Prevalence, Causes and Clinical Implications of Pleural Effusion in Pulmonary ICU and Correlation with Patient Outcomes

Thesis submitted for partial fulfillment of the MD degree in Chest Diseases

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2018

Acknowledgement

Thanks first and last to ***Allah*** as we owe him for his great care, support and guidance in every step in our life.

I would like to express my cordial appreciation and infinite gratitude to ***Prof. Mohammed Ali Farrag***, Professor of Chest Diseases, Faculty of Medicine, Ain Shams University, who brought this work to light and guide me all through the way. Without his constant support, this thesis would not have ended successfully. He provided valuable and constructive criticism and was continuously reviewing my work till the end.

I would like to express my deep thanks and gratitude to ***Dr. Ahmed Abd El Gawad El Masry,*** Assistant Professor of Chest diseases, Faculty of Medicine, Ain Shams University for his constant support which has been of greatest value in accomplishing this work.

I would extend my thanks to ***Dr. Amr Mounir Shoukri*** Assistant Professor of Chest Diseases, Ain Shams University for his help & support all through the time of the research.

Lastly, I would like to express my deep thanks to all the staff of Abbaseia Chest Hospital; doctors and nurses for their great efforts to help me to finish this research.

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List of abbreviations

Abbreviation	Detail
2D	Two-dimensional
AAFB	Alcohol, acid fast bacilli
ADA	Adenosine deaminase
AF	Atrial fibrillation
ANA	Antinuclear antibodies
ARDS	Adult respiratory distress syndrome
CAPD	Continuous ambulatory peritoneal dialysis
CBC	Complete blood count
CHF	Congestive heart failure
Cm	Centimeter
COPD	Chronic obstructive pulmonary disease
CT	Computerized tomography
CVS	Cerebrovascular stroke
CXR	Chest x ray
dl	Deciliter
DM	Diabetes mellitus
DNA	Deoxy ribonucleic acid

E. coli	Escherichia coli
ECHO	Echocardiography
EPTB	Extra pulmonary tuberculosis
ESR	Erythrocyte sedimentation rate
FOB	Fiber optic bronchoscopy
gm	Gram
HF	Heart failure
HIV	Human immunodeficiency virus
HLAB8	Human leucocytic antigen B8
ICM	Ischemic cardiomyopathy
ICU	Intensive care unit
IHD	Ischemic heart disease
IU	International unit
Kg	Kilogram
LDH	Lactate dehydrogenase
L^p	hydraulic conductivity
MHz	Megahertz
MICU	Medical intensive care unit
ml	Milliliter
MPE	Malignant pleural effusion
MRI	Magnetic resonance imaging
MRSA	Methicillin resistant staphylococcus aureus

NSAIDs	Non steroidal anti inflammatory drugs
OHVS	Obesity hypoventilation Syndrome
PA film	Postero-anterior film
PCR	polymerase chain reaction
PE	Pulmonary embolism
PNL	Polymorph nuclear leukocytes
PPPE	Parapneumonic pleural effusion
RA	Rheumatoid arthritis
S. aureus	Staphylococcus aureus
SLE	Systemic lupus erythematosis
TB	Tuberculosis
TST	Tuberculin skin test
U/S	Ultrasound
VEGF	Vascular endothelial growth factor

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Introduction

Pleural effusion is common in critically ill patients. Most effusions in intensive care unit (ICU) patients are of limited clinical significance; however, some are important and require aggressive management **(David et al., 2011).**

Medical ICU patients appear to be at risk for developing pleural effusion; as many patients present with hypotension and hemodynamic instability and are treated with aggressive hydration leading to fluid overload resulting in transudative effusions that are often bilateral even in the absence of heart failure **(Sahn et al., 1995).** Importantly, mechanical ventilation leads to areas of atelectasis that may be associated with pleural effusions. Sedation, paralysis, acute lung injury, and limited mobility can lead to the collapse of basal lung segments **(Zocchi 2002).**

Transudative effusions in ICU are commonly caused by volume overload, decreased plasma oncotic pressure, and regions of altered pleural pressure attributable to atelectasis and mechanical ventilation. Exudates could be sequelae of pulmonary or pleural infection, pulmonary embolism, postsurgical complications, and malignancy **(David et al., 2011).**

Pleural effusions present in ICU patients are frequently uncomplicated. Empyema is relatively uncommon, but when it is encountered, it appears to be resistant to antibiotic treatment alone in the ICU **(Fartoukh et al., 2002).**

When clinical suspicion for infection is low, observation of pleural effusions in the ICU is warranted initially, because most

are caused by noninfectious processes that should improve with treatment of the underlying disease **(Lalaine et al., 1997).**

Mechanically ventilated patients with pleural effusions should be semirecumbant and treated with higher levels of positive-end expiratory pressure **(David et al., 2011).** Detection of small pleural effusions is problematic in these patients, because chest radiographs are usually obtained with the patient in the supine or semi recumbent position and a pleural fluid volume of <500 ml may produce only a subtle increased haziness over the lower lung zone **(Wiener et at., 1991).**

Chest ultrasonography is a portable, low-cost, radiation-free method, showed consistently high sensitivity, specificity and accuracy in detecting fluid in the pleural space, in different populations and clinical settings **(Alexandre et al., 2010).**

Aim of the work

The aim of this study is to determine the prevalence, causes and clinical significance of pleural effusion in critically ill patients and to compare different methods of assessment and management, and finally to correlate all data with patient outcomes.

Anatomy of the pleura

The pleura is made of two serosal membranes, one covering the lung (visceral pleura) and one covering the inner chest wall (parietal pleura). Their surfaces glide over each other, facilitating proper lung movements during the various phases of respiration **(Bertin and Deslauriers, 2011).**

The visceral pleura covers the lung parenchyma, not only at its points of contact with the chest wall, diaphragm, and mediastinum but also in the inter-lobar fissures. The parietal pleura lines the inside of the thoracic cavity. It is subdivided into the costal, mediastinal, and diaphragmatic parietal pleura. The visceral and the parietal pleura meet at the lung root. At the pulmonary hilus, the mediastinal pleura is swept laterally onto the root of the lung. Posterior to the lung root, the pleura is carried downward as a thin double fold called the pulmonary ligament **(Light, 2007).**

A film of fluid (pleural fluid) is normally present between the parietal and the visceral pleura. This thin layer of fluid acts as lubricant and allows the visceral pleura covering the lung to slide along the parietal pleura lining the thoracic cavity during respiratory movements. The potential space, between the two layers of pleura is designated as the pleural space. It is a potential space rather than an actual one **(Wang, 1998).**

Both pleural surfaces are lined by a squamous epithelial layer, often called mesothelium, whose surface is richly endowed with long microvilli **(Ochs and Weibel, 2008).**

The pleura is a monolayer of mesothelial cells which rest on a matrix of collagen, elastic fibers, blood vessels, and lymphatics, which allow the lung and chest to expand and contract, protected from friction by the pleural fluid and

properties of the mesothelial cells. With a rich blood supply and lymphatic system just deep to the mesothelial layer, the pleura is a dynamic layer protecting the lung and pleural cavity from infection while transmitting the forces of respiration without damage to the underlying lung parenchyma **(Finley and Rusch ,2011)**.

Mesothelial cells are metabolically active and appear to have several functions. They are likely to be responsible, at least in part, for regulating the composition and amount of pleural fluid and there is evidence that they have the ability to produce components of the sub-mesothelial connective tissue. Surface-active phospholipids similar to alveolar surfactant, most likely produced by mesothelial cells, have been found in the pleural space, where they may act as lubricants to facilitate pleural surface movement **(Fraser, 2005)**.

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 pericardiophrenic 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 **(Peng and Wang, 2003)**.

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