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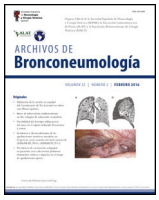

Editorial

Should Thoracentesis be Performed to Diagnose Pleural Effusion of Cardiac Origin?☆

¿Debe realizarse una toracocentesis para diagnosticar el derrame pleural de origen cardiaco?

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Heart failure (HF) is a clinical syndrome caused by a structural and/or functional abnormality of the heart, resulting in a decrease in cardiac output with or without raised intracardiac pressures. It occurs in 1%–2% of the adult population, but this prevalence increases to over 10% in individuals over the age of 70 years.[1]

Pleural effusion (PE) is a common finding in patients with decompensated HF. Eighty-seven percent of patients hospitalized for decompensated HF requiring diuretics show PE on computed tomography (CT).[2] PE is caused by increased hydrostatic pressure in the capillaries of the visceral pleura that causes fluid to flow into the pleural space.[3] HF is the most common cause of transudate and in some series it has even been reported to be the cause of all PEs.[4] Diagnosis usually is simple, established on the basis of the clinical picture and the presence of cardiomegaly and right or bilateral PE on chest X-ray. However, in certain circumstances, it is more difficult to establish a diagnosis. This confusion can be due to the presence of atypical symptoms (fever or chest pain), absence of cardiomegaly, marked asymmetry in the size of the PE, or lack of response to diuretics.

The first step in studying PE is to determine if the fluid is a transudate or exudate. In transudates, the pleura is not affected, and fluid accumulates because of an imbalance between the hydrostatic and oncotic pressures of the pleural vessels and the pleural space. In exudates, the disease that produces the PE directly affects the pleura, resulting in increased capillary permeability or obstruction of lymphatic drainage. Clinicians try to differentiate between the 2 types of effusion by determining the different biochemical parameters in PE and blood. It is assumed that, in transudates, where the pleura is intact, fewer solutes will pass into the pleural space compared with the greater capillary permeability of exudates. Criteria published by Light et al. have been used for differentiating between exudates and transudates since 1972. PE is considered a transudate if the ratio between proteins in pleural fluid and serum (PF/serum) is $\leq$0.5, the ratio of lactate dehydrogenase (LDH) in PF and serum is $\leq$0.6, and if LDH in PF is $\leq$2/3 of the upper limit of normal in blood.[5] Fluid that does not fulfill 1 or more of these criteria is considered an exudate. In the original article, only 1 of the 47 transudates was classified erroneously as an exudate.[5]

Although Light's criteria have greater diagnostic accuracy than initial pre-thoracentesis clinical presumption for differentiating transudates from exudates (93% vs 84%; P<.01),[6] they lead to the misclassification of a high percentage of transudates (around 30%),[7] and may not therefore be the ideal parameters for this task. Several factors can be involved. In the original series, almost all thoracenteses were performed prior to initiating treatment with diuretics, something that is rare today. With diuretics, water is first eliminated from the blood, followed by the diffusion of all extravascular fluids. As water diffuses from PE to a greater extent than from blood, the concentration of the various substances, such as proteins or LDH, and their respective ratios (PF/serum) become high enough for these effusions to meet the biochemical characteristics of exudate.[8] Sometimes there are several causes of PE. A recent series showed that 30% of unilateral PEs can be attributed to 2 possible causes,[9] and this percentage increases to 83% if the PE is bilateral.[10] In these circumstances, it is difficult to interpret the results of the fluid, since it is impossible to determine the influence of each disease on the biochemistry of the PF. Performing multiple thoracentesis to analyze PF can increase LDH (an intracellular enzyme), since the technique itself induces cell lysis, leading to the misclassification of the effusion as exudate.[8] Finally, the presence of a large number of red blood cells may influence LDH values and cause the fluid to behave biochemically as an exudate.[11] Given these results, other parameters have been proposed (pleural cholesterol, serum-PF albumin gradient, and PF/serum bilirubin ratio, instead of Light's criteria), but none showed superiority over the others.[12] For this reason, experts recommend abandoning the pursuit of a better PE marker.[13]

Cardiac myocytes respond to increased pressure and distension of the cardiac chambers by secreting natriuretic hormones as N-terminal fragment of pro-brain natriuretic peptide (NT-proBNP). The sensitivity and specificity of elevated NT-proBNP in PF for the

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diagnosis of cardiac PE is 94% and 91%, respectively, with a similar yield in blood.[14] Normal NT-proBNP values exclude HF as a cause of a unilateral PE. However, high NT-proBNP values do not mean that heart disease is the only cause of unilateral PE, since this finding could be due to occult heart disease, common with advancing age, or other causes. Furthermore, this marker is not always available, and laboratory methodologies vary widely, requiring the use of different cut-off points.[15] To overcome these difficulties, an index has recently been developed on the basis of age, PE laterality, LDH values in PF, and albumin and protein gradients to identify pleural exudates of cardiac origin.[15] Although results have been good (diagnostic accuracy 92% and positive likelihood ratio 12.7), this model remains arbitrary and not always easy to apply in clinical practice.

In summary, the results shown here seem to indicate that in a clinical context of HF, the cardiac origin of a PE must be confirmed by other procedures,[1] and not by thoracentesis. This technique should be performed only if there is suspicion of a second cause of PE.

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