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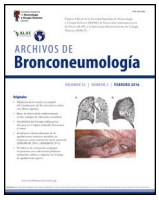

Editorial

Heart Failure: Is it a Lung Disease?☆

Insuficiencia cardíaca: ¿una patología neumológica?

The heart and lung are two closely related organs that collaborate in maintaining the metabolic balance of the body by participating in gas exchange and oxygen transport. From a pathophysiological point of view, these organs, together with the kidney, form a functional unit: hypercapnia and hypoxemia modify ventricular preload, afterload, and diastolic function, which can alter the distribution of renal vascular flow, and participate in the hormonal control of the salt and water exchange.[1] From a clinical point of view, the failure of one of these organs can affect the functioning of the others and trigger compensation mechanisms where the cardinal symptom is usually dyspnea, often accompanied by respiratory failure and alterations on chest X-ray. In these situations, the challenge for the clinician is to detect the level of dysfunction of each organ and the most appropriate therapeutic measures.

Heart failure (HF) occurs in up to 2% of the adult population in developed countries, and in up to 10% of individuals over 70 years of age. From a pathogenic point of view, we can differentiate between two mechanisms: 1. Deficiencies in left ventricular contractility (systolic dysfunction) and 2. Alterations in ventricular filling (diastolic dysfunction). In both cases, the consequence is reduced cardiac output and/or elevated pressure in the cardiac cavities.[2] Systolic dysfunction is generally caused by a primary heart problem, while diastolic dysfunction has a more complex pathophysiological mechanism that presents with different phenotypes, often related to the patient's overall comorbidity burden [hypertension, diabetes, chronic kidney disease, chronic obstructive pulmonary disease (COPD), and obesity].[2]

Clinical practice guidelines on the management of HF recommend performing echocardiography to assess left ventricular contractility and distinguish between systolic dysfunction (left ventricular ejection fraction <40%) and diastolic dysfunction. In case of the latter, clinical signs and symptoms, natriuretic peptides, and structural and functional data that can be extracted from the echocardiography should be taken into account.[2]

COPD is one of the most common diseases in respiratory medicine, and it is estimated that up to 25% of cases over 65 years of age present with concomitant HF.[3] Furthermore, up to 30% of patients with HF have COPD, so the association between COPD and HF is well known and has been addressed in multiple publications.[2,4] The coexistence of both diseases increases morbidity, use of resources, and mortality. The diagnosis of COPD is based on demonstrating airflow obstruction, but HF in itself can also induce spirometry alterations of both an obstructive and non-obstructive nature[5,6]: all these factors complicate the diagnostic process.

Obesity is currently a public health problem closely related to respiratory diseases, such as sleep apnea-hypopnea syndrome (SAHS), obesity-hypoventilation syndrome (OHS), or asthma. Obese individuals show very diverse alterations in lung function tests[7] and have an high risk of HF that can complicate the clinical picture in the presence of asthma, SAHS, or OHS.[8–10] A recent study reported that up to 46% of patients with stable OHS have hemodynamic alterations consistent with HF, detected using cardiothoracic impedance, a technique rarely used even in our specialty.[11]

HF is also associated with central apneas and breathing pattern changes in the form of Cheyne-Stokes respiration. These cases require assessment and diagnostic studies in sleep units and sometimes need specific treatment with continuous positive airway pressure (CPAP) or Servo ventilators.[12] Servo ventilation is prescribed only in patients with normal left ventricular contractility, so echocardiography is necessary.

When considering diagnosis and communication with other professionals (cardiologists), it should be noted that lung function changes and respiratory failure are very well defined, so the difference between normality and abnormality can be established with some clarity. However, the diagnosis of HF will often be based on a clinical syndrome and will require a combination of clinical, analytical and radiological data.[2] The pulmonologist is then faced with diseases within the respiratory medicine spectrum in which HF may be present, and must be detected, evaluated, and treated. Despite the fact that the association of COPD and HF is well known, it is clearly underestimated,[13] and we can only assume that HF occurring with obesity-related diseases will also present this problem. Logically, failure to diagnose leads to incomplete treatment, so we could probably improve the prognosis of our COPD and OSH patients (especially the most advanced cases) by including the relevant cardiological examinations in our routine diagnostic processes. The appropriate treatment for HF (diuretics, β-blockers and/or ACE inhibitors) will then be added to the treatment for the associated disease. It is worth mentioning β-blockers here, one of the key HF treatments that is often suspended in the presence of COPD.

Other chronic respiratory diseases that can affect lung function and gas exchange may also be associated with a certain degree of HF that constitutes a factor for exacerbations and the need for hospital

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admissions. To establish the degree to which HF is involved in this process, clinical, analytical, radiological, and echocardiographic criteria will need to be applied.[1]

Finally, it seems clear that the high prevalence of HF means that many patients are treated not in cardiology units, but rather in Primary Care, Internal Medicine or Geriatrics.[14–16] We pulmonologists, too, will probably have to develop diagnostic and therapeutic skills, and foster our communication with cardiologists. The next challenge for our specialty will be to add basic echocardiography and non-invasive hemodynamic techniques, such as cardiothoracic impedance, to our procedures in order to more accurately characterize the cardiological status of our patients and thereby improve our knowledge and support of these diseases.

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