High output heart failure caused by a large pelvic arteriovenous malformation

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We describe a case of high output heart failure due to a large and spontaneous pelvic arteriovenous malformation, highlighting its importance in the differential diagnosis of heart failure syndromes.

Case report

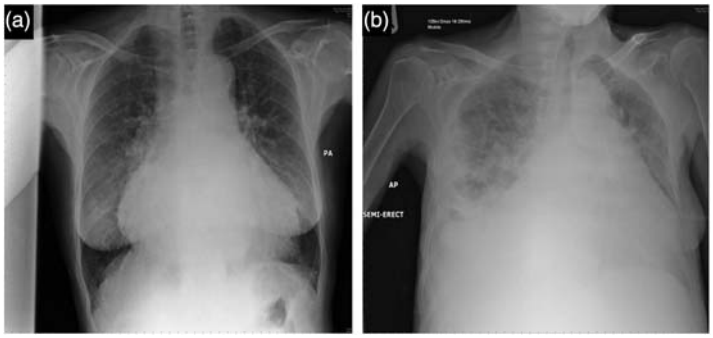
A 73-year-old woman was referred to our department due to mild breathlessness on exertion. She denied chest pains but occasionally had palpitations, which were not troublesome. She did not complain of fluid retention and nor did she have orthopnoea or paroxysmal nocturnal dyspnoea. Her previous medical history included abdominal aortic aneurysm (AAA) repair 15 years ago for infra renal aneurysm. On examination she was in atrial fibrillation with a rate of 64 bpm, was normotensive and with no pedal oedema. Cardiovascular examination revealed significantly raised JVP with systolic V waves, a pansystolic murmur at left lower sternal edge and systolic flow murmur in pulmonary area. Lung fields were clear with no added sounds. A continuous loud bruit was noted on examination of her lower abdomen.

Routine blood tests showed she had normocytic anaemia with a haemoglobin level of 10.4 g/dl, but both liver and renal function tests were normal. Her electrocardiogram (ECG) revealed rate controlled atrial fibrillation with no ischaemic changes. Chest X-ray showed massive cardiomegaly (Figure 1a). Her transthoracic echocardiogram (Figure 2a) showed normal left ventricular systolic and diastolic function but dilatation of all four heart chambers with severe biatrial dilatation. There was moderate to severe tricuspid regurgitation with only mild pulmonary hypertension and no evidence of intra-cardiac shunting. Subsequently, her cardiac output evaluated by echocardiography was elevated at 9.8 L/min with cardiac index of 6.0 L/min/m^2 (>3.9 L/min/m^2 considered as high cardiac output). Further confirmation of her echocardiographic findings by invasive cardiac catheterization was not undertaken as the values obtained were definitive and not borderline, Doppler echocardiography has been validated against catheter-derived parameters and serial assessment of cardiac output is more conveniently assessed non-invasively for elderly patients.[1]

Contrast CT scan of abdominal aorta and pelvic vessels was arranged (Figure 2b). This showed grossly enlarged iliac vessels forming a massive pelvic arteriovenous malformation. The inferior vena cava was dilated (3.8 cm diameter) with high attenuation consistent with high flow. The relationship of the arteriovenous malformation to her previous AAA surgery remained undetermined, as previous operation notes and imaging were unavailable.

Despite treatment with salt restriction and loop diuretics, she suffered increasing breathlessness on minimal exertion and developed worsening pedal oedema. Further chest X-ray showed pulmonary oedema with bilateral pleural effusions (Figure 1b). In view of her frail condition and large size of the arteriovenous malformation, it was decided not to consider surgical correction. As an alternative, she underwent repeated courses of alcohol sclerotherapy administered percutaneously to embolize the fistula. Transiently, her symptoms improved and serial echocardiographic assessment of cardiac output revealed a reduction to a nadir of 5.4 L/min but eventually further deterioration ensued with recurrent

Figure 1
a. Chest x-ray on initial presentation showing massive cardiomegaly; b. Chest X-ray on follow-up showing pulmonary congestion with bilateral pleural effusion



admissions due to decompensated congestive heart failure and fluid retention. Finally, she died due to urinary sepsis.

Discussion

Normally congestive heart failure is characterized by low cardiac output with reduced left ventricular function, but the syndrome of heart failure in the presence of high cardiac output states is called as high output heart failure. The main causes of high output heart failure include large arteriovenous malformations or fistulas, arteriovenous shunts such as those seen in Paget's disease, thyrotoxicosis and acutely in septic shock. Although arteriovenous malformations are an uncommon cause of high output heart failure, both iatrogenic and spontaneous forms have been described.[2,3]

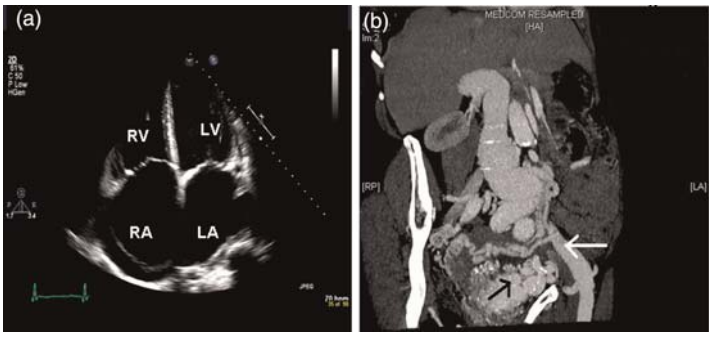
A high cardiac output is defined as cardiac output of more than 8 L/min or a cardiac index of more than >3.9 L/min/m^2.[4] The important physiological event in high output heart failure is reduced systemic vascular resistance. This in turn leads to activation of the renin-angiotensin-aldosterone system, causing salt and water retention and clinical heart failure. Important physical finding in high output heart failure patients is the presence of warm peripheries rather than cold peripheries (as in low cardiac output states) due to low systemic vascular resistance and vasodilatation.

Mixed venous oxygen saturation (SvO2) provides an estimate of the body oxygen consumption/delivery ratio. A low SvO2 (<65%) is associated with an inadequate cardiac output and conversely a high SvO2 (>75%) may be due to a high cardiac output state. The echocardiography often shows compensatory left ventricular dilatation and left ventricular hypertrophy but preserved or normal left ventricular systolic function.[5] In addition, patients have dilated right-sided heart chambers due to increased venous return.

As the heart function is usually normal, the aim of the management is to treat the underlying cause of high output state and to eliminate the fluid retention. Diuretics are the main stay of treatment in high output heart failure, especially with symptoms of volume overload. Avoid vasodilators as they reduce systemic vascular resistance resulting in deterioration in the condition.

Arteriovenous malformations are treated by either percutaneous embolization or surgery. Specific treatments for arteriovenous malformations are mainly based on case reports and small series. Alcohol is a powerful sclerosing agent and causes thrombosis of the malformation.[6] While cure is not always possible, alcohol sclerotherapy can often reduce the flow in and size of the lesion to an extent that will achieve symptomatic relief. Surgical treatment

Figure 2
a. Transthoracic echocardiogram (apical four chamber view) showing grossly dilatation of all four cardiac chambers including severe biatrial dilatation. RV = right ventricle; LV = left ventricle; RA = right atrium; LA = left atrium; b. Contrast CT scan of aorta and iliac vessels showing large pelvic aretriovenous malformation (black arrow) arising from left iliac artery (white arrow) with calcification indicating that it is likely to be longstanding



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for arteriovenous fistula is recommended when it is large enough to produce high output state causing cardiac enlargement and fluid retention.[7]

Conclusion

Although high output heart failure states are generally rare clinical entities, it should still be considered as part of the differential diagnosis of patients with heart failure symptoms and normal left ventricular systolic function as prompt diagnosis and specific treatment of the high output states can often resolves their heart failure, while conventional treatment with vasodilators can aggravate the condition. Unfortunately clinical trial data are lacking in this area and further studies are required to better guide management.

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