Evaluation of Medical Thoracoscopy in Diagnosis of Unidentified Exudative Pleural Effusion in Abbassia Chest Hospital from January 2010 to June 2012

Thesis

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قَالُوا سُبْحَانَكَ لاَ عِلْمَ لَنَا إِلاَّ مَا عَلَّمْتَنَا إِنَّكَ أَنتَ الْعَلِيمُ الْحَكِيمُ

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Hisham Mabrouk Abd Elnabi

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List of Abbreviations

ANA	:	Antinuclear antibodies.
cm	:	Centimeter.
CT	:	Computerized tomography.
Dr.	:	Doctor.
ECG	:	Electrocardiogram.
EPTB	:	extra-pulmonary tuberculosis.
FDG	:	Fluorodeoxyglucose.
FEV_1	:	Forced expiratory volume in the first second.
gm	:	Gram.
HS	:	Highly significant.
HIV	:	Human immunodeficiency virus.
INR	:	International normalized ratio.
IU/L	:	International unit/liter.
Kg	:	kilogram.
kPa	:	kilopascal.
LDH	:	Lactate dehydrogenase.
LE	:	Lupus erythematosus.
mg/dl	:	Milligram/deciliter.
µm	:	Micrometer
ml	:	Milliliter.
mm	:	Millimeter.
MPE	:	Malignant pleural effusion.
MRI	:	Magnetic resonance imaging.

List of Abbreviations (Cont.)

NS	:	Non significant.
NSCLC	:	Non small cell lung cancer.
PET	:	Positron emission tomography.
pH	:	Negative logarithm hydrogen ion.
PMN	:	Polymorph nuclear leucocytes.
S	:	Significant.
SD	:	Standard deviation.
SLE	:	Systemic lupus erythematosus.
SPSS	:	Statistical package of the social science.
TB	:	Tuberculosis.
TPE	:	Tuberculous pleural effusion.
US	:	Ultrasound.
VATS	:	Video assisted thoracoscopic surgery.
VEGF	:	Vascular endothelial growth factor.
WHO	:	World health organization.

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Introduction

Pleural effusion is an accumulation of fluid in the pleural space, as a result of excessive transudation or exudation from the Pleural surface ***(Peek et al., 2000).***

The differential diagnosis of pleural diseases is often a lengthy process fraught with pitfalls. When a pleural effusion is diagnosed in a patient, the need for a timely and systematic evaluation is indicated ***(Porcel and Vives, 2003).***

Thoracoscopy has been the procedure of choice in various chest diseases such as: undiagnosed pleural effusions; recurrent, post traumatic or complicated spontaneous pneumothorax; it is also especially indicated for accurate staging of lung cancer with pleural spread and for primary pleural malignancies **(*Petrakis et al., 2000)***.

Thoracoscopy allows visualization of the pleural cavity including the diaphragmatic, visceral pleura as well as the lung. The procedure does not only give information on the extent of the disease itself but also allows adequate tissue biopsy sampling which helps to distinguish between viable tumors and other lesions as for example fibrotic reaction ***(Prosst et al., 2002).***

Aim of the Work

The aim of this study this study is to detect the diagnostic yield of medical thoracoscopy in the diagnosis of cases of exudative pleural effusions of unidentified aetiology at Abbassia Chest hospital retrospectively from January 2010 to November 2011 and prospectively from December 2011 to June 2012.

Anatomy of the Pleura

The pleura is a thin, glistening, slippery serous membrane. Embryologicaly, the pleural membrane is developed from mesenchyme ***(Gray and Skandalakis, 1985)***.

The pleura lines the thoracic wall and diaphragm, where it is known as the parietal pleura. It is reflected onto the lung, where it is called the visceral pleura. The visceral pleura envelopes and invests the entire surface of the lungs, mediastinum and diaphragm. At the lower border of the hilum, the pleural reflections from the dorsal and ventral surface of the lung usually extend to the diaphragm as a double layer of mesothelial tissue; the pulmonary ligament ***(Johnston and Green, 1983).***

The facing surfaces of the parietal and visceral pleura slide smoothly against each other during respiration. The contact between the parietal and visceral pleura depends on the atmospheric pressure on the outside of the chest wall and inside the alveoli (which are connected to the exterior by the bronchial tree). On the other hand, the two pleural layers tend to be separated by the elasticity of the thoracic wall (directed outward) and the lungs (stretched by inspiration). The pleural membranes facilitate the movement of the lungs within the thorax during breathing. Another function is coupling the lungs to the chest wall ***(Sheldon et al., 1981).***

- ## Blood Supply of the Pleura:

The parietal pleura receives its blood supply from the systemic capillaries. Small branches of the intercostal arteries supply the costal pleura, whereas the mediastinal pleura is supplied principally by the pericardiacophrenic artery. The diaphragmatic pleura is supplied by the superior phrenic and

musculophrenic arteries. The venous drainage of the parietal pleura is primarily by the intercostal veins, which empty into the inferior vena cava or the brachiocephalic trunk. The venous drainage of the diaphragm is either caudally into the inferior vena cava through the inferior phrenic veins, or cranially into the superior vena cava through the superior phrenic veins ***(Peng and Wang, 2003)***.

All investigators agree that the bronchial artery supplies most of the visceral pleura facing the mediastinum, the pleura covering the interlobular surfaces, and a part of the diaphragmatic surface. The blood supply for the remaining portions of the visceral pleura is less understood and is thought by some to be through the pulmonary artery ***(Peng and Wang, 2003)***.

The venous drainage of the visceral pleura is through the pulmonary veins ***(Light, 2007).***

- **Pleural Lymphatics:**

The lymphatic plexuses in the costal pleura are mainly confined to the intercostal spaces and are absent or minimal over the ribs ***(Peng and Wang, 2003).***

The lymphatic vessels of the costal pleura drain ventrally toward nodes along the internal thoracic artery and dorsally toward the internal intercostal lymph nodes near the heads of the ribs. The lymphatic vessels of the mediastinal pleura pass to the tracheobronchial and mediastinal nodes, whereas the lymphatic vessels of the diaphragmatic pleura pass to the parasternal, middle phrenic, and posterior mediastinal nodes ***(Parungo et al., 2005).***

The visceral pleura has extensive lymphatics, but they do not connect to the pleural space ***(Albertine et al., 1982).***

The parietal pleural lymphatics connect to the pleural space via stomas, holes of 8 to 10 μm in diameter that are formed by discontinuities in the mesothelial layer where mesothelium joins to the underlying lymphatic endothelium ***(Li and Li, 2003).***

The stomas can accommodate particles as large as erythrocytes. In various experimental studies, these lymphatics have been shown to be the major route of exit of liquid from the pleural space ***(Broaddus et al., 1988).*** From the stomas, liquid drains to lacunae, spider like submesothelial collecting lymphatics, which then drain to infracostal lymphatics, to parasternal and periaortic nodes, to the thoracic duct, and into the systemic venous system. Lymphoid cells have been described lying within aggregates underneath morphologically different mesothelial cells, forming raised structures called *Kampmeier's foci* that may have an immune function ***(Pereira et al., 1994).***

- **Nerve Supply of the Pleura:**

Only the parietal pleura contains sensory nerve fibers, supplied by the intercostal and phrenic nerves. The costal and peripheral diaphragmatic regions are innervated by the intercostal nerves, and pain from these regions is referred to the adjacent chest wall. The central diaphragmatic region is innervated by the phrenic nerve, and pain from this region is referred to the ipsilateral shoulder. The visceral pleura does not contain sensory nerve fibers; therefore, pain, whether from inflammation, tumor, or catheters advanced far out in the lung during bronchoscopy, indicates involvement of the adjacent parietal pleura ***(Broaddus and Light, 2005).***

Physiology of the Pleural Fluid

The pleural space and the fluid within it are under dynamic conditions. Pleural fluid constantly moves into and out of the pleural space. In normal subjects, a dynamic equilibrium is reached when the rates of the fluid entry and efflux are about equal, the volume of the pleural fluid remains virtually constant ***(Light, 2007).***

Normally the composition of the thin layer of pleural fluid between the parietal and visceral pleura is that of an ultra filtrate of plasma.

Table (1): Normal composition of pleural fluid

Volume	0.1-0.2 ml/kg
Cells/mm^2	1000-5000
% mesothelial cells	3-70%
% monocytes	30-75%
% lymphocytes	2-30%
% granulocytes	10%
Protein	1-2 gm/dl
% albumin	50-70% of plasma level
Glucose	= plasma level
LDH	<50% of plasma level

The volume of fluid normally present in the pleural space is small (5 to 15 ml). The rate of turnover of pleural fluid in humans is rapid.

The movement of fluid between the pleural capillaries and the pleural space is believed to be governed by Starling's law of transcapillary exchange ***(Parameswaran et al., 1999).***

- ## Pathogenesis of Pleural Effusion*:*

Pleural fluid accumulates when the rate of pleural fluid formation exceeds the rate of pleural fluid absorption. Normally, a small amount (0.01 ml/kg/hour) of fluid constantly

enters the pleural space from the capillaries in the parietal pleura. Almost all of this fluid is removed by the lymphatics in the parietal pleura, which have a capacity to remove at least 0.20ml/kg/hour ***(Light, 2001a).***

1- Increased pleural fluid formation:

Increased pleural fluid formation can occur when there is increased pulmonary interstitial fluid or where one of the terms in Starling's equation is changed such that more fluid is formed ***(Light, 2001b).***

2- Increased interstitial fluid:

The most common cause of increased pleural fluid formation is increased interstitial fluid in the lung. Whenever the amount of oedema in the lung exceeds 5 gm per gram of dry lung weight, pleural fluid accumulates whether the oedema is high protein or low protein. This appears to be the predominant mechanism for the formation of pleural effusions in patients with congestive heart failure, parapneumonic effusions, the acute respiratory distress syndrome, and lung transplantation ***(Sadikot et al., 2000).***

3- Increased hydrostatic pressure gradient:

If there is an increase in the gradient between the intravascular pressure and the pleural pressure, there will be an increase in the rate of pleural fluid formation through Starling's equation. Increases in the intravascular pressure can occur with right ventricular failure, left ventricular failure, pericardial effusion, or the superior vena cava syndrome. The most common situation producing a decrease in the pleural pressure is bronchial obstruction leading to atelectasis of a lower lobe or complete lung. A decrease in the pleural pressure also occurs when the visceral pleura becomes coated with a collagenous peel and the lung becomes trapped. In these