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Review

Management of parapneumonic pleural effusion in adults☆

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ABSTRACT

Pleural infections have high morbidity and mortality, and their incidence in all age groups is growing worldwide. Not all infectious effusions are parapneumonic and, in such cases, the organisms found in the pleural space are not the same as those observed in lung parenchyma infections. The diagnostic difficulty lies in knowing whether an infectious effusion will evolve into a complicated effusion/empyema, as the diagnostic methods used for this purpose provide poor results.

The mainstays of treatment are to establish an early diagnosis and to commence an antibiotic regimen and chest drain as soon as possible. This should preferably be carried out with fine tubes, due to certain morphological, bacteriological and biochemical characteristics of the pleural fluid. Fluid analysis, particularly pH, is the most reliable method for assessing evolution. In a subgroup of patients, fibrinolytics may help to improve recovery, and their combination with DNase has been found to obtain better results. If medical treatment fails and surgery is required, video-assisted thoracoscopic surgery (VATS) is, at least, comparable to decortication by thoracotomy, so should only undertaken if previous techniques have failed.

Further clinical trials are needed to analyze factors that could affect the results obtained, in order to define new evidence-based diagnostic and therapeutic strategies that provide more effective, standardized management of this disease.

Manejo del derrame pleural paraneumónico en adultos

Palabras clave:
Derrame pleural paraneumónico
Derrame pleural paraneumónico complicado
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Drenaje torácico
Análisis del líquido pleural
Cultivo del líquido pleural
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Toracoscopia

RESUMEN

Las infecciones pleurales presentan una elevada morbimortalidad, y su incidencia está aumentando en todos los países del mundo y en todos los grupos de edad. No todos los derrames infecciosos son paraneumónicos, y en esos casos, los organismos que se encuentran en el espacio pleural no son los mismos que se observan en las infecciones del parénquima pulmonar. La dificultad diagnóstica radica en saber si un derrame infeccioso evolucionará hacia un derrame complicado/empiema, ya que los métodos diagnósticos utilizados con este fin ofrecen pobres resultados.

Los pilares del tratamiento son establecer un diagnóstico precoz e instaurar, lo antes posible, una pauta antibiótica y un drenaje torácico. Este se llevará a cabo, preferiblemente con tubos de pequeño calibre, ante la presencia de determinadas características morfológicas, bacteriológicas y bioquímicas del líquido pleural. El análisis del líquido es el método más fiable para valorar su evolución, sobre todo la determinación del pH. En un subgrupo de pacientes los fibrinolíticos pueden contribuir a mejorar la recuperación, y su combinación con deoxirribonucleasa se relaciona con la obtención de mejores resultados. Si fracasa el tratamiento médico y es necesaria la cirugía, la rentabilidad de la cirugía toracoscópica videoasistida es, al menos, comparable a la decorticación por toracotomía, por lo que esta solamente se realizará si han fallado las técnicas anteriores.

Abreviaturas: AUC, área bajo la curva; IL, interleucina.

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Son necesarios más ensayos clínicos que analicen factores que puedan influir sobre los resultados obtenidos para conformar nuevas estrategias diagnósticas y terapéuticas basadas en la evidencia, que proporcionen un manejo más efectivo y estandarizado de esta enfermedad.

Introduction

Parapneumonic pleural effusion (PPE) refers to a pleural effusion (PE) associated with bacterial pneumonia, a pulmonary abscess, or infected bronchiectasis.[1] When not accompanied by parenchymal disease, it is known as a pleural infection (PI) or complicated PE (CPE). Although most PPEs can be resolved with antibiotic treatment alone, a subgroup of patients can present severe complications, such as: PPE refractory to antibiotic treatment and chest drainage (CD), requiring surgical drainage (33%)[2]; pleural fibrosis (14%); prolonged hospital stay (mean 12–15 days and >1 month in 25% of cases)[2,4,5]; and high mortality rates (10–20%).[2,4,6] In order to avoid these, a firm diagnosis must be reached as soon as possible, and close clinical monitoring established.

Epidemiology

Of the one million patients hospitalized annually in the United States for pneumonia, around 60,000 will present empyema, and a further 25,000 will develop it for other reasons.[7] Despite clinical advances, the incidence of PI, irrespective of its stage of development, is increasing worldwide[5,8] across all age groups.[9–11] In the United States, the rate of hospitalization for empyema doubled from 1996 to 2008 (from 3.04 to 5.98/100,000 population), across all age groups.[12]

Physiopathology

There are 3 phases in the evolution of PPE. In the first (exudative) phase, pleural fluid (PF) forms as a consequence of localized inflammation and activation of the immune system. The activated neutrophils can cause an endothelial lesion, which will increase capillary permeability and lead to onset of PE.[13,14] The pleural damage can lead to commencement of the second phase (fibrinopurulent), with the activation of pro-inflammatory and profibrinotic components, and initiation of the coagulation cascade. The local inflammatory response is thus amplified by the presence of various cytokines, which stimulate chemotaxis of neutrophils and fibroblasts.[14] Furthermore, membrane permeability can allow bacteria to enter the pleural space. In the latter stages of this phase, PF resembling empyema may be observed due to the presence of cellular degradation products and bacterial remains. During this period, there is also a decrease in fibrinolysis, thereby increasing fibrin formation in the pleural space. This process can progress to the final (organizing) phase, in which a layer of fibrosis is formed on both pleural surfaces due to the increase in fibroblastic infiltration. As a result, these cases can be extremely difficult to manage without resorting to surgery[15,16] (Fig. 1). The appearance, analysis and culture of PE can vary in each of the evolutionary phases of PPE. The physiopathological changes that take place in the pleural space mean that, from a clinical point of view, PPE can be classified as: uncomplicated PPE (UPPE), which is resolved with antibiotic treatment; complicated PPE (CPPE), which will require CD or surgery for resolution; and empyema, i.e. the presence of pus in the pleural space, which must always be drained.

Diagnosis

Diagnosing PPE is not complex in patients presenting with classic symptoms, which are similar to pneumonia.[17] A poor response to pneumonia therapy could suggest the presence of a PPE or empyema as a complication of the disease.[18] It is sometimes difficult to suspect a PI, as the symptoms are atypical, and there is no evidence of pneumonia on chest radiograph.[19] Blood cultures are only positive in 12% of cases, PF culture is negative in more than 40% of samples[2] and, occasionally, the microorganisms responsible are very rare and can only be identified by molecular microbiology.[20] Although the criteria for defining CPPEs are well established,[17] there are no clinical or radiological data to determine which patients will go on to develop CPPE/empyema. As these are easily identifiable by their appearance, the difficulty lies in detecting (as early as possible) which patients with non-purulent PPE might progress to CPPE. Various diagnostic methods have been used for this purpose, but results have so far been disappointing.

Pneumonia severity scales and scores

Pneumonia severity scales[18,21] have been used to predict 30-day mortality following admission, but not to assess the likelihood of developing CPPE/empyema. Chalmers et al.[22] showed that these scales and generic sepsis scoring systems (APACHE II, SEWS and SIRS) cannot predict the development of CPPE/empyema. However, multivariate logistic regression has identified that: albumin < 30 g/l, Na < 130 mmol/l, platelet count > 400,000, C-reactive protein (CRP) > 100 mg/l and a history of alcohol abuse and intravenous drug use are independent risk factors for CPPE/empyema.[22]

Rahman et al.[23] developed a validated clinical risk score (RAPID) to identify patients with PI and high risk of dying, to enable clinicians to implement the best management strategy. The score includes urea levels, age, presence of purulent PF, source of infection (community or hospital), and dietary factors (albumin). Patients can then be divided into low, medium and high-risk groups according to the score. The odds ratio for mortality at 3 months for the medium- and high-risk groups, using the low-risk group as a reference, is 24.4 and 192.4, respectively. Using this system, patients with PI at presentation can be stratified according to risk.

Imaging tests

Radiographs, chest ultrasound (CU) and chest computed tomography (CT) can provide information on the size, extent and nature of the PE. PF volumes of >200–250 cm^3 are usually seen on the chest radiograph, and CPPE may be suspected if opacification of the pleural space does not change with the gravitational effects of the PF.

CU is a key technique in the management of PE, particularly PPE, and pulmonologists must be competent in its use. It is more sensitive than radiography for detecting minor PE, establishes the echogenicity of the PF, precisely locates the loculated fluid, estimates the volume and depth of the PE, differentiates between PF and underlying consolidation or atelectasia, improves the yield of thoracocentesis, and reduces the risk of complications compared to physical examination plus radiography.[24] An ultrasound finding of septa suggests CPPE (Fig. 2), and hyperechogenicity is associated with pus in the pleural cavity.

CT provides the clearest image of the pleura, and can be used to reconstruct images, determine loculations, and reveal lesions in the underlying lung. It also differentiates between a peripheral pulmonary abscess and a loculated PI, though the split pleura sign.[25]