



Bacterial Pleura and Pulmonary Interstitial Infections

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Abstract

The visceral and parietal pleurae are continuous with one another at the root of the lung, where the hilar airways and vessels enter the lung parenchyma, and are closely opposed to the individual pulmonary lobes, the inner aspect of the thoracic cage, and the lateral margin of the mediastina. The resultant pleura space contains scant fluid and is normally a potential space that becomes a true space only in disease states that cause accumulation of pleura fluid. The visceral pleura is attached to the lung surface and is contiguous with the sub-pleura pulmonary interstitials. It is thick and apparently derives its blood supply from both pulmonary and systemic arteries, draining to the pulmonary veins. The visceral pleura individually invest pulmonary lobes.

Keywords: Traumatic and iatrogenic; Pleura effusions; Clinical expression; Defence responses; Pleura fluid; Antibiotics

Introduction

The interlobar fissures seen radio-graphical or by computed tomography are due to the additive thickness of the visceral pleura layers of the participating lobes. The normal pleura fluid volume is negligible and invisible by imaging. The major, or oblique, fissure separates the lower lobe from the upper lobe of the left lung. The minor, or horizontal, fissure separates the right middle lobe from the upper lobe. The parietal pleura are composed of four layers but are slightly thinner than the visceral pleura. It is surrounded by a thin layer of extra pleura or subcostal fat, which is surrounded by the fibro elastic endothoracic fascia that constitutes the boundary of the thoracic cavity [1]. The endothoracic fascia is attached to the perichondrium of the costal cartilage, the ribs and inter-costal muscles, and the pre vertebral fascia surrounding the vertebral bodies and intervertebral disks. The extra-pleura fat layer is normally thick but may become radiological detectable in normal patients. It increases diffusely in the presence of empyema, but not in obese patients. The parietal pleura is supplied and drained by systemic vessels. The lymph of the pleura space is drained by stoma in the parietal pleura, which represents the predominant-if not exclusive- mechanism by which liquid is cleared from the pleura space [2]. The parietal pleura have abundant sensory innervation and should be well anesthetized before it is manipulated or punctured. Although the quantity of pleura fluid is small, it efficiently couples the lung to the diaphragm and chest wall during breathing and lubricates the movement of those structures.

Methodology

Nevertheless, little or no functional impairment results when the pleura space is obliterated either experimentally or because of clinical necessity [3]. Anatomic anomalies of the pleura are rarely of clinical consequence but can cause confusing radiological patterns. Accessory fissures are very frequently encountered at surgery or post mortem, but only two types are commonly encountered in practice. The inferior accessory fissure separates the medial basal segment of the right lower lobe from the other basal segments of the lower lobe. Such fissures occur in people, usually in incomplete forms invagination the lower lobe at its diaphragmatic aspect. Superior accessory fissures are present in patients [4]. This variant fissures are roughly horizontal and separate the superior segment of a lower lobe from the basal segments of that lobe. They may mimic a horizontal fissure on a chest radiograph. Pleura effusions develop because of increased hydrostatic pressure or decreased oncotic pressure associated with cardiac, renal, hepatic, or metabolic disease.

Other factors contributing to their development include alterations in pleura permeability due to non-infectious inflammatory diseases, infection, toxic injury, malignancy, or trauma [5]. The pleura space is normally sterile yet readily colonized once pleura fluid has accumulated. Host factors predisposing patients to empyema include pneumonia and para-pneumonic effusions as well as contiguous infections of the oesophagus, mediastina, or sub-diaphragmatic areas that may extend to the pleura as shown in (Figure 1). Both traumatic and iatrogenic injury to adjacent structures may lead to secondary infection and involvement of the pleura [6]. Similarly, retropharyngeal, retroperitoneal, vertebral, or paravertebral infection can extend to the pleura. Pleura effusions are nutritionally rich culture media in which WBC defences are severely impaired. The classic studies of Wood and co-workers showed that effective phagocytosis of bacteria by neutrophils requires a structure upon which white blood cells can move and can ingest bacteria prior to development of specific antibodies [7]. Later in the course of infection, phagocytosis is enhanced by antibodies and opsonic factors. However,



Figure 1: Predisposing patients to empyema include pneumonia and para-pneumonic effusions.

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Received: 28-Aug-2023, Manuscript No. JRM-23-115751; **Editor assigned:** 30-Aug-2023, PreQC No. JRM-23-115751 (PQ); **Reviewed:** 14-Sep-2023, QC No. JRM-23-115751; **Revised:** 20-Sep-2023, Manuscript No. JRM-23-115751 (R); **Published:** 27-Sep-2023, DOI: 10.4172/jrm.1000183

Citation: Saunders M (2023) Bacterial Pleura and Pulmonary Interstitial Infections. J Respir Med 5: 183.

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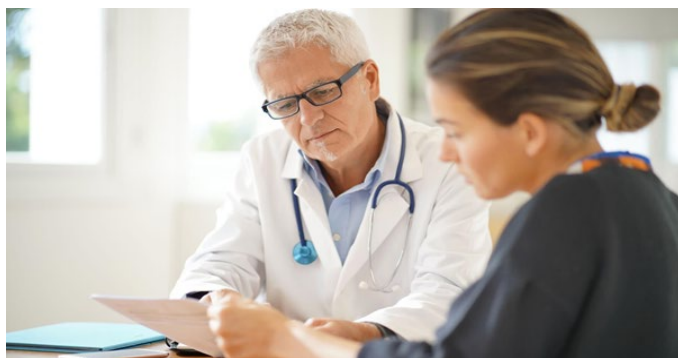


Figure 2: Growth rates of bacteria to lactam antibiotics incompletely understood.

in a fluid filled environment, bacteria can float away from phagocytic cells and multiply relatively unimpeded. In current parlance this defect reflects the fact that white cells can't jump and thus cannot efficiently fulfil their host defence function in a liquid medium, whether in the infected pleura, pericardium, joint, or meninges [8].

The formation of an empyema has been arbitrarily divided into an exudative phase, during which pus accumulates, a fibro-purulent phase, during which fibrin deposition and flocculation of pleura exudate occurs, and an organization phase, during which fibroblast proliferation and scar formation cause lung entrapment.

Discussion

Prompt diagnosis and intervention should circumvent the second and third phases of empyema formation [9]. To achieve this goal, physicians need to appreciate the subtleties of clinical expression of pleura empyema and the adverse effects of the supportive environment on antimicrobial efficacy and tissue injury in the pleura space [10]. Bacteria in pleura fluid elicit a complex series of host defence responses that are incompletely understood despite significant recent advances, the cytokine cascade, and perturbations of endothelial cell and leukocyte interactions during infection. When the inflammatory response is too little or too late, bacteria may multiply until they reach a stagnant growth phase, associated with concentrations of bacteria [11]. Empyema fluid is relatively deficient in opsonins and complement and becomes progressively more acidic, hypoxic, and depleted of glucose as infection proceeds. Gram negative aerobic bacilli may release endotoxins, and streptococci or staphylococci may release enzymes that lyse granulocytes in pleura fluid. During the inflammatory process, leukocytes release intracellular constituents such as bactericidal permeability increasing protein, defensins, lysozyme, cationic proteins, lactoferrin, and zinc-binding proteins [12]. The latter two components may contribute to suppression of bacterial growth by lowering concentrations of iron and zinc. Pneumococci and perhaps other organisms may undergo autolysis in overtly purulent empyema fluid, thus accounting for a portion of rate of sterility of empyema fluid. Late in the course of infection, the inflammatory response leads to loculation and occasionally to its spontaneous drainage by erosion through the chest wall. Bacteria within empyema are relatively unresponsive to antibiotics. In that milieu bacteria may release, lactamase enzymes capable of degrading, lactamase susceptible, lactam antibiotics. Similarly, microbial enzymes in pus may degrade chloramphenicol [13]. Overtly purulent empyema fluid may be quite acidic, even in the absence of oesophageal rupture. Since aminoglycoside incorporation by bacteria is ordinarily oxygen-dependent and acid-inhabitable, aminoglycoside efficacy is suppressed in the hypoxic and acidic milieu of pleura empyema. Furthermore,

the calcium and magnesium concentrations in pus, the avid binding of aminoglycosides, and the reduced bacterial metabolism in pus may inhibit aminoglycoside activity in empyema fluid. Bacteria within abscesses or involved in chronic inflammatory states multiply slowly, with generation times that may reach few hours [14]. Tuomanen and co-workers found that there was a direct relationship between the multiplication rate of *Escherichia coli* and their death rate after exposure to cephalosporin in vitro rapidly multiplying organisms were killed quickly, whereas slowly growing organisms were killed less rapidly in proportion to their growth rate. When killing curves were expressed in relationship to the doubling time of the bacteria exposed to antibiotics, there was a linear relationship between cell division and the rate at which bacteria were killed by, lactam agents. The mechanisms by which growth rates of bacteria modify their susceptibility to, lactam antibiotics are incompletely understood as shown in (Figure 2). Stevens and colleagues demonstrated a progressive reduction of penicillin binding proteins in streptococci as they entered a stagnant phase of growth. It appears likely that the rate of bacterial division affects the quantity and type of penicillin binding proteins that are available to interact with, lactam antibiotics [15]. This may in part explain why bacteria in pus are refractory to antibiotics and why it is necessary to give prolonged antibiotic therapy to patients with poorly drained, supportive infections. Prolonged therapy may be needed because slowly growing organisms in pus require prolonged contact with, lactam antibiotic in order to induce sufficient cell wall injury to kill bacteria. Fortunately, this impediment can be circumvented by abscess drainage, which removes large numbers of metabolically inert bacteria and their toxins and removes inflammatory components of the empyema milieu that are capable of suppressing bacterial responsiveness to antibiotics and injuring host tissues.

Conclusion

Thus, pressure models in man fall short in explaining the existence of a constant physiological amount of fluid in the pleura cavity and also, they do not give a clue as to the further fate of the fluid. In other words, volume control mechanisms need to be involved in addition to pressures.

Acknowledgement

None

Conflict of Interest

None

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