The Effects of Pleural Effusion

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Introduction

In healthy subjects, the pleural space, which is delimited by the visceral and parietal pleura, contains only a small amount of fluid, ranging from 10 to 20 ml [1]. This liquid usually originates from the capillaries of the parietal pleura and is drained by the lymphatics of the parietal pleura. However, in ill patients the fluid can originate from the visceral pleura or directly from the peritoneal cavity through the diaphragm. Basically, liquid accumulates every time there is an excess in liquid formation or a reduction in drainage. Medical or surgical patients are rarely admitted to the intensive care unit (ICU) for primary pleural disease; however, the pleura can be affected by various pulmonary or extrapulmonary conditions that promote the development of pleural effusions, which can affect the respiratory system [2].

The incidence of pleural effusion is estimated at 1 million cases in the United States per year [3]. The incidence of parapneumonic effusions among individuals with pneumonia ranges from 20 to 97% [4–7], while in decompensated congestive heart failure the incidence may be as high as 87% [8]. In a series of medical ICU patients, the incidence of pleural effusion was 8.4% per year [9]. In a group of one hundred consecutive patients admitted to medical intensive care, 41 had pleural effusion at admission and an additional 21 patients developed an effusion during their ICU stay [2]. The mean age of patients with pleural effusion was 54 ± 2 years and of those without effusion was 47 ± 2 years. The albumin concentration in the group with pleural effusions was less (2.4 ± 0.1 g/dl) than that in patients without effusion (3.0 ± 0.1 g/dl) and those with effusions had longer ICU stays (9.8 ± 1.0 vs 4.6 ± 0.7 days) and duration of mechanical ventilation (7.0 ± 1.3 vs 1.9 ± 0.7 days).

Pathogenesis

Pleural effusions can originate from pulmonary or extrapulmonary conditions, including lung infections, tumors, congestive heart failure, hypoalbuminemia, atelectasis, sepsis, and acute kidney injury [10]. The altered pleural liquid turnover leads to a pleural effusion when filtration exceeds absorption or when one of the important absorbing systems is primarily altered and the compensatory mechanisms cannot maintain the homeostasis. The formation of pleural effusions may be due to several causes that act by modifying the pressure mounted by the pleura, by damaging the lymphatic drainage system, or by increasing the permeability of membranes in the capillary endothelium and mesothelium. The result is an increase in fluid in
the pleural cavity with or without an increase in protein concentration, a condition which allows the effusion to be classified as a transudate or an exudate. Transudates are defined as the liquid that accumulates because of increased permeability of capillaries and the mesothelium, which alters the filtration rate of the pleura; it may also accumulate in absence of altered pleural liquid turnover by entering the pleural space through a non-physiological means, such as pleuro-peritoneal communication during peritoneal dialysis or presence of ascitic fluid driven by the pressure gradient between the abdomen and the pleural space. Exudates, according to Light’s criteria, are primarily generated by an impairment in lymphatic drainage and caused, for example, by tumors, infections and gastrointestinal diseases. An exudative effusion is defined by a ratio between pleural fluid proteins and plasma proteins of greater than 0.5, a ratio of lactate dehydrogenase in the pleural fluid and plasma greater than 0.6, or by a lactate dehydrogenase content of the pleural fluid equal to 2/3 of the value contained in normal serum [11]. Transudative effusions are usually related to congestive heart failure, hepatic hydrothorax, or nephrotic syndrome. Exudative effusions may be due to infection, e.g., empyema or parapneumonic effusion, or non-infectious conditions, e.g., surgery or intra-abdominal malignancy [12].

In a report from a population of critically ill patients, non-infectious causes of pleural effusion were present in 82% of patients; heart failure was the leading cause of all effusions (35%) and atelectasis was the second cause (23%) [2]. The most common cause of bilateral effusions was again heart failure (38%), while atelectasis was the most frequent cause of unilateral effusion (36%) [2]. Effusions due to hypoalbuminemia were reported in only 8% of the patients and were caused by severe malnutrition. Roch et al. reported that pleural effusion was predominantly parapneumonic (41%) and/or consecutive to heart failure in 20% of cases, to hypoalbuminemia in 18%, and to peritonitis in 11% [13].

Effects of Acute Pleural Effusion on Lung Volume, Respiratory System Mechanics, and Gas Exchange

Animal studies, in which fluid was introduced into the pleural space, demonstrated a gravitationally oriented gradient of pleural pressure of about 1 cmH$_2$O per cm of height [14]. This increase in pleural pressure gradient should promote lung atelectasis and airway closure.

Krell and Rodarte studied the effect of acute pleural effusion on the lung and chest wall in six head-up anesthetized dogs [15]. The functional residual capacity (FRC) was measured by a helium dilution technique, while total lung capacity was defined as the volume at an airway pressure of 30 cmH$_2$O. Mean effusion volumes were 9, 25 and 45% of the total lung capacity and were introduced into the thorax via a polyethylene catheter in the right pleural space. An esophageal balloon positioned in the lower half of the esophagus, approximately at the level of the heart, was used to estimate the pleural pressure. More than 90% of the added saline was recovered from the dogs’ thoracic cavities, indicating that no significant absorption of saline occurred during the study. Both FRC and total lung capacity decreased with increasing amounts of saline added to the pleural space. The decrease in FRC was about one third the amount of saline added; the other two thirds of the saline volume must, therefore, have increased the chest wall volume. A similar volume change was observed at total lung capacity although the decrease in gas lung volume was slightly less than one third the added saline volume. The pleural effusion did not