INTRODUCTION

Pleural Effusion is an abnormal collection of fluid in the pleural space resulting from excess fluid production or decreased absorption **(Diaz-Guzman and Dweik., 2007).**

The tests most commonly used to diagnose and evaluate pleural effusion include Chest x-ray, Computed tomography (CT) scan of the chest, Ultrasound of the chest (US), Thoracocentesis and pleural fluid analysis. When the pleural effusion has remained undiagnosed despite previous, less-invasive tests, such as Abrams' needle pleural biopsy and thoracoscopy may be performed **(National Cancer Institute., 2006).**

Transthoracic ultrasound has received increased interest from chest physicians in recent years as it has the advantages of bedside availability, absence of radiation, and guided aspiration of fluid-filled areas and solid tumors **(Beckh et al., 2002).**

The value of Chest Ultrasound for diagnosis of pleural effusion is well documented. Even small amounts of pleural effusion can be detected accurately with Chest Ultrasound examination **(Daniel., 2007).**

Chest Ultrasound has been proved a reliable, efficient, and informative imaging modality for the evaluation of a wide variety of chest diseases **(Beckh et al., 2002).**

The Chest Ultrasound image of pleural effusion is characterized by an echo-free space between the visceral and parietal pleura. This space may change in shape with respiration. The effusion can be free or encapsulated. The compressive atelectasis of the lungs in a large effusion can be seen as a tongue like structure within the effusion. Chest Ultrasound is helpful in determining the nature of pleural opacity, identifying minimal or loculated effusion, and discriminating between subpulmonary and subphrenic effusions. Ultrasound is very powerful in evaluation of pleural effusion, differentiating minimal pleural effusion from pleural thickening may sometimes be difficult **(Daniel., 2007).**

Abrams needle biopsy is easy to use, safe, inexpensive, rapid, and can be performed as a bedside procedure in the department. **(Chakrabarti., 2006).**

Abrams needle pleural biopsy still has a place in the diagnosis of exudative pleural effusions and should not be abandoned because of its lower cost and its relatively high safety, simplicity, and diagnostic sensitivity for metastatic pleural diseases and tuberculous pleurisy **(Baumann., 2006).**

Chest Ultrasound is recommended to decrease incidence of post procedure pneumothorax, which increase in blind Abrams pleural biopsy **(Gouda., 2006).**

Aim of the Work

This study aims to compare the diagnostic yield and complication of Abrams' needle pleural biopsy with or without ultrasound guidance in patients with undiagnosed exudative pleural effusion.

ETIOLOGY OF PLEURAL EFFUSION

The initial step in establishing the etiology of an effusion is to determine whether the fluid is a transudate or an exudate. Since these two types of effusions arise by different mechanisms, the classification is useful in identifying the underlying disorder **(Heffner., 1998)**.

A. Transudative pleural Effusions:

Transudative pleural effusions accumulate as a result of imbalance in the normal relationship between capillary hydrostatic pressure and colloid osmotic pressure.Because the pleural surfaces are not affected, the protein concentration and lactic dehydrogenase levels in the fluid are low. Also, white and red blood cells counts are low and pleural fluid glucose and amylase levels are close to those in the plasma. **(Porcel and Vives., 1999).**

I. Congestive heart failure:

Congestive heart failure is one of the most common causes of pleural effusions **(Seaton., 2000)**.

Pleural effusions regularly develop with biventricular heart failure in the presence of both systemic and pulmonary venous hypertension **(Sue., 2000).**

II. Liver Cirrhosis:

Pleural effusions occur in approximately 6% of patients with cirrhosis of the liver and clinical ascites **(Kirsh., 2000).**

III. Nephrotic Syndrome:

Pleural effusion results from reductions in intravascular oncotic pressure that allows an increased movement of fluid out of pleural capillaries and into the pleural space. The pleural effusions are usually bilateral and are commonly intrapulmonary in location. **(Van Beck., 1999)**.

IV. Other Renal Causes:

A) Peritoneal dialysis:

Pleural effusions are regularly found in patients undergoing peritoneal dialysis. The mechanisms are similar to that responsible for pleural effusions in association with cirrhosis and ascites. **(Tag El-Din et al., 2000a)**.

B) Acute glomerulonephritis:

Acute glomerulonephritis in children is associated with pleural effusion in about half of the cases. **(Sahn., 1988)**.

C) Urinary tract obstruction: (Kinasewitz., 1997).

V. Constrictive Pericarditis:

Up to 55% of the cases of constrictive pericarditis were associated with pleural effusions. Constrictive pericarditis should be considered in any patient who presents with unexplained pleural effusion, particularly if there is evidence of elevated systemic venous pressure **(Sadikot et al., 2000)**.

VI. Myxoedema:

Myxoedema may cause pleural effusion, because of either ascites or pericardial effusion or very rarely directly **(Kinsewitz., 1997).**

VII. Pulmonary embolism:

A pleural effusion due to pulmonary embolism is transudative about 25% of cases. Transudative effusions seem to result from the systemic venous hypertension of right ventricular failure. When no obvious cause of a transudative effusion is apparent, pulmonary embolism should be considered **(Oudkark., 1999).**

VIII. Iatrogenic transudative pleural effusion:

Misplacement of a central venous catheter into the pleural space or mediastinum can result in the infusion of intravenous fluid into the pleural space. The effusions are usually unilateral but may be bilateral when fluid is mistakenly infused into the mediastinum. This occurrence is suggested by the rapid accumulation of pleural fluid following placement of a central venous line and is confirmed by demonstrating pleural fluid glucose and protein concentrations equal to those of the infused fluid **(Connors and Altose., 1994).**

IX. Effusion Evacuee:

Pleural adhesions sometimes prevent the re-expansion of a lung following pneumothorax. The continued absorption of intrapleural gas then produces markedly subatmospheric pleural pressures that favor the formation and accumulation of a transudate **(Sahn., 1988).**

B. Exudative Pleural Effusions:

Exudative pleural effusions occur as a result of:

1. Increased permeability of the pleural capillaries to protein
2. Reduced re-absorption of fluid by the visceral pleura

3. Impaired removal of protein and fluid by the pleural lymphatics.
4. Abnormally subatmospheric pleural pressures due to local or generalized reduction of the lung compliance.

Exudative effusions are most commonly associated with inflammatory processes, infections and neoplastic involvement **(Connors and Altose., 1994).**

Causes:

I. Infections:

a. Bacterial, as empyema and parapneumonic effusions.

b. Tuberculous.

c. Viral.

d. Fungal.

e. Parasitic.

f. Mycoplasma.

g. Rickettsial.

II. Neoplasms:

a. Direct pleural involvement as in:

   1. Metastasis.

   2. Direct invasion from surrounding structures.

3. Primary pleural malignancy.

b. Indirect involvement as in:

1. Lymphoma.

2. Ovarian Neoplasm (Meig's Syndrome).

III. Thromboembolic disease:

a. Pulmonary embolism.

b. Pulmonary infarction.

IV. Immune mediated diseases:

a. Rheumatoid disease.

b. Systemic lupus erythematous.

c. Drug induced lupus.

d. Wegner's granulomatosis.

e. Sarcoidosis.

f. Post-cardiac injury syndrome.

g. Angioimmunoblastic lymphadenopathy.

h. Sjorgren's syndrome.

i. Progressive systemic sclerosis.

V. Intra-abdominal disorders:

a. Pancreatitis.

b. Oesophageal perforation.

c. Sub-phrenic abscess.

d. Intra-hepatic abscess.

e. Splenic abscess.

f. Oesophageal variceal scleropathy.

g. After abdominal surgery.

h. Post-partum.

VI. Drug Induced pleural disease:

As Nitrofurantoin, Methysergide, Dantrolene.

VII. Inhalation of organic dusts (Occupational):

As asbestos.

VIII. Other Causes:

a. Yellow nail syndrome

b. Uremic pleurisy

c. Radiation Pleuritis.

d. Myxoedema.

e. Spontaneous pneumothorax.

f. Familial Mediterranean fever.

g. Trapped lung.

It is noticed that pleural effusion due to pulmonary emboli, Sarcoidosis, myxoedema and Meig's syndrome can

be either transudates or exudates. In conclusion, the etiology of pleural effusion is so diverse and subject to so many factors, that needs to be thoroughly investigated to reach a proper diagnosis **(White et al., 2000)**.

1. Parapneumonic effusions:

About half of all bacterial pneumonias are associated with parapneumonic pleural effusions, which cause greater morbidity and mortality than pneumonia alone. Exudative parapneumonic effusions usually contain no bacteria and resolve promptly with treatment of the underlying pulmonary infection. Some so-called complicated parapneumonic effusions persist, become loculated and undergo organization and fibrosis. An empyema may result when the pleural fluid becomes infected and large numbers of polymorphnuclear leucocytes accumulate **(Light., I998)**.

Approximately, 60% of patients with pneumococcal pneumonia **(Lim and Yim., 1999)** and 40% of all bacterial pneumonias are associated with pleural effusion **(Davies and Glesson., 1998)**. Initially the effusion may be amber colored; containing predominantly polymorphs, but it may progress to increasing turbidity with a high white cell count. The effusion associated with bacterial pneumonia is initially sterile. However, it may frequently be invaded by the

causative bacteria, leading to empyema or eventual healing by fibrosis. Viral and mycoplasma pneumonia rarely cause effusion **(Sasse et al., 1999).**

Cultures for both aerobic and anaerobic bacteria will identify the responsible microorganism in about 40 percent of parapneumonic effusions (70 percent if fluid is grossly purulent) **(Porcel and Light., 2004).**

2. Tuberculous pleural effusions:

Extra-pulmonary tuberculosis (EPTB) can occur in isolation or along with a pulmonary focus as in the case of patients with disseminated tuberculosis. The human immunodeficiency virus (HIV) and acquired immunodeficiency syndrome pandemic has resulted in changing epidemiology and has once again brought EPTB into focus which constitutes about 15 to 20 percent of the cases of (TB) in immune-competent patients and accounts for more than 50% of the cases in HIV positive individuals. Lymph nodes are the most common site of involvement followed by the pleura **(Sharma and Mohan., 2004).**

The onset of the disease may be abrupt with severe pleural pain, cough, dyspnea and fever, but it usually begins more insidiously. Tuberculous effusions are usually unilateral and of small to moderate amount. In up to one half of cases, it is

accompanied by parenchymal infiltrates. Its presence can be easily detected by physical and/or roentgenographic examination **(Liam et al., 2000a).**

The diagnosis of pleural TB is often difficult and failure to diagnose and treat pleural TB can result in progressive disease with involvement of other organs in as many as 65% of patients **(Ghanei et al., 2004)**.

The diagnosis of tuberculous pleural effusion (TPE) depends on the presence of a positive tuberculin test or a lymphocyte rich exudates, the demonstration of tubercle bacilli in the sputum, pleural fluid or pleural biopsy specimens, or the demonstration of tuberculous granuloma in pleural biopsy specimens **(Morimoto et al., 2006).**

Withdrawal of fluid for diagnostic study is desirable, its appearance is usually clear to slight cloudy, it has specific gravity seldom outside the range of 1016 to 1022 and it frequently coagulates. The total protein concentration is somewhat elevated to approximately 5 gm. /100 ml or more. The lactate dehydrogenase levels are somewhat elevated around 535 IU/L or more. Glucose level determination in pleural fluid is variable and of little help but it has been described that tuberculous effusions contain low glucose content, below 50%. The white cell counts ranges from 1000-6000/ml and is

predominantly, lymphocytes. Mesothelial cells account for less than 5% of total cells, their absence on careful examination of the pleural fluid can be suggestive of tuberculous pleural infection. Similarly, greater than 10% eosinophils are rarely seen in untreated and uncomplicated tuberculous effusion **(Riantawan et al., 1999).**

Both pleural fluid and sputum should be cultured for mycobacteria when tuberculous pleurisy is suspected. The yield of sputum cultures in tuberculous pleural effusion varies from 10 to 60 percent, largely dependent on the extent of associated pulmonary involvement **(Davies., 2004).**

Because delayed hypersensitivity plays a major role in the pathogenesis of tuberculous pleurisy, it is not possible to isolate Mycobacterium tuberculosis from pleural fluid samples in more than 60 to 70 percent of patients **(Porcel and Vives., 2003).**

The use of broth medium (e.g., BACTEC radiometric system) with inoculation provides higher yields and faster results (one to two weeks) than conventional methods **(Hasaneen et al., 2003).**

Smears of the pleural fluid for mycobacteria are rarely positive (5 percent) unless the patient has a tuberculous