Acute shortness of breath and chest tightness in an elderly patient: clinical history tells it best!

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Abstract

Almost 50% of patients presenting with heart failure have no systolic left ventricular dysfunction and thus are diagnosed with diastolic heart failure (HF). Diastolic HF reflects an increased sensitivity to volume status and vasoreactivity of patients with underlying diastolic dysfunction, a condition commonly observed in elderly patients with numerous comorbidities. In this case report, an example of the diagnostic and therapeutic challenges in a patient with diastolic HF is illustrated. Indeed, while defined international guidelines are available for patients with systolic HF, the management of diastolic HF patients is less well defined and largely dependent on patients’ characteristics.

Keywords: Comorbidities; comprehensive evaluation; diastolic dysfunction; diastolic heart failure; non invasive assessment.

Case report

A 78-year-old woman was admitted to our clinic for evaluation of acute shortness of breath and chest tightness. She had a history of hypertension, diabetes and mild chronic renal insufficiency, with no previous ischemic heart disease or pulmonary disease. In the last few months before admission the patient reported a history of accidental trauma resulting in chronic lumbosacral pain that she had been managing with non steroidal anti-inflammatory drugs. Her medical history was unremarkable for other pathological conditions.

On physical examination she presented with increased jugular venous pressure, peripheral edema (1+) and blood pressure levels of 195/110 mmHg. Arterial oxygen saturation was 88% on a 100% non rebreather mask. The heart rate was regular at 93 beats/min. On lung auscultation there were rales at the bases, and cardiac examination revealed a normal S1 and S2 with an early peaking systolic ejection murmur (2/6 L), but no S3. The abdomen was tender and there were no bruits or palpable masses. The 12-lead electrocardiogram showed normal sinus rhythm and diffuse non specific ST-T segment changes. On chest X-ray there were signs of pulmonary congestion, with no signs of cardiomegaly (Fig. 1). An echocardiogram revealed mild left ventricular hypertrophy with preserved dimensions and ejection fraction (63%), increased left atrium dimensions with only mild mitral insufficiency and no other valve
dysfunction. There was an abnormal ventricular Doppler filling pattern with elevated peak E-velocity, short E-deceleration time and a markedly increased E/A ratio, reduced E’ velocity of tissue Doppler images, increased E/E’ ratio (>16) and reverse S/D ratio, all findings consistent with elevated pulmonary-capillary wedge pressure. Blood tests documented increased brain natriuretic peptide (960 pg/mL) and creatinine levels (2.4 mg/dL), with almost normal troponin I, creatine phosphokinase and myoglobin levels.

Discussion
According to major international guidelines, heart failure (HF) is a syndrome in which patients have the following features: symptoms of HF, typically shortness of breath at rest or during exertion, and/or fatigue; signs of fluid retention such as pulmonary congestion or ankle swelling; and objective evidence of an abnormality of the structure or function of the heart at rest [1, 3].

Although there was no direct evidence of structural abnormalities of the heart, the described is clearly a case of HF (symptoms and objective findings of HF). In addition, there was indirect evidence (left atrium enlargement, altered Doppler indices) of increased pulmonary-capillary wedge pressure, which could justify the clinical presentation.

Diastolic dysfunction refers to an abnormal distensibility and/or relaxation of the left ventricle that can take place regardless of the overall left ventricular systolic function. The condition is now recognized as a common cause of HF, perhaps representing up to 50% of all HF patients, with a similar dire prognosis [3]. Diastolic dysfunction can be diagnosed through the identification of abnormalities in diastolic filling pattern, commonly assessed with non invasive Doppler techniques. Diastolic HF (or HF with preserved systolic function) reflects an increased sensitivity to volume status and vasoreactivity [4]. Therefore, hypertensive episodes due to labile hypertension, medical and dietary non compliance, non steroidal anti-inflammatory drugs, atrial fibrillation, ischemia, or iatrogenic volume overload are all conditions known to precipitate acute HF in this patient population. In addition, there is a remarkable female predominance [5], and as compared to systolic HF, a relative increase in the hospitalization rate [6].

The diagnostic flowchart of patients with diastolic HF is similar to that of patients with systolic HF, and includes a complete blood count, urinalysis, serum electrolytes, glycohemoglobin and blood lipids, as well as tests of both renal and hepatic function, a chest radiograph, a 12-lead electrocardiogram and echocardiographic evaluation; elevated levels of natriuretic peptides lend additional support to the diagnosis [7]. However, the diagnosis of diastolic HF is basically based on the exclusion of a systolic abnormality of left ventricular function. The presence of high left ventricular filling pressures is accurately assessed by invasive hemodynamic evaluation, which represents the gold standard method. However, in clinical practice, invasive assessment is difficult to apply, and filling pressures are commonly extrapolated from combined echocardiographic parameters and natriuretic peptides levels.

Our patient did not have systolic dysfunction, whereas Doppler indices and blood tests suggested a diagnosis of acute HF with preserved ejection fraction, probably secondary to acute renal injury.

Implementation of the therapeutic strategy represents another challenge in diastolic HF. Indeed, despite its clinical and epidemiological significance, treatment of diastolic HF remains largely empirical and not evidence based. The few available clinical trials only evaluated the effectiveness of renin–angiotensin system inhibitors, with none showing survival benefit [8–10]. Even less is known about the therapeutic
benefits of β-blockers and diuretics, and the lack of evidence appears secondary to the numerous associated comorbidities that are major contributors to clinical outcomes in this patient population [11,12].

Following the identification of a worsening renal function with fluid retention as a possible precipitation factor, our patient was treated with intravenous diuretics and fluids, as well as continuous oxygen support through a non rebreather mask, obtaining rapid improvement of the clinical status. On day 2 after admission, brain natriuretic peptide and creatinine levels were 195 pg/mL and 1.5 mg/dL, respectively, and blood pressure levels were normal. Furthermore, care was taken to optimize the discharge therapy allowing for an optimal 24 hours blood pressure control and renal protection by adjusting angiotensin converting enzyme inhibitor and diuretic dosages. The patient was also advised to limit non steroidal anti-inflammatory drugs and encouraged to perform regular exercise training, a measurement that appears to confer benefit in terms of enhancements in exercise capacity and health-related quality of life [13].

Conclusion

As previously mentioned, no specific treatment regimens have been shown to benefit diastolic HF patients, and treatment will often be multifactorial and individualized to each patient. Currently, many studies (in vitro and animal) are assessing the effects of several growth factors, cytokines and signaling molecules that have been shown to reverse myocardial fibrosis, a determinant factor for increased ventricular stiffness and diastolic dysfunction [14]. However, we are still far from adopting these new therapeutic agents. Until more progress is made in this area, treatment of precipitating factors (ie, aggressive blood pressure control by restricting salt intake and administration of diuretics, thus enhancing renal function; maintenance of sinus rhythm to preserve atrial contraction and heart rate control to improve diastolic function; and treatment of underlying comorbidities, using an integrated and multidisciplinary approach) appears the most effective strategy to reduce morbidity and hospitalization in this patient population.

References