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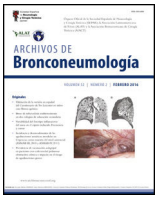

Editorial

Can Right Heart Failure Cause Pleural Effusion?☆

¿Puede causar derrame pleural la insuficiencia cardiaca derecha?

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Pleural effusion (PE) is a common finding in patients with heart failure (HF).[1,2] In 1985, Wiener-Kronish et al. reported that in patients with congestive HF due to ischemic heart disease or cardiomyopathy, PE correlated with elevated left atrial, but not right atrial pressure,[3] and that PE was not detected in patients with increased right atrial pressure or pulmonary hypertension (PHT).[4] Since then it has been accepted that these situations are not the root cause of PE, although the evidence is scant, because few patients undergo thoracentesis in the acute phase, and the hemodynamic data obtained from bilateral catheterizations from virtually all patients is extremely limited.

The controversy arose when studies published in 2009 and 2011 described patients with PHT and only right HF who did present PE.[5,6] This claim was based on the hemodynamic results of 47 patients with idiopathic or familial pulmonary arterial hypertension (PAH) (19 patients)[5] or connective tissue disease (CTD) (28 patients) and PE,[6] who had a significantly greater mean right atrial pressure (mRAP) than those who did not present PE. PE could develop because the increase in systemic venous pressures causing right HF would prevent venous lymphatic drainage or increase hydrostatic pressure in the bronchial veins and the chest wall, as observed in animal studies when the superior vena cava is acutely obstructed.[7,8] A subsequent study showed that 11% of patients with PHT present PE, and that this is a predictor of mortality. Although the hemodynamic data of patients were not provided, markers of right heart failure were associated with lower survival.[9] The results of these studies deserve comment.

In the first article,[5] the mRAP of patients with idiopathic or familial PHT with PE was significantly higher than those without PE; however, when the comparison was made between patients with right HF, with or without PE, no differences were observed for any of the hemodynamic parameters measured. In the second study,[6] patients with CTD and PE had significantly higher pulmonary vascular resistance and mRAP (11.3±5.1 mmHg) than those without PE (8.3±4 mmHg mRAP). Patients with PE also had significantly lower cardiac index and output, but no differences in mean pulmonary artery pressure, right ventricular diastolic pressure (RVDP) and pulmonary artery occlusion pressure.[6] The authors conclude that PE in these patients is associated with right HF. However, some issues remain unclear. Patients with PE had significantly higher mean values of brain natriuretic peptide, suggesting that other factors might have contributed to PE accumulation. It is difficult to understand why differences were observed in mRAP but not in RVDP. One explanation may be because it is a dynamic parameter, and situations such as the previous use of diuretics or the level at which "zero" is set on the pressure transducer could produce small variations in its measurement (2–3 mmHg would be sufficient) that would explain the differences (which are only 3 mmHg). In contrast, Wiener-Kronish et al. did not detect PE in a study of 4 patients with an mRAP of 20 mmHg.[4] PHT can also be caused by left heart disease.[10] Left ventricular diastolic dysfunction due to right ventricular pressure overload, causing deviation of the interventricular septum towards the left ventricle, may cause congestive HF.[11] Left ventricular end-diastolic pressure was not measured,[5,6] and while pulmonary artery occlusion pressure alone was slightly elevated in some patients, this possibility cannot be ruled out without confirmation by left catheterization. Data from studies in animal models with acute superior vena cava obstruction are not fully extrapolated to situations of HF. In the study in dogs,[7] the low concentration of proteins in plasma or acutely modelled systemic venous hypertension could explain the PE. In contrast, patients included in the studies of Wiener-Kronish et al. had signs and symptoms lasting more than one year, suggesting that their increased pulmonary artery and right atrial pressure were chronic.[3,4]

Both publications[5,6] raise critical questions that are not examined in depth. In 3 of the 50 patients in both series (6%), PE was not due to right HF, but the authors fail to explain the causes. Ascites, pericardial effusion, and CTD are all causes of PE.[12–14] Eight of the patients with right HF and PE from the first study and 9 from the second study (34%) had ascites, and 8 patients with right HF and PE from the first study and 21 from the second (38%) had pericardial effusion. In both studies, ascites and pericardial effusion were significantly more frequent in the group with PE. Given that 10% of patients with liver cirrhosis and ascites[10] and one-third of patients with pericardial effusion[12] have PE, it cannot be ruled out that these were the causes of the PE in some cases.

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PE was observed in 41.7% of patients with scleroderma (20/48), 33.3% of patients with systemic lupus erythematosus (5/15), and 23.5% of patients with mixed connective tissue disease (4/17) (29 patients; 58% of the total of both studies).[6] CTD can cause PE,[14] and the percentages obtained are within the range of the published figures, except for scleroderma, which was higher.[14] Therefore, it cannot be ruled out that the PE of some of these patients may be due to their CTD, rather than to right HF.

In summary, we believe that insufficient data are available to confirm that right HF alone can produce PE. Cases of PE have been described in patients without left HF, but a considerable proportion of these cases have possible alternative diagnoses, and moreover, the hemodynamic conditions of these patients have not been sufficiently studied.

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