (7.81%)	4 (13.33%)	0 (0%)	
Empyema	(n=3)	1 (1.56%)	2 (6.67%)	0 (0%)	
Malignant	(n=2)	2 (3.12%)	0 (0%)	0 (0%)	
Total		64	30	6	

P value = 0.002

Graph 22.



In our study of 100 patients, we observed the correlation of side of pleural effusion with etiology as depicted in the above table.

Table 23: Comparison of pleural ADA with age group (N=100)

Age (in years)	Pleural ADA				Chi square	P-value
	<30 ADA (N=51)	31-40 ADA (N=4)	41-60 ADA (N=26)	>60 ADA (N=19)		
<30	9 (17.6%)	0 (0%)	1 (3.8%)	7 (36.8%)	30.54	0.002
31-40	18 (35.3%)	1 (25%)	4 (15.4%)	2 (10.5%)		
41-50	6 (11.8%)	0 (0%)	4 (15.4%)	6 (31.6%)		
51-60	11 (21.6%)	0 (0%)	6 (23.1%)	2 (10.5%)		
>60	7 (13.7%)	3 (75%)	11 (42.3%)	2 (10.5%)		

P value = 0.002

Above table depicts the influence of age on Pleural ADA.

Table 24: Comparison of pleural ADA with pleural LDH (N=100)

Pleural LDH	Pleural ADA				Chi square	P-value
	<30 ADA	31-40 ADA	41-60 ADA	>60 ADA		
>222 LDH (N=63)	15 (23.8%)	3 (4.8%)	26 (41.3%)	19 (30.2%)	51.35	<0.001
<222 LDH (N=37)	36 (97.3%)	1 (2.7%)	0 (0%)	0 (0%)		

P value = <0.001

The above table depicts the correlation between Pleural ADA and Pleural Fluid LDH.

DISCUSSION

In the present study of 100 patients with pleural effusion clinical and etiological factors were analyzed and an attempt was made to diagnose the cause of pleural effusion.

All 100 patients who presented with pleural effusion their age ranged from 18 to 84 years. Maximum number of cases were in the age group of 31-40 i.e. 26 patients (26%). Similar observations were made by Parikh P et al[43] and Shah H. This is in sharp contrast to study done by Dhital KR et al who found effusion common in 21- 30 years of age in their study group.

In our study there was a male preponderance with a male to female ratio of 2.125 to 1 (68 males,32 females).This observation is similar to study done by Parikh P et al[43] and Manu Mohan K et al[45] This is in sharp contrast to a study done by Dhital KR et al who found male to female ratio of 1:1.08(48 males, 52 females).

Majority of our patients presented with illness of < 1month duration, i.e. 73 patients (73%),15 patients (15%) between 1 to 3 months and only 12 patients (12%) >3 months. This is in sharp contrast to Manu Mohan K et al[45]

In our study of 100 patients, patients presented with different clinical presentations like chest pain, fever, cough, breathlessness and overlapping symptoms. 74 patients (74%) had overlapping symptoms, fever was seen (7%), breathlessness (12%) cough (5%) remaining were having pleuritic chest pain (2%). Similar observations were made by Dhital KR et al and Parikh P et al.[43]

In our present study, patients had overlapping co-morbiditiesand other co-morbidities depicted in table number 5.This is in sharp contrast to study done by Parikh P et al.[43]

In our study we noticed majority i.e. 64 patients (64%) had Right sided effusion, 30 patients (30%) had Left sided effusion 6 (6%) were Bilateral. Similar observation was made by Dhital KR et al.

In our 100 patients we tried to categorize the size of pleural effusion based on x ray chest finding and we found mild effusion in 30 patients (30%) moderate in 42 patients (42%), massive in 28 patients (28%).This is in sharp contrast to a study done by Berger et al who observed mild to moderate in majority and massive in only 2.

In 95 patients the gross appearance of pleural effusion was yellow (dark yellow 58, pale yellow 37), hemorrhagic in 2 patients (2%), both were malignant (mesotheliomas) and frank pus in 3 patients (3%). Color of effusion in most of the cases is diagnostic. In all our patients of tubercular effusion, color wasdark yellow, pale yellow in patients with transudative effusion and patients with malignancy it was hemorrhagic. This is in sharp contrast to study done by Manu Mohan K et al[45] who found straw colored as well as hemorrhagic in their patients with tubercular effusion and also some patients with straw color with malignancy.

In our study we observed the cell count ranging from <200 to >5000 which is depicted in table number 9.Further attempt was made to see the type of cell. We found majority i.e. 71 patients (71%) had lymphocytic predominance of which 49 were tubercular effusion.27 patients (27%) had neutrophilic predominance of which 9 were parapneumonic, 3 were empyemas. Books mention lymphocytic or neutrophilic predominance may be observed in transudative effusion and also neutrophilic predominance in the early phase of tubercular effusion.

On analyzing Pleural fluid protein, majority of patients i.e. 65 patients (65%) had >2g/dl and remaining had <2g/dl (35 patients35%)). A study done by Dhital KR et al found mean protein of >3g/dl in Tubercular, malignant and parapneumonic

effusions, and <3g/dl in Congestive cardiac failure and renal disease. Our study of 100 patients we observed >4g/dl of mean protein in tubercular, malignant, empyema and parapneumonic effusion, and <2g/dl in Protein Energy Malnutrition and congestive cardiac failure.

We also attempted analyzing by Light's criteria the Pleural fluid protein to serum protein ratio and found to have 63 patients (63%) had ratio >0.5 and remaining 37 patients (37%) had ratio <0.5.Almost all studies done by Valdes et al[46], Ram KN et al and Parikh P et al[43] have found the ratio of Pleural fluid protein to serum protein of > 0.5 in their patients.

Pleural fluid glucose estimation revealed 52 patients (52%) had >60g/dl and 48 patients (48%) had <60g/dl. Similar observations were made by Fa Al- Alusi et al. In our study group all patients with diabetes mellitus the pleural fluid glucose more than 90g/dl and in 1 patient it was >300g/dl. In 1 patient with empyema and Diabetes Mellitus the pleural fluid glucose was <40g/dl.

Attempt to analyze Pleural fluid LDH, 64 patients (64%) had >222 LDH levels and 36 patients (36%) <222 LDH levels. Mean pleural LDH was 309. The mean Pleural fluid LDH was more in parapneumonic, empyema and malignant effusion whereas in tubercular effusion it was more but not grossly elevated as observed in the study by Dhital KR et al. In all of their patients it was >1000 in patients with exudative effusions.

By Light's criteria, pleural fluid LDH to serum LDH ratio revealed 63 patients (63%) had >0.6 and 37 patients (37%) had <0.6. Similar observations were made by Valdes et al[46], Ram KN et al and Parikh P et al.[43]

We observed by doing pleural fluid gram stain, culture and AFB and found to have only 2 patients (2%) had gram stain positive (Gram positive cocci), Culture

positive in 2 patients(Acinetobacter baumanii &Klebsiella) and AFB positive in 3 patients (3%), which is depocted in Table number 18.A study done by Manu Mohan K et al[45] found similar observations in their study group. But none of their study found smear positive for acid fast bacilli and culture positive. probably because of era being of antibiotics and patients might have received antibiotics prior to hospital admission.

In our study pleural fluid ADA revealed in 19 patients (19%) had >60U/l, the remaining levels are depicted in table number 19. All patients having >60U/L (19 patients) were tubercular effusions. Similar observations were made by Dhital KR et al and Jindal S et al.[47]

We tried to categorize patients of effusion as exudative and transudative. 63 patients (63%) had exudative, remaining were transudative. Patients with exudative effusions were tubercular 49, parapneumonic 9, empyema 3, malignant 2 and remaining 37 patients with transudative effusion were 25 Protein Energy Malnutrition, 12 Congestive Cardiac Failure. Similar observations were made by Valdes et al[46] and Parikh P et al[43].

We also tried to find out side of effusion with etiology. Patient with right studied effusion 64 (64%), majority had tubercular (33), followed by PEM (21) and para pneumonic (5). In patients with left sided effusion (30), 16 were tubercular 6 were CCF, 4 were Parapneumonic. Details of other etiologies with side of effusion has been depicted in table number 22.Similar observations were made by Manu Mohan K et al[45] and Dhital KR et al.[53]

We also found positive correlation of ADA with advancing age. Same observations were made by Tunn Ray et al[48] This is explained by immunosenescence leading to decreased ADA levels with advancing age.

We also found a positive correlation between Pleural ADA and Pleural LDH .Similar observation was made by Kashibawara et al[49] and Tunn Ren et al[48]

In our present study of 100 patients with pleural effusion, the most common presentation was with overlapping symptoms (fever, cough, breathlessness and chest pain). In our study we observed the common age group with effusion was between 31 to 40 years. Patients with ADA >60 U/L were in the age group of <30 years (7 patients) and between 41 to 50 years (6 patients). All were Tubercular effusions. In our 100 patients of pleural effusion the side of effusion was on the right side, left side and bilaterally. Majority of patients with right sided effusion were exudative effusions, tuberculosis was the commonest cause followed by parapneumonic effusions. Patients with left sided effusion commonest cause was tuberculosis again. Patients with Bilateral effusion the commonest cause was congestive cardiac failure. And on further analysis of pleural fluid for cytology, revealed patients with lymphocytic predominance, tuberculosis was the commonest cause and in patients with neutrophilic predominance parapneumonic effusion was the commonest cause. 2 patients were malignant effusion (mesothelioma).Simple ADA estimation helped us to categorize patients as Tubercular vs Non tubercular , however with increasing age , levels were low probably by mechanism of immunosenescence with advancing age . Correlating Pleural Fluid ADA with Pleural LDH we found statiscally significant correlation. In patients with Pleural LDH >222 had also Pleural ADA of >60 U/L, and patients with LDH <222 none had Pleural ADA >60U/L.

We feel with simple investigations which are done in most of the hospitals, like pleural fluid, protein, sugar, ADA, LDH and Pleural fluid cytology we can arrive at diagnosis whether it is tuberculosis or non tuberculosis. Light's criteria helps to distinguish whether fluid is transudative or exudative with further analysis of fluid for

cytology gram stain, culture and acid fast bacilli would help to arrive at a final diagnosis. We did not attempt Pleural biopsy in our study population as it's an invasive procedure with antecedent complications. In most of our patients we could arrive at a diagnosis with simple means of Lab parameters carried out for patients which is discussed above. But it may be necessary to subject patients for pleural biopsy in difficult situations like in patients of malignancy with lymphocytic pleocytosis. In our study only 3 were smear positive for Acid fast bacilli (3/100) and 2 were positive for malignancy (mesotheliomas).

CONCLUSION

In our present study of 100 patients with Pleural Effusion the commonest age group was between 31 to 40 years followed by > 60 years. The commonest etiology in the age group 31 to 40 years was Tubercular and Parapneumonic effusions, whereas in patients of >60 years again Tubercular was the commonest etiology and 1 was malignancy (mesothelioma). Males were more in our study group as compared to females, malignancy was observed in 2 patients both were females. In majority of our patients duration of illness was less than 1month. Most of our patients presented with overlapping symptoms. Majority of our patients had co-morbid conditions. All our patients with retroviral disease had tubercular effusions. Patients with Diabetes Mellitus, most of them had Tubercular effusions.

Patients with Heart conditions (Ischemic Heart Disease and Rheumatic Heart Disease) none had tubercular effusion. Patients with Chronic Liver Disease, Heart Conditions and Renal diseases had transudative effusions. Majority of our patients had Right sided effusions. Most of our patients had moderate to massive effusions. Fluid analysis revealed exudative effusions in most of the patients, of which majority were tubercular effusions followed by parapneumonic effuusions. Most of the patients cell count ranged from 200 to 5000. Majority of the patients, lymphocytic predominance was observed followed by neutrophilic predominance.

Lymphocytic predominance was common in Tubercular effusions while neutrophilic was common in parapneumonic effusions and empyema. Most of the patients had protein more than 2g/dl. Increased proteins were observed in Tubercular effusions, Empyema, Parapneumonic effusions and malignancy. Light's criteria revealed majority of patients had ratio of more than 0.5. Gram stain was positive only in 2 of our patients so was culture positive (2 patients) and Acid fast bacilli was

positive in 3 patients. Most of the patients ADA was elevated which helped to categorize our patients as tubercular vs non tubercular. Side of effusion did not have significance to diagnose the cause of effusion, but in patients with Congestive cardiac failure effusion was bilateral.

Owing to a small sample size of 100 patients we feel it is worth to study by adjusting variables like age, sex, duration of illness , side of effusion and co-morbidities, to see whether these factors have any effect on etiologies. In most of our patients by simple means like Pleural fluid analysis for protein, sugar, cytology, LDH and ADA we could arrive at a diagnosis in all our 100 patients. Most of the centers especially peripherals do not have facilities for higher modalities of investigations. These simple measures would help to arrive at a diagnosis. In difficult situations, it may be required to subject patients for further study by Computed tomography, Pleural Biopsy and other modalities of investigations. To address these issues, a larger sample size may be needed.

SUMMARY

In the present study of 100 patients with Pleural Effusion admitted in Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre in the study period from January 2017 to December 2017 was undertaken to find the etiology of Pleural Effusion.

The results observed on analysing the pleural fluid were majority of patients with exudative effusions of which most of the patients were tubercular followed by Parapneumonic effusions.

There was a positive correlation between Pleural LDH and Pleural ADA, and Pleural ADA with advancing age.

However we did not find significant correlations with factors like age, sex, duration of illness, side of effusion and size of effusion.

BIBLIOGRAPHY

1. Krueger JJ, Bain T, Patterson JR JL. Elevation gradient of intrathoracic pressure. Journal of applied physiology. 1961;16(3):465-8.
2. Sahn SA. Pleural effusions of extravascular origin. Clinics in chest medicine. 2006;27(2):285-308.
3. Light RW. Clinical practice. Pleural effusion. N Engl J Med. 2002;346(25):1971-7.
4. Desalu OO, Oluwafemi JA, Ojo O. Respiratory diseases morbidity and mortality among adults attending a tertiary hospital in Nigeria. Jornal Brasileiro de Pneumologia. 2009;35(8):745-52.
5. Maher GG, Berger HW. Massive pleural effusion: malignant and nonmalignant causes in 46 patients. American Review of Respiratory Disease. 1972;105(3):458-60.
6. Light RW. Parapneumonic effusions and empyema. Proceedings of the American Thoracic Society. 2006;3(1):75-80.
7. Light RW. The undiagnosed pleural effusion. Clinics in chest medicine. 2006;27(2):309-19.
8. Hasley PB, Albaum MN, Li Y-H, Fuhrman CR, Britton CA, Marrie TJ, et al. Do pulmonary radiographic findings at presentation predict mortality in patients with community-acquired pneumonia? Archives of internal medicine. 1996;156(19):2206-12.
9. Shaw PH, Agarwal R. Pleurodesis for malignant pleural effusions. Cochrane database of systematic reviews. 2004(1).

10. Shallenberger DW, Daniel TM. Quantitative determination of several pleural fluid proteins. American Review of Respiratory Disease. 1972;106(1):121-2.
11. Sahebjami H, Loudon RG. Pleural effusion: pathophysiology and clinical features. Semin Roentgenol. 1977;12(4):269-75.
12. Huang Y, Huang H, Li Q, Browning RF, Parrish S, Turner Jr JF, et al. Transbronchial lung biopsy and pneumothorax. Journal of thoracic disease. 2014;6(Suppl 4):S443.
13. Huang Y, Huang H, Li Q, Browning RF, Parrish S, Turner Jr JF, et al. Approach of the treatment for pneumothorax. Journal of thoracic disease. 2014;6(Suppl 4):S416.
14. Anie Y, Sumi S, Varghese P, Madhavi LG, Sathish M, Radhakrishnan VV. Diagnostic approaches in patients with tuberculous pleural effusion. Diagn Microbiol Infect Dis. 2007;59(4):389-94.
15. Agrawal A, Tandon R, Singh L, Chawla A. Clinico-pathological profile and course of malignant pleural effusion in a tertiary care teaching hospital in western UP with special reference to lung cancer. Lung India: official organ of Indian Chest Society. 2015;32(4):326.
16. Babu KA, Sundararajan L, Prabu P, Parameswaran A. Disseminated plasma cell myeloma presenting as massive pleural effusion. Eur Clin Respir J. 2015;2.
17. Gadewad N, Deokar K, Ghorpade S. Clinical Profile of Patients Presenting with Malignant Pleural Effusion to a Tertiary Health Care Centre. J Assoc Physicians India. 2017;65(8):28-31.
18. Kulaylat AN, Engbrecht BW, Pinzon-Guzman C, Albaugh VL, Rzucidlo SE, Schubart JR, et al. Pleural effusion following blunt splenic injury in the pediatric trauma population. J Pediatr Surg. 2014;49(9):1378-81.

19. Banka R, Udwadia Z. Imatinib-induced pleural effusion: A case report. J Postgrad Med. 2017;63(1):55-7.
20. Chaturvedi A, Kumar A. Malignant Pleural Effusion with Filariasis. J Assoc Physicians India. 2017;65(5):106-7.
21. Arora VK, Gowrinath K. Pleural effusion due to lymphatic filariasis. Indian J Chest Dis Allied Sci. 1994;36(3):159-61.
22. Gupta S, Sathya B, Padhy N, Kundavi S, Thomas BE, Varma TR. Isolated bilateral pleural effusion as the sole manifestation of late onset ovarian hyperstimulation syndrome. J Hum Reprod Sci. 2009;2(2):83-6.
23. Jaggi S, Kundu R, Binji S, Handa U, Saini V. Germ cell tumor causing pleural effusion: A diagnostic dilemma. Indian J Tuberc. 2018;65(1):80-3.
24. Joshi S, Periwal P, Dogra V, Talwar D. Sarcoidosis as unusual cause of massive pleural effusion. Respir Med Case Rep. 2015;16:143-5.
25. Kaur N, Singh J, Haq S, Garg S, Bhatnagar S. Pleural and Mediastinal Lipomatosis with Subpleural Fat as a Mimicker of Pleural Effusion- A Rare Case Report. J Clin Diagn Res. 2017;11(7):Td03-td4.
26. Kumar J, K CS, Mukhopadhyay K, Ray S. A misplaced peripherally inserted central catheter presenting as contralateral pleural effusion. BMJ Case Rep. 2018;2018.
27. Kumar S, Verma SK, Singh R, Prasad R. Hemorrhagic pleural effusion secondary to sarcoidosis: A brief review. Ann Thorac Med. 2009;4(1):27-31.
28. Mitra S, Sarma MK, Das AK. Curious case of a black pleural effusion: Mediastinal teratoma presenting as massive pleural effusion. Lung India. 2018;35(1):87-9.

29. Chinchkar NJ, Talwar D, Jain SK. A stepwise approach to the etiologic diagnosis of pleural effusion in respiratory intensive care unit and short-term evaluation of treatment. Lung India. 2015;32(2):107-15.
30. Sharma M, Rameshbabu CS. Endoscopic ultrasound-guided evaluation of the pleura and cases of pleural effusion. Lung India. 2017;34(5):441-7.
31. Sharma SK, Banga A. Diagnostic utility of pleural fluid IFN-gamma in tuberculosis pleural effusion. J Interferon Cytokine Res. 2004;24(4):213-7.
32. Wu H, Khosla R, Rohatgi PK, Chauhan SS, Paal E, Chen W. The minimum volume of pleural fluid required to diagnose malignant pleural effusion: A retrospective study. Lung India. 2017;34(1):34-7.
33. Bansal A, Tandon S, Kharb S. Diagnostic value of sialic acid in pleural effusion. Zhongguo Fei Ai Za Zhi. 2010;13(4):349-51.
34. Welagedara S, Swe TM, Sriram KB. Investigating a unilateral pleural effusion: A tale of a medical error and diagnostic delays. Lung India. 2017;34(1):82-4.
35. Biswas B, Sharma SK, Negi RS, Gupta N, Jaswal VM, Niranjan N. Pleural effusion: Role of pleural fluid cytology, adenosine deaminase level, and pleural biopsy in diagnosis. J Cytol. 2016;33(3):159-62.
36. James P, Gupta R, Christopher DJ, Balamugesh T. Evaluation of the diagnostic yield and safety of closed pleural biopsy in the diagnosis of pleural effusion. Indian J Tuberc. 2010;57(1):19-24.
37. Nattusamy L, Madan K, Mohan A, Hadda V, Jain D, Madan NK, et al. Utility of semi-rigid thoracoscopy in undiagnosed exudative pleural effusion. Lung India. 2015;32(2):119-26.

38. Rajawat GS, Batra S, Takhar RP, Rathi L, Bhandari C, Gupta ML. Diagnostic yield and safety of closed needle pleural biopsy in exudative pleural effusion. Avicenna J Med. 2017;7(3):121-4.
39. Matthai SM, Kini U. Diagnostic value of eosinophils in pleural effusion: a prospective study of 26 cases. Diagn Cytopathol. 2003;28(2):96-9.
40. Sehgal IS, Dhooria S, Aggarwal AN, Behera D, Agarwal R. Diagnostic Performance of Xpert MTB/RIF in Tuberculous Pleural Effusion: Systematic Review and Meta-analysis. J Clin Microbiol. 2016;54(4):1133-6.
41. Gupta R, Gupta A, Ilyas M. Spectrum of pleural effusion etiology revisited in 18–70 years of age group: A tertiary care center-based study of 1000 patients. CHRISMED Journal of Health and Research. 2018;5(2):110.
42. Porcel JM, Esquerda A, Vives M, Bielsa S. Etiology of pleural effusions: analysis of more than 3,000 consecutive thoracenteses. Archivos de Bronconeumología (English Edition). 2014;50(5):161-5.
43. Parikh P, Odhwani J, Ganagajalia C. Study of 100 cases of pleural effusion with reference to diagnostic approach. International Journal of Advances in Medicine. 2017;3(2):328-31.
44. Memon SA, Shaikh SJ. The etiology of pleural effusion in children: Hyderabad experience. Pak J Med Sci. 2007;23(1):86-7.
45. Manu MK. Etiology and clinical profile of pleural effusion in a teaching hospital of South India: A descriptive study. Pulmon. 2012;14(3):89-96.
46. Valdes L, Alvarez D, San Jose E, Penela P, Valle JM, Garcia-Pazos JM, et al. Tuberculous pleurisy: a study of 254 patients. Arch Intern Med. 1998;158(18):2017-21.

47. Gilhotra R, Sehgal S, Jindal S. Pleural biopsy and adenosine deaminase enzyme activity in effusions of different aetiologies. Lung India. 1989;7(3):122.

48. Tay TR, Tee A. Factors affecting pleural fluid adenosine deaminase level and the implication on the diagnosis of tuberculous pleural effusion: a retrospective cohort study. BMC infectious diseases. 2013;13(1):546.

49. Kashiwabara K, Okamoto T, Yamane H. When pleural potassium exceeds 5.0 mEq/L, high pleural adenosine deaminase levels do not necessarily indicate tuberculous pleuritis. Respirology. 2012;17(1):92-8.

50. Light RW. Pleural Diseases. 5. Philadelphia: Lippincott Williams & Wilkins; 2007.

51. Light RW, Glickmen RM, Isselbreher K. Disorders of pleura, mediastinum and diaphragm. In: Light RW, Glickmen RM, Isselbreher K, eds. Harrison's Principle of Internal Medicine. 14th ed. New York: McGraw-Hill; 1998: 1473-1476

52. FA AL- ALUSI Pleural Effusion In Iraq : A Prospective Study Of 100 Cases Thorax 1986; 41: 492-493.

53. Dhital KR, Acharya R, Bhandari R, Kharel P, Giri KP, Tamrakar R. Clinical profile of patients with pleural effusion admitted to KMCTH. Kathmandu Univ Med J (KUMJ). 2009 Oct-Dec;7(28):438-44.

INFORMED CONSENT

TITLE OF RESEARCH AND STUDY: "CLINICAL AND ETIOLOGICAL STUDY OF PLEURAL EFFUSION, ONE YEAR LONGITUDINAL STUDY"

Principal Investigator:-

Dr. _____

Post Graduate Student,

Department Of General Medicine,

JNMC, Belgaum.

Guide:-

Dr. _____

Professor & Head of Unit,

Department of General Medicine,

JNMC, Belgaum.

Introduction and Purpose:-This research is intended to study clinically pleural effusion and etiology of pleural effusion. The principal investigator of the study is Dr. _____under the guidance of Dr. _____. This study is intended for early diagnosis of pleural effusion and knowing the etiology for helping in early therapeutic approach.

Procedure:

If you agree to be part of the research study, you will be asked the relevant history and will be subjected to relevant clinical examination and investigations. You will also have to give blood and sputum samples for the necessary investigations.

Risk and Benefits:

The only risk and possible discomfort you might get is while doing thoracocentisis for pleural fluid analysis. It may cause pain, at the site where the tapping is done.

You may/may not be benefitted by these investigations but you will be part of this study which is going to be useful to others in the future.

Alternatives:

Taking part in this study is voluntary. You may choose not to take part in this study. If you decide to take part you can later change your mind and withdraw from the study. Your decision will not change the present or future health care. If you choose not to take part in the study, you will receive the standard treatment for patients with your condition.

Privacy and Confidentiality:

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy:

Does not apply to this research

Financial incentives for participation:

You will not be paid / offered any gifts /incentives for participating in the study.

Authorization to publish the results:

The results of the study would be forwarded to the KLE University, Belgaum as part of requirement towards the completion of MD degree, review and publishing.

In case of the queries during study or in future you may contact following persons,

1. **DR.** _____ Professor& Head, Dept. of Pathology, J.N. Medical College, K.L.E. University, Belgaum - 10	2. **Dr.** _____ Professor & Head of Unit, Dept of General Medicine, JNMC, Belgaum.

3. **Dr.**_____
Investigator,
PG in General Medicine,
JNMC, Belgaum.

CONSENT FORM

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read this consent form, or it has been read to me and has been explained to me in my vernacular language and all my questions have been answered. I will be given a copy of this consent form.

Signature / Left Thumb print of the Participant or legally authorized representative

Participant's name :……...

Signature / Left thumb impression :……...

of the participant

Name of the legally authorized :……...

representative / guardian

Signature / Left thumb impression :……...

Witness' name :……...

Signature / Left thumb impression :……...

Investigator's name and signature :……...

Date:

Place:

ತಿಳುವಳಿಕೆ ಒಪ್ಪಿಗೆ ಪತ್ರ

<u>ಸಂಶೋಧನೆಯ ಶಿರ್ಷಿಕೆ</u> : ಒಂದು ವರ್ಷದ ಎದೆಗೂಡಿನ ಪೊರೆ ಪ್ರವಾಹ ಮತ್ತು ಊಹಾತ್ಮಕ ವೈದ್ಯಕೀಯ ಅಧ್ಯಯನ.

ಮುಖ್ಯ ಸಂಶೋಧಕರು:–

ಡಾ.

ಸ್ನಾತಕೋತ್ತರ ವೈದ್ಯಕೀಯ ವಿದಾರ್ಥಿ

ಸಾಮಾನ್ಯ ಔಷಧಿಗಳ ವಿಭಾಗ

ಜೆ.ಎನ್.ಎಮ್.ಸಿ, ಬೆಳಗಾವಿ

ಮಾರ್ಗದರ್ಶಕರು:–

ಡಾ.

ಪ್ರಧ್ಯಾಪಕರು ಮತ್ತು ತಂಡದ ಮುಖ್ಯಸ್ಥರು

ಸಾಮಾನ್ಯ ಔಷಧಿಗಳ ವಿಭಾಗ

ಜೆ.ಎನ್.ಎಮ್.ಸಿ, ಬೆಳಗಾವಿ

ಸಂಶೋಧನೆಯ ಪರಿಚಯ ಮತ್ತು ಉದ್ದೇಶ :–

ಈ ಸಂಶೋಧನೆಯ ಉದ್ದೇಶ ಎದೆಗೂಡಿನ ಪೊರೆ ಪ್ರವಾಹ ಮತ್ತು ಕಾರಣಗಳನ್ನು ಅಧ್ಯಯನ ಮಾಡುವುದು. ಡಾ. ವಿಜಯ ಜಿ. ಸೋಮನ್ನವರ ಇವರ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ಡಾ. ರಿಶಿ ರಾಮನ ಸಂಶೋಧನೆ ಮಾಡಿರುತ್ತಾರೆ. ಎದೆಗೂಡಿನ ಪೊರೆ ಪ್ರವಾಹಕ್ಕೆ ಕಾರಣಗಳನ್ನು ಅತಿ ಬೇಗನೆ ತಪಾಸಣೆಯ ಮೂಲಕ ತಿಳಿದುಕೊಂಡು ಅದಕ್ಕೆ ಚಿಕಿತ್ಸೆಯನ್ನು ನೀಡುವುದಾಗಿದೆ.

ವಿಧಾನ:–

ನೀವು ಈ ಸಂಶೋಧನೆಯ ಭಾಗವಾಗಲು ಒಪ್ಪಿಕೊಂಡಲ್ಲಿ ನಿಮಗೆ ಖಾಯಿಲೆಗೆ ಸಂಬಂಧಿಸಿದ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲಾಗುವುದು ಹಾಗೂ ವೈದ್ಯಕೀಯ ತಪಾಸಣೆಗೆ ಒಳಪಡಬೇಕಾಗುತ್ತದೆ. ಇದರ ಜೊತೆಗೆ ನೀವು ನಿಮ್ಮ ರಕ್ತ ಹಾಗೂ ಕಫದ ಮಾದರಿಯನ್ನು ನೀಡಬೇಕಾಗುತ್ತದೆ.

ಅನುಕೂಲ ಹಾಗೂ ಅನಾನುಕೂಲತೆಗಳೂ :–

ಈ ಸಂಶೋಧನೆಯ ಸಮಯದಲ್ಲಿ ಆಗುವ ಏಕೈಕ ಅನಾನುಕೂಲತೆಯೆಂದರೆ ತಪಾಸಣಾ ಸಂದರ್ಭದಲ್ಲಿ ಆಗುವ ನೋವು, ಆದರೆ ಇದು ನಿಮಗೆ ನೋವಾಗಿ ಕಂಡರೂ ಮುಂದಿನ ಪೀಳಿಗೆಗೆ ಈ ಸಂಶೋಧನೆಯು ತೊಂಬಾ ಲಾಭದಾಯಕವಾಗಿರುತ್ತದೆ.

ಪರ್ಯಾಯಗಳು :–

ಈ ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ನೀವು ಸ್ವ ಇಚ್ಛೆಯಿಂದ ಮತ್ತು ಒಪ್ಪಿಗೆಯಿಂದ ಭಾಗವಹಿಸಬಹುದು ಅಥವಾ ನಿರಾಕರಿಸಲೂಬಹುದು. ಒಂದು ವೇಳೆ ನೀವು ಸಂಶೋಧನೆಯ ಭಾಗವಾಗಲು ಒಪ್ಪಿಕೊಂಡು ನಂತರದ ದಿನಗಳಲ್ಲಿ ಮನಸ್ಸು ಬದಲಾಯಿಸಿದರೆ ನೀವು ಇದರಿಂದ ಹೊರಬರಬಹುದು. ನಿಮ್ಮ ಈ ನಿರ್ಧಾರವು ನಿಮಗೆ ಈಗ ದೊರೆಯುತ್ತಿರುವ ವೈದ್ಯಕೀಯ ಹಾಗೂ ಇನ್ನಿತರ ಸೇವೆಗಳ ಮೇಲೆ ಯಾವುದೇ ಪ್ರಭಾವ ಬೀರುವುದಿಲ್ಲ ಹಾಗೂ ನಿಮಗೂ ಸಹ ಇತರೆ ರೋಗಿಗಳಂತೆ ನಿಮಗಿರುವ ಖಾಯಿಲೆಗನುಸಾರವಾಗಿ ಚಿಕಿತ್ಸೆ ದೊರೆಯುವುದು.

ಗೌಪ್ಯತೆ :–

ಸಂಶೋಧನೆಯ ಸಂದರ್ಭದಲ್ಲಿ ಸಂಗ್ರಹಿಸಿರುವ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಕಾನೂನಿಗನುಸಾರವಾಗಿ ಗೌಪ್ಯವಾಗಿಡಲಾಗುವುದು. ಎಲ್ಲಿಯೂ ನಿಮ್ಮ ಹೆಸರು ಪ್ರಕಟವಾಗದೇ ನಿಮ್ಮನ್ನು ಗೌಪ್ಯ ಸಂಖ್ಯೆಯಿಂದ ಗುರುತಿಸಲಾಗುವುದು. ಮುಂದಿನ ದಿನಗಳಲ್ಲಿ ಈ ಸಂಶೋಧನಾ ವರದಿಯು ಪ್ರಕಟಗೊಂಡಾಗಲೂ ಸಹ ನಿಮ್ಮ ಹೆಸರನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುವುದು.

ಸಂಸ್ಥೆ ಅಥವಾ ಪ್ರಾಯೋಜಕರ ನೀತಿಗಳು :–

ಅನ್ವಯವಾಗುವುದಿಲ್ಲ.

ಪ್ರೋತ್ಸಾಹ ಧನ :–

ನಿಮಗೆ ಈ ಸಂಶೋಧನೆಯ ಭಾಗವಾಗಲು ಯಾವುದೇ ರೀತಿಯ ಹಣ, ಉಡುಗೊರೆಯನ್ನು ನೀಡಲಾಗುವುದಿಲ್ಲ.

ಫಲಿತಾಂಶ ಪ್ರಕಟಿಸಲು ಇರುವ ಅಧಿಕಾರಗಳು :–

ಈ ಸಂಶೋಧನೆಯ ಫಲಿತಾಂಶವನ್ನು ಕೆ.ಎಲ್.ಇ. ವಿಶ್ವವಿದ್ಯಾಲಯ ಬೆಳಗಾವಿ ಇವರಿಗೆ ಎಮ್.ಡಿ. ಪದವಿ ಪೂರ್ಣಗೊಳಿಸಲು, ವಿಮರ್ಶಿಸಲು ಹಾಗೂ ಪ್ರಕಟಿಸಲು ಕಳುಹಿಸಿಕೊಡಲಾಗುವುದು.

ಸಂಶೋಧನೆಯ ಸಂದರ್ಭದಲ್ಲಿ ಅಥವಾ ಮುಂದೆ ಯಾವುದಾದರೂ ಪ್ರಶ್ನೆಗಳು ಅಥವಾ ಅನುಮಾನಗಳಿದ್ದಲ್ಲಿ ಈ ಕೆಳಗಿನವರನ್ನು ಸಂಪರ್ಕಿಸಬಹುದಾಗಿದೆ.

ಡಾ. ಪ್ರಧ್ಯಾಪಕರು ಮತ್ತು ಮುಖ್ಯಸ್ಥರು ಪ್ಯಾಥೋಲಾಜಿ ವಿಭಾಗ ಜೆ.ಎನ್.ವೈದ್ಯಕೀಯ ಕಾಲೇಜ ಕೆ.ಎಲ್.ಇ. ವಿಶ್ವವಿದ್ಯಾಲಯ, ಬೆಳಗಾವಿ	ಡಾ. ಪ್ರಧ್ಯಾಪಕರು ಮತ್ತು ತಂಡದ ಮುಖ್ಯಸ್ಥರು ಸಾಮಾನ್ಯ ಔಷಧಿಗಳ ವಿಭಾಗ ಜೆ.ಎನ್.ವೈದ್ಯಕೀಯ ಕಾಲೇಜ ಕೆ.ಎಲ್.ಇ. ವಿಶ್ವವಿದ್ಯಾಲಯ, ಬೆಳಗಾವಿ
ಡಾ. ಸಂಶೋಧಕರು ಸ್ನಾತಕೋತ್ತರ ವೈದ್ಯಕೀಯ ವಿದಾರ್ಥಿ ಸಾಮಾನ್ಯ ಔಷಧಿಗಳ ವಿಭಾಗ ಜೆ.ಎನ್.ಎಮ್.ಸಿ, ಬೆಳಗಾವಿ	

ಸಮ್ಮತಿ ಪತ್ರ

ನಾನು ಈ ಕೆಳಗೆ ಸಹಿ ಮಾಡಿ ಸ್ವಚ್ಛಯಿಂದ ಅಭ್ಯಾಸದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪಿಕೊಂಡಿರುತ್ತೇನೆ. ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ನನ್ನ ಒಪ್ಪಿಗೆಯನ್ನು ಹಿಂದೆ ಪಡೆಯಬಹುದು. ನಾನು ಈ ಸಮ್ಮತಿ ಪತ್ರಕ್ಕೆ ಸಹಿ ಮಾಡುವುದರಿಂದ ನನಗೆ ಲಭ್ಯವಿರುವ ಕಾನೂನಿನ ಯಾವುದೇ ಹಕ್ಕುಗಳನ್ನು ಬಿಟ್ಟುಕೊಟ್ಟಿರುವುದಿಲ್ಲ. ಕೆಳಗೆ ನಾನು ಸಹಿ ಮಾಡಿರುವುದರ ಅರ್ಥ ಏನೆಂದರೆ ಮೇಲಿನ ವಿಷಯ ಓದಿ ಅಥವಾ ಓದಿಸಿ ಕೇಳಿ ಪೂರ್ಣ ಸಮ್ಮತಿ ಪತ್ರದಲ್ಲಿರುವ ಎಲ್ಲ ಪ್ರಶ್ನೆಗಳಿಗೆ ಉತ್ತರಿಸಿರುತ್ತೇನೆ.

ಭಾಗವಹಿಸುವವರ ಹೆಸರು — ಸಹಿ/ಹೆಬ್ಬರಳಿನ ಗುರುತು

ಸಾಕ್ಷಿಯ ಹೆಸರು — ಸಹಿ/ಹೆಬ್ಬರಳಿನ ಗುರುತು

ಸಂತೋಷದಕರ ಹೆಸರು — ಸಹಿ

ದಿನಾಂಕ: — ಸ್ಥಳ:

माहिती मिळाल्यानंतर दिलेली सहमती

संशोधनाच्या अभ्यासाचे नांव :

छातीमध्ये पाणी साचण्याच्या रोगाचा वैद्यकिय दृष्टिकोणातून केलेला शास्त्रोक्त अभ्यास- एक वर्णाचा संशोधन अभ्यास-

मुख्य संशोधक: डॉ.
पोस्ट ग्रॅज्युएट विद्यार्थी
सामान्य औषधोपचार विभाग
जे. एन. एम. सी , बेळगांव

मार्गदर्शक : डॉ.
प्रोफेसर व विभाग प्रमुख
सामान्य औषधोपचार विभाग
जे. एन. एम. सी बेळगांव

* प्रस्थावना व उद्देश :

वैद्यकिय दृष्टिकोनातून छातीमध्ये पाणी होवून साचण्याचा आणि असे पाणी उत्पन्न होण्याची कारणे याच्या अभ्यासासाठी हे संशोधन आहे. या अभ्यासाचे प्रमुख संशोधक डॉ. ऋषी रामन व त्यांना मार्गदर्शन करणारे डॉ. विजय जी. सोमन्नवर हे आहेत.

हा अभ्यासक्रम प्राथमिक अवस्थेत छातीमध्ये पाणी साचण्याच्या रोगावरील कारणे शोधून त्याचे प्राथमिक अवस्थेत रोग निवारण कारण्याच्या औषधोपचारचा आहे.

पध्दत :-

जर का तुम्ही या संशोधनात सहभागी होणार असाल तर, तुम्हास तुमच्या संबंधीची माहिती व पूर्व इतिहास विचारण्यात येईल व वैद्यकिय दृष्टिकोणातून शारीरिक तपासणी करुन त्यासंबंधी संशोधन करण्यात येईल. या संशोधनासाठी

तुमचा रक्ताचा व थुंकीचा नमुना घेणे जरुरीचे असेल व तो घेऊन त्याची योग्य अशी तपासणी केली जाईल.

*** धोके व फायदेः-**

या अभ्यास संशोधनासाठी तुमच्या छातीमधील तरल पदार्थाचा नमुना घेण्यात येतो. व त्यावेळी तुम्हास ज्याठिकाणी कोदरण्याची क्रिया केली जाईल व त्यामुळे त्या भागामध्ये त्रास व वेदना होवून कळा येतील.

या तपासणीमुळे/ संशोधनामुळे तुम्हास कोणता फायदा होईल किंवा नाही हे आम्हांस सांगता येत नाही. परंतु हा एक अभ्यासाचा भाग आहे आणि भविष्यात या संशोधनाचा उपयोग इतरांना होणार आहे.

***पर्याय :-**

या संशोधन अभ्यासामध्ये तुम्ही स्वतःहून सहभागी होवू शकता. तुम्ही या अभ्यासक्रमामध्ये सहभागी होत नाही असे सुध्दा सांगू शकता. जर का तुम्ही स्वतःहून या अभ्यासक्रमात सहभागी झाला असाल तर व नंतर तुमच्या मनात तुमचे नांव या अभ्यासक्रमातून काढून घेऊ इच्छित असाल तर ते सुध्दा तुम्ही करु शकता. या तुमच्या निर्णयामुळे तुम्हास भविष्यात मिळणारी वैद्यकिय मदत किंवा सेवा यावर कोणताही परीणाम होणार नाही.

हा अभ्यासक्रम कोणत्याही वेळी संशोधक करणारे डॉक्टर किंवा या कार्यक्रमाचे प्रयोजक हे हा अभ्यासक्रम बंद करु शकतात. जर तुम्ही या अभ्यासात सहभागी होत नसाल तरी सुध्दा तुम्हास या ठिकाणी योग्य ते संपूर्ण वैद्यकिय उपचार मिळतील.

* गुप्तता :-

या संशोधनामध्ये तुमच्या संदर्भात मिळवलेली माहीती ही कायद्याच्या तरतुदीनुसार गुप्त ठेवण्यात येईल. या संशोधन अभ्यासक्रमामध्ये

तुम्हास कोड नंबर देवून तुमची ओळख केली जाईल. या अभ्यासक्रमामध्ये मिळवलेली माहिती प्रसिध्द केली जाईल. परंतु तुमचे नांव व सहभाग हा या प्रसिध्दीपासून गुप्त ठेवण्यात येईल.

* **संस्थेचे/प्रयोजन करण्याऱ्याचे धोरण:-**

या संशोधनास हे लागू नाही

* **कार्यक्रमात भाग घेतल्याबद्दल मिळणारी आर्थिक मदत :-**

या अभ्यासक्रमामध्ये भाग घेतल्याबद्दल तुम्हास कोणतीही आर्थिक मदत किंवा भेट वस्तु देण्यात येणार नाही.

* **अभ्यासाचे निष्कर्ष प्रसिध्द करण्याचे आधिकार :-**

या अभ्यासक्रमाचे निष्कर्ष हे के. एल. ई युनिव्हर्सिटी बेळगांव यांना पाठविण्यात येतील व हा अभ्यासक्रम तपासून व प्रसिध्दीस देऊन एम डी. ही पदवी पूर्ण करण्याचा हा एक आमचा भाग आहे.

* **अभ्यासक्रमासंबंधी कोणतेही प्रश्न असल्यास किंवा भविष्यात शंका निर्माण झाल्यातर या बाबत खालील व्यक्तिशी संपर्क साधावा.**

१. डॉ.
प्रोफेसर व पॅथॉलॉजी विभाग प्रमुख
जे एन. एम सी मेडिकल कॉलेज
के.एल.ई युनिव्हर्सिटी बेळगांव-10

२. डॉ.
प्रोफेसर व विभाग प्रमुख
सामान्य औषधोपचार विभाग
जे एन. एम सी मेडिकल कॉलेज
बेळगांव 9845710945

३. डॉ.
संशोधक
पोष्ट ग्रॅज्युएट,
सामान्य औषधोपचार विभाग
जे एन. एम सी मेडिकल कॉलेज
बेलगांव 847969903

सम्मती पत्र

मी खाली सही करणारा स्वत:हून अभ्यासामध्ये भाग घेण्यासाठी हे मान्य करत आहे. मी माझे नांव यातून कोणत्याही क्षणी काढून घेवू शकतो. हा नमूना फार्म सही केल्यामुळे मी माझे कोणतेही नैतिक अधिकार सोडून देत नाही आहे. हे वाचून पाहिल्यानंतर किंवा ते वाचून दाखविल्या नंतर मी माझी सही या सम्मती पत्रावर करत आहे. व अशा प्रकारे मी सर्व प्रश्नाची उत्तरे देत आहे.

भाग घेणाऱ्याचे नांव : .....सही/अंगठा

साक्षीदाराचे नांव: .....सही/अंगठा

तपासणाऱ्याचे नांव:.....सही

तारीख: ठिकाण:

जानकारी प्राप्त होने के बाद दि गई सम्मति

संशोधन अभ्यास का नाम

सीने में पानी के संचय की बिमारिका वैद्यकिय बाते ध्यान में रखकर किया हुआ शास्त्रोक्त अभ्यास- एक साल का संशोधन अभ्यास.

मुख्य संशोधक : डॉ.
पोस्ट ग्रॅज्युएट छात्र
सामान्य औषधोपचार विभाग
जे. एन. एम. सी, बेळगांव

मार्गदर्शक : डॉ.
प्रोफेसर व विभाग प्रमुख
सामान्य औषधोपचार विभाग
जे. एन. एम. सी, बेळगांव

* प्रस्तावना और हेतु :

वैद्यकिय बाते ध्यान में लेकर सीने में पानी का संचय और ऐसा पाणी जमा होने की वजह इसका यह संशोधन अभ्यासक्रम है। इस अभ्यास के प्रमुख संशोधक डॉ. ऋषि रामन और उनके मार्गदर्शक डॉ. विजय जी. सोमन्नवर है।

यह अभ्यासक्रम प्राथमिक स्थिती में सीने में पानी जमा होना, क्यों होता है ? और उसकी वजह क्या है ? इसका परामर्श करना और उसका प्राथमिक स्थितीमें औषधोपचार करके रोग का निवारण करना है।

* पध्दती :-

आप यदी इस संशोधन में सामिल होते है तो आपको आपका इसके बारे में पूर्व इतिहास पुछा जाएगा और वैद्यकिय बाते ध्यानमें रखकर आपके शरीर की जाँच करके, इस संबंधमें संशोधन किया जाएगा। इस संशोधन हेतु आपके रक्त का और थुक का नमुना लिया जाना जरुरी है और उसके बाद उनकी वैद्यकिय जांच की जायेगी।

* धोका और फायदे:

इस अभ्यास संशोधन हेतु आपके सीने में से तरल पदार्थ का नमुना लिया जाएगा। और वह लेते समय आपको जिस जगह पर हम खुरचणे की प्रक्रिया करते है उस जगह पर वेदना और दर्द होगा।

इस जांच में आपको कोई फायदा होना या नही, यह हम बता नही सकते लेकिन इस जांचकी वजहसे भविष्यमें अन्य लोगोंको इसका लाभ होगा।

* पर्याय :-

इस संशोधन अभ्यासमें आप खूद सम्मलित हो सकते है। आप इस अभ्यासमें सम्मलित नही होना चाहते यह भी आप बना सकते है। यदि आप इस अभ्यासमे खूद सम्मलित हो गये है तो आपको इसके बाद भी आपके मनमें शंका उत्पन्न हो गयी तो, आपका नाम इस अभ्यासक्रम से आप कम करते है। इस निर्णय से आपको भविष्य में मिलनेवाले वैद्यकिय मदत अथवा सेवाओं पर कोई भी असर नही होगा।

यह अभ्यास संशोधन कभी भी संशोधक करनेवाले डॉक्टर या इसके प्रयोजक, इसको समाप्त कर सकते है। यदी आप इस अभ्यासक्रमसे सम्मलित नही होते है तो भी आपको इस जगह से योग्य ऐसी अभी वैद्यकिय चिकित्सा मिलेगी।

* गोपनियता :-

इस संशोधनमें आपके द्वारा प्राप्त की हुई जानकारी कानून के मुताबिक गोपनिय रखी जायेगी। इस संशोधन अभ्यासक्रममें आपको एक कोड नंबर दिया जायेगा और कोड नंबर की मदत से आपकी पहचान की जायेगी। इस अभ्यासक्रम में मिली हुई जानकारी प्रसिध्द कि जायेगी किंतु आपका नाम और सम्मति इस प्रसिध्दी में गोपनिय रखी जाएगी।

* संस्थाका या प्रयोजन करनेवाले का हेतु-

इस संशोधन के लिए यह लागू नही है।

* सम्मिलित होनेवाले को मिलनेवाली आर्थिक सहायता-

इस अभ्यासक्रम में सम्मिलित होनेवाले को कोई भी आर्थिक सहायता या इनाम नही मिलेगा।

* अभ्यास के निष्कर्ष प्रसिध्द करने के आधिकार-

इस अभ्यास के निष्कर्ष के के. एल ई युनिव्हर्सिटी बेलगाम को भेजे जायेगे और इस अभ्यासक्रम को प्राप्त करके तथा उसको प्रसिध्दी देकर एम डी. पदवी प्राप्त करना यह एक इस संशोधन का हिस्सा है।

आपके इस संशोधन हेतु कोई प्रश्न है, या भविष्य में शंका हो गई तो आप निम्नलिखित व्यक्ति से संपर्क कर सकते है।

१. डॉ.
प्रोफेसर व पथालाजा विभाग प्रमुख
जे एन. एम सी मेडिकल कॉलेज
के.एल.ई युनिव्हर्सिटी बेळगांव-10

२. डॉ.
प्रोफेसर व विभाग प्रमुख
सामान्य औषधोपचार विभाग
जे एन. एम सी मेडिकल कॉलेज
बेळगांव

३. डॉ.
संशोधक
पोष्ट ग्रॅज्युएट,
सामान्य औषधोपचार विभाग
जे एन. एम सी मेडिकल कॉलेज
बेळगांव

सम्मती पत्र

मै निचे सही करनेवाला स्वइच्छेसे इस अभ्यासमें भाग लेने के लिए मान्यता देता हू । मै अपना नाम किसी भी वक्त इसमेसे वापस ले सकता हू और इस सम्मती के कारण मै मेरे कोई भी कानुनी हक्क नही छोड़ रहा हू । यह सब उपर के विषय के बारे में स्वयं पढकर या पढने के सूनने के बाढ मैं इस सम्मती पत्र पर अपने हस्ताक्षर कर के सभी प्रश्नो का उत्तर दिया हू ।

हिस्सा लेने वाले का नाम : .....सही/अंगठा

साक्षीदार का नाम: .....सही/अंगठा

परीक्षण करने वाले का नाम:.....सही

तारीख: स्थळ:

ANNEXURE II – PROFORMA

CASE NO:

NAME:

AGE/SEX:

IP NO.:

ADDRESS:

OCCUPATION:

HISTORY:

COMPLAINTS AT PRESENTATION: YES/NO

FEVER

COUGH

CHEST PAIN

BREATHLESSNESS

Any other complaint

Past history: Yes/No

Tuberculosis

Bronchial Asthma

Ischemic heart disease

Diabetes mellitus

Hypertension

Any other

Family history

Personal history

Treatment history

PHYSICAL EXAMINATION:

Temperature:

Pulse:

Respiratory rate:

Blood pressure:

Spo2:

SYSTEMIC EXAMINATION:

R. S.:

C.V.S.:

P.A.:

C.N.S.:

Pleural Cell Type	
Pleural Cell Count	
Pleural ADA	
Pleural Protein	
Pleural Glucose	
Pleural LDH	
Serum Protein	
Gram Stain	
Culture	
AFB	

Diagnosis:

KEY TO MASTER CHART

IP No.	:	Inpatient Number
TB	:	Tuberculosis
IHD	:	Ischemic Heart Disease
RHD	:	Rheumatic Heart Disease
RD	:	Renal Disease
RVD	:	Retroviral Disease
CLD	:	Chronic Liver Disease
Nil	:	No co-morbidities
Pleural LDH	:	Pleural Lactate Dehydrogenase
1	:	<222
2	:	>222
Pleural ADA	:	Adenosine Deaminase
1	:	<30U/L
2	:	31- 40U/L
3	:	41-60U/L
4	:	>60U/L
Pleural Cell Count		
1	:	<200
2	:	>200-1000
3	:	1000- 5000
4	:	>5000
Pleural Cell Type		
L	:	Lymphocytic Predominance
N	:	Neutrophilic Predominance

Pleural Glucose

1 : <60 g/dl

2 : >60 g/dl

Sr no	IP No.	Age	Sex	Fever	Cough	Chest Pain	Breathlessness	Co-morbidities	Chest Xray Size	Chest Xray Side	Pleural fluid Gross appearance	Cell Count	Cell Type	Pleural LDH	Pleural protein:Serum protein	Pleural Glucose	Pleural ADA	Gram Stain	Culture	AFB	Pleural Cytology for Malignant Cells	Type	Etiology
1	819191	71	Male	No	Yes	Yes	Yes	IHD	Moderate	Left	Pale Yellow	1	L	1	2.8: 5.2	2	1	Negative	Negative	Negative	Negative	Transudative	CCF
2	821658	67	Male	No	Yes	No	Yes	CLD	Mild	Right	Pale Yellow	1	L	1	0.9:4.7	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
3	821438	60	Female	No	Yes	No	Yes	Overlapping	Moderate	Right	Pale Yellow	1	N	1	0.7:6.3	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
4	887326	67	Female	Yes	Yes	Yes	Yes	DM	Moderate	Right	Haemorrhagic	4	N	2	4.8:6.4	2	1	Negative	Negative	Negative	Positive	Exudative	Malignant
5	888546	42	Male	No	Yes	Yes	Yes	HTN	Mild	Left	Dark Yellow	4	N	2	3.5:6.3	2	1	Negative	Positive	Negative	Negative	Exudative	Parapneumonic
6	771433	52	Male	Yes	Yes	Yes	Yes	RVD	Moderate	Left	Dark Yellow	3	L	2	5.3:6.8	1	4	Negative	Negative	Negative	Negative	Exudative	TB
7	785623	47	Male	No	No	No	Yes	IHD	Moderate	Left	Pale Yellow	1	L	2	1.3:5.6	2	1	Negative	Negative	Negative	Negative	Transudative	CCF
8	836980	74	Male	Yes	Yes	No	No	Overlapping	Moderate	Right	Dark Yellow	2	L	2	4.3:7.2	2	4	Negative	Negative	Negative	Negative	Exudative	TB
9	802367	39	Male	No	Yes	No	Yes	CLD	Mild	Right	Pale Yellow	1	N	1	0.8:5.2	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
10	795409	56	Male	Yes	Yes	No	Yes	DM	Massive	Left	Dark Yellow	1	L	2	4.8:7	2	4	Negative	Negative	Negative	Negative	Exudative	TB
11	784125	65	Female	No	No	Yes	Yes	IHD	Moderate	Bilateral	Pale Yellow	1	L	1	2.1:6.8	2	1	Negative	Negative	Negative	Negative	Transudative	CCF
12	815634	63	Female	No	No	No	Yes	RD	Moderate	Left	Pale Yellow	1	L	1	1.1:6.0	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
13	836008	20	Male	Yes	Yes	No	Yes	Nil	Massive	Right	Dark Yellow	2	L	2	5.3:7.2	2	3	Negative	Negative	Negative	Negative	Exudative	TB
14	769078	58	Male	No	No	No	Yes	DM	Massive	Right	Dark Yellow	2	L	2	5.0:7.5	2	3	Negative	Negative	Negative	Negative	Exudative	TB
15	809432	24	Female	Yes	Yes	No	Yes	Nil	Massive	Left	Pus(Empyema)	4	N	2	5.5:7.0	1	1	Negative	Negative	Negative	Negative	Exudative	Empyema
16	746923	37	Male	No	Yes	No	Yes	CLD	Moderate	Left	Pale Yellow	1	L	1	1.2:6.2	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
17	797312	35	Male	Yes	No	No	Yes	DM	Moderate	Right	Pale Yellow	2	L	2	5.1:7.8	1	2	Negative	Negative	Negative	Negative	Exudative	TB
18	889317	55	Female	Yes	No	No	No	DM	Mild	Left	Dark Yellow	2	L	2	4.1:6.8	1	3	Negative	Negative	Negative	Negative	Exudative	TB
19	794096	64	Female	Yes	Yes	No	Yes	RD	Moderate	Right	Pale Yellow	1	N	1	0.4:6.3	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
20	785108	19	Male	Yes	Yes	No	Yes	Nil	Moderate	Left	Dark Yellow	2	L	2	4.1:7.0	1	4	Negative	Negative	Negative	Negative	Exudative	TB
21	810749	34	Male	No	Yes	No	No	Nil	Mild	Right	Dark Yellow	3	N	2	4.3:7.6	1	1	Negative	Negative	Negative	Negative	Exudative	Parapneumonic
22	840854	61	Male	No	Yes	Yes	No	IHD	Mild	Right	Pale Yellow	1	N	1	0.8: 5.6	2	1	Negative	Negative	Negative	Negative	Transudative	CCF
23	858074	39	Female	No	No	No	Yes	CLD	Massive	Right	Pale Yellow	1	N	1	0.8:6.4	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
24	780531	18	Female	No	No	No	Yes	Nil	Moderate	Left	Dark Yellow	3	L	2	3.8:6.1	1	4	Negative	Negative	Negative	Negative	Exudative	TB
25	846109	54	Male	Yes	Yes	No	Yes	RD	Moderate	Bilateral	Pale Yellow	1	L	1	0.9:4.3	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
26	854096	28	Male	Yes	Yes	No	No	Nil	Mild	Right	Dark Yellow	2	L	2	3.8:7.0	1	4	Negative	Negative	Negative	Negative	Exudative	TB
27	770091	56	Male	No	Yes	No	Yes	CLD	Massive	Right	Pale Yellow	1	L	1	1.3:6.1	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
28	829155	64	Male	No	No	No	Yes	HTN	Moderate	Left	Dark Yellow	2	L	2	4.1::7.0	1	2	Negative	Negative	Negative	Negative	Exudative	TB
29	829153	32	Female	Yes	Yes	No	Yes	CLD	Massive	Right	Dark Yellow	1	L	1	1.5:5.9	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
30	829480	52	Male	Yes	Yes	No	No	DM	Mild	Left	Dark Yellow	2	L	2	4.3:6.8	1	3	Negative	Negative	Negative	Negative	Exudative	TB
31	847581	18	Female	Yes	No	No	Yes	DM	Massive	Left	Pus(Empyema)	4	N	2	4.3:7.4	1	1	Negative	Negative	Negative	Negative	Exudative	Empyema
32	826113	57	Male	No	Yes	No	Yes	Overlapping	Massive	Right	Dark Yellow	2	L	2	4.4:6.8	2	3	Negative	Negative	Negative	Negative	Exudative	TB
33	825981	26	Female	Yes	No	No	Yes	Nil	Massive	Right	Dark Yellow	3	L	2	4.2:7.2	1	4	Negative	Negative	Negative	Negative	Exudative	TB
34	827943	59	Male	Yes	Yes	No	No	Overlapping	Moderate	Right	Dark Yellow	2	L	2	4.1:7.3	1	3	Negative	Negative	Negative	Negative	Exudative	TB
35	827908	38	Male	Yes	No	No	No	HTN	Massive	Right	Dark Yellow	4	N	2	4.8:6.9	1	1	Negative	Positive	Negative	Negative	Exudative	Parapneumonic
36	827498	53	Female	No	Yes	No	Yes	HTN	Massive	Left	Dark Yellow	2	L	2	3.3:6.9	2	3	Negative	Negative	Negative	Negative	Exudative	TB
37	887326	22	Female	Yes	No	No	Yes	Nil	Moderate	Left	Dark Yellow	4	L	2	4.0:7.0	2	4	Negative	Negative	Positive	Negative	Exudative	TB
38	813239	38	Male	Yes	No	No	No	Overlapping	Mild	Right	Dark Yellow	4	L	2	5.7:7.4	1	3	Negative	Negative	Negative	Negative	Exudative	TB
39	813466	35	Male	Yes	No	No	Yes	HTN	Moderate	Right	Dark Yellow	4	N	2	5.1:7.8	2	1	Negative	Negative	Negative	Negative	Exudative	Parapneumonic
40	810951	76	Male	Yes	No	No	No	Overlapping	Mild	Left	Dark Yellow	2	L	2	5.1:7.4	1	3	Negative	Negative	Negative	Negative	Exudative	TB
41	832096	46	Male	Yes	No	No	Yes	CLD	Moderate	Right	Pale Yellow	1	L	1	1.0:6.9	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
42	840941	37	Female	No	Yes	Yes	Yes	RD	Moderate	Bilateral	Pale Yellow	1	L	1	1.3:6.2	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
43	798431	82	Male	Yes	No	No	Yes	Overlapping	Moderate	Left	Dark Yellow	2	L	2	4.2:6.8	1	3	Negative	Negative	Negative	Negative	Exudative	TB
44	764239	52	Female	No	No	Yes	Yes	RHD	Mild	Left	Pale Yellow	1	L	1	1.7:6.8	2	1	Negative	Negative	Negative	Negative	Transudative	CCF
45	785430	74	Female	Yes	No	Yes	No	HTN	Mild	Right	Dark Yellow	3	L	2	3.1:6.4	2	3	Negative	Negative	Negative	Negative	Exudative	TB
46	780965	36	Female	Yes	No	No	Yes	DM	Moderate	right	Pus(Empyema)	4	N	2	5.1:8.0	1	1	Negative	Negative	Negative	Negative	Exudative	Empyema
47	786549	25	Male	Yes	No	No	No	Nil	Moderate	left	Dark Yellow	3	L	2	4.5:7.6	1	4	Negative	Negative	Positive	Negative	Exudative	TB
48	765329	67	Male	No	Yes	Yes	No	Overlapping	Mild	right	Dark Yellow	3	L	2	4.4:6.8	2	3	Negative	Negative	Negative	Negative	Exudative	TB
49	788542	27	Female	No	No	No	Yes	RHD	Mild	left	Pale Yellow	2	N	1	0.3:5.2	2	1	Negative	Negative	Negative	Negative	Transudative	CCF
50	809608	70	Female	Yes	No	No	Yes	Overlapping	Massive	right	Dark Yellow	3	L	2	4.9:7.4	2	3	Negative	Negative	Negative	Negative	Exudative	TB
51	816874	61	Male	Yes	No	No	No	HTN	Mild	right	Dark Yellow	3	L	2	5.5:6.7	2	3	Negative	Negative	Negative	Negative	Exudative	TB
52	798540	39	Male	No	Yes	No	Yes	CLD	Moderate	right	Pale Yellow	2	N	1	0.4:5.8	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
53	785996	44	Female	No	Yes	No	Yes	CLD	Massive	right	Pale Yellow	1	N	1	0.7:6.2	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
54	763497	49	Male	No	Yes	No	Yes	IHD	Massive	Left	Pale Yellow	2	N	1	0.5:6.7	2	1	Negative	Negative	Negative	Negative	Transudative	CCF
55	754398	69	Female	Yes	No	No	Yes	DM	Moderate	Right	Dark Yellow	3	L	2	3.8:6.2	1	3	Negative	Negative	Negative	Negative	Exudative	TB
56	765486	64	Male	No	Yes	No	No	HTN	Moderate	right	Dark Yellow	3	L	2	4.3:6.7	1	3	Negative	Negative	Negative	Negative	Exudative	TB
57	765987	47	Male	No	Yes	No	Yes	CLD	Massive	right	Pale Yellow	2	L	1	1.8:6.8	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
58	796541	78	Female	Yes	Yes	No	Yes	Overlapping	Moderate	right	Dark Yellow	2	L	2	4.9:7.6	2	3	Negative	Negative	Negative	Negative	Exudative	TB
59	845612	54	Female	No	Yes	No	Yes	CLD	Massive	right	Pale Yellow	1	N	1	1.1:6.9	1	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
60	813406	27	Male	Yes	Yes	No	Yes	Nil	Massive	left	Dark Yellow	3	L	2	3.1:6.3	1	4	Negative	Negative	Positive	Negative	Exudative	TB
61	823986	54	Male	No	Yes	No	Yes	RD	Moderate	Bilateral	Pale Yellow	2	L	1	0.3:5.8	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia

62	854387	65	Female	Yes	No	No	No	DM	Mild	right	Dark Yellow	3	L	2	3.3:7.1	1	3	Negative	Negative	Negative	Negative	Exudative	TB
63	796543	35	Male	Yes	Yes	No	Yes	DM	Massive	right	Dark Yellow	3	L	2	3.2:6.2	1	3	Negative	Negative	Negative	Negative	Exudative	TB
64	795487	23	Male	No	No	Yes	No	Nil	Mild	right	Pale Yellow	2	N	1	0.4:5.9	1	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
65	754008	48	Male	Yes	No	No	Yes	HTN	Massive	right	Dark Yellow	3	L	2	3.7:6.0	1	3	Negative	Negative	Negative	Negative	Exudative	TB
66	765433	27	Male	No	No	Yes	No	Nil	Mild	right	Pale Yellow	2	L	1	0.8:6.0	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
67	795321	43	Female	Yes	No	No	Yes	DM	Massive	right	Dark Yellow	3	L	2	3.8:7.1	1	3	Negative	Negative	Negative	Negative	Exudative	TB
68	813288	62	Female	Yes	No	No	No	HTN	Mild	right	Dark Yellow	3	L	2	4.4:6.6	1	3	Negative	Negative	Negative	Negative	Exudative	TB
69	846475	57	Female	No	Yes	Yes	Yes	IHD	Massive	left	Pale Yellow	3	N	1	0.8:6.5	1	1	Negative	Negative	Negative	Negative	Transudative	CCF
70	815601	47	Male	No	No	Yes	No	Overlapping	Mild	right	Dark Yellow	3	L	2	4.5:7.9	1	3	Negative	Negative	Negative	Negative	Exudative	TB
71	839641	60	Female	Yes	Yes	No	Yes	HTN	Massive	right	Haemorrhagic	4	L	2	4.5:7.2	1	1	Negative	Negative	Negative	Positive	Exudative	Malignant
72	838514	43	Male	Yes	No	No	No	Nil	Moderate	left	Dark Yellow	2	L	2	4.1:6.8	1	4	Negative	Negative	Negative	Negative	Exudative	TB
73	841075	45	Male	Yes	No	No	Yes	RVD	Massive	right	Dark Yellow	3	L	2	4.1:7.0	1	3	Negative	Negative	Negative	Negative	Exudative	TB
74	817320	59	Male	No	No	Yes	Yes	DM	Mild	right	Pale Yellow	2	N	1	1.3:6.8	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
75	808127	32	Male	Yes	Yes	No	No	Nil	Mild	right	Dark Yellow	4	L	2	3.8:6.4	1	3	Negative	Negative	Negative	Negative	Exudative	TB
76	846319	20	Male	Yes	No	No	Yes	Nil	Moderate	left	Dark Yellow	3	N	2	5.0:7.6	1	1	Negative	Negative	Negative	Negative	Exudative	Parapneumonic
77	848752	39	Male	Yes	No	Yes	No	HTN	Mild	right	Dark Yellow	4	L	2	3.9:6.5	2	3	Negative	Negative	Negative	Negative	Exudative	TB
78	756031	39	Male	No	Yes	No	Yes	Overlapping	Moderate	right	Pale Yellow	2	L	1	1.4:7.0	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
79	839066	47	Male	Yes	Yes	No	No	HTN	Massive	right	Dark Yellow	4	L	2	4.3:6.4	1	4	Negative	Negative	Negative	Negative	Exudative	TB
80	838482	22	Male	Yes	Yes	Yes	No	HTN	Mild	right	Dark Yellow	3	N	2	4.1:7.2	2	1	Positive	Negative	Negative	Negative	Exudative	Parapneumonic
81	843547	39	Male	No	No	No	Yes	CLD	Massive	right	Dark Yellow	2	L	1	1.3:7.0	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
82	844842	42	Male	No	No	No	Yes	RVD	Moderate	left	Dark Yellow	2	L	2	4.1:7.0	1	4	Negative	Negative	Negative	Negative	Exudative	TB
83	741808	52	Male	No	Yes	No	No	IHD	Mild	right	Pale Yellow	2	N	1	1.2:6.4	2	1	Negative	Negative	Negative	Negative	Transudative	CCF
84	846298	49	Male	No	No	No	Yes	Nil	Moderate	right	Dark Yellow	3	L	2	3.0:6.5	1	4	Negative	Negative	Negative	Negative	Exudative	TB
85	845531	38	Male	No	Yes	No	Yes	IHD	Moderate	left	Pale Yellow	2	L	1	1.8:6.8	2	1	Negative	Negative	Negative	Negative	Transudative	CCF
86	836534	44	Male	No	Yes	No	No	HTN	Mild	right	Dark Yellow	3	L	2	3.6:7.0	1	4	Positive	Negative	Negative	Negative	Exudative	TB
87	846545	25	Female	No	Yes	No	Yes	Nil	Moderate	left	Pale Yellow	4	N	2	4.3:6.8	2	1	Negative	Negative	Negative	Negative	Exudative	Parapneumonic
88	846799	46	Male	Yes	Yes	No	No	HTN	Moderate	right	Dark Yellow	3	L	2	5.2:8.2	1	4	Negative	Negative	Negative	Negative	Exudative	TB
89	844896	35	Male	No	Yes	No	No	DM	Moderate	right	Dark Yellow	3	L	2	5.0:7.6	1	4	Negative	Negative	Negative	Negative	Exudative	TB
90	847469	32	Male	Yes	Yes	No	No	CLD	Moderate	right	Dark Yellow	2	L	2	5.1:6.9	1	4	Negative	Negative	Negative	Negative	Exudative	TB
91	847898	28	Male	No	Yes	No	No	Nil	Mild	left	Dark Yellow	3	N	2	4.1:7.5	1	1	Negative	Negative	Negative	Negative	Exudative	Parapneumonic
92	876484	65	Male	No	No	Yes	No	Overlapping	Mild	right	Dark Yellow	3	L	2	4.0:6.8	2	4	Negative	Negative	Negative	Negative	Exudative	TB
93	737004	39	Female	No	Yes	No	Yes	IHD	Massive	left	Pale Yellow	2	L	1	1.2:6.3	1	1	Negative	Negative	Negative	Negative	Transudative	CCF
94	787687	63	Male	Yes	No	No	No	Nil	Mild	right	Dark Yellow	3	L	1	3.9:7.0	1	2	Negative	Negative	Negative	Negative	Exudative	TB
95	841709	37	Male	No	Yes	No	Yes	RD	Moderate	Bilateral	Pale Yellow	2	L	1	1.5:6.8	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
96	770235	62	Female	No	No	No	Yes	Overlapping	Moderate	Right	Dark Yellow	3	L	2	4.2:6.7	2	2	Negative	Negative	Negative	Negative	Exudative	TB
97	748291	34	Male	No	No	Yes	Yes	RD	Massive	Right	Pale Yellow	3	L	1	0.9:5.9	1	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
98	781987	32	Female	No	Yes	No	No	HTN	Mild	Right	Dark Yellow	3	N	2	4.0:6.8	2	1	Negative	Negative	Negative	Negative	Exudative	Parapneumonic
99	793906	36	Male	No	Yes	No	Yes	RD	Moderate	Bilateral	Pale Yellow	2	L	1	1.0:6.8	2	1	Negative	Negative	Negative	Negative	Transudative	Hypoproteinemia
100	800210	56	Male	No	Yes	No	Yes	IHD	Moderate	Right	Pale Yellow	2	L	1	0.9:6.0	1	1	Negative	Negative	Negative	Negative	Transudative	CCF

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