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High-Output Heart Failure Resulting from a Remote Traumatic Aorto-Caval Fistula: Diagnosis by Echocardiography

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Summary

Congestive heart failure (CHF) due to high output states is known to occur in a variety of systemic illnesses and in patients with arterial-venous fistulas. This paper reports the case of a 45-year-old man admitted to the emergency room with a diagnosis of new onset atrial fibrillation and CHF, whose past medical history was not significant except for a gunshot wound to his abdomen 22 years previously. The etiology of his CHF together with the cardiomegaly and hyperdynamic left ventricular systolic function was unknown. A subcostal view routinely done during transthoracic echocardiography revealed a severely dilated inferior vena cava and the presence of an aorto-caval fistula by color doppler. The patient underwent successful corrective repair with dramatic improvement in symptoms and resolution of the atrial fibrillation, and cardiac size returned to normal. This rare case emphasizes that patients with refractory CHF must be closely examined with particular attention to palpation and auscultation over all scars, irrespective of the duration since any traumatic or surgical event.

Key words: congestive heart failure, arterial-venous (A-V) fistula, pseudoaneurysm, atrial fibrillation, cardiomegaly

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Introduction

Congestive heart failure (CHF) as a result of a high cardiac output state is well described[1] and usually occurs when the resting cardiac output is significantly increased. A high output state may be suggested when signs and symptoms of CHF are refractory to conventional therapy or when systemic illnesses known to cause a high output state, such as hyperthyroidism, thiamine deficiency, severe anemia, cirrhosis, or Paget's disease of the bone, are uncovered on a complete history and physical examination.[2] Rarely do arterial-venous (A-V) fistula, A-V malformations, or abnormal connections between the arterial and venous systems result in high output failure. These conditions may be difficult to detect and frequently may be a result of traumatic injury or even diagnostic testing, such as renal biopsy.[3] Hemodynamically significant A-V fistula can be seen relatively frequently in patients with end-stage renal disease who have fistulas created by hemodialysis.[4] Previously unknown A-V fistulas may become clinically evident when cardiac demands are increased, such as with pregnancy.[5]

The presence of an aortocaval fistula as the etiology of CHF is an even rarer entity. Herein, we report a case of high-output heart failure, diagnosed by transthoracic echocardiography, that resulted from an aortocaval fistula following a remote penetrating traumatic injury in a 45-year-old man with no prior cardiac disease. Symptoms and signs of CHF completely resolved in this patient after surgical closure of the fistula.

Case Report

A 45-year-old man was admitted to the cardiology service for evaluation of new onset atrial fibrillation complicating severe CHF. He initially presented to the emergency room reporting markedly increased shortness of breath associated with paroxysmal nocturnal dypsnea and two-pillow orthopnea of 4 to 6 months' duration. He denied chest pain or any other major cardiovascular complaints, but had noticed "racing" of his heart during heavy manual labor. Of note, the patient also reported a "balloon-like throbbing" sensation in his stomach ever since he suffered a gunshot wound to his abdomen 22

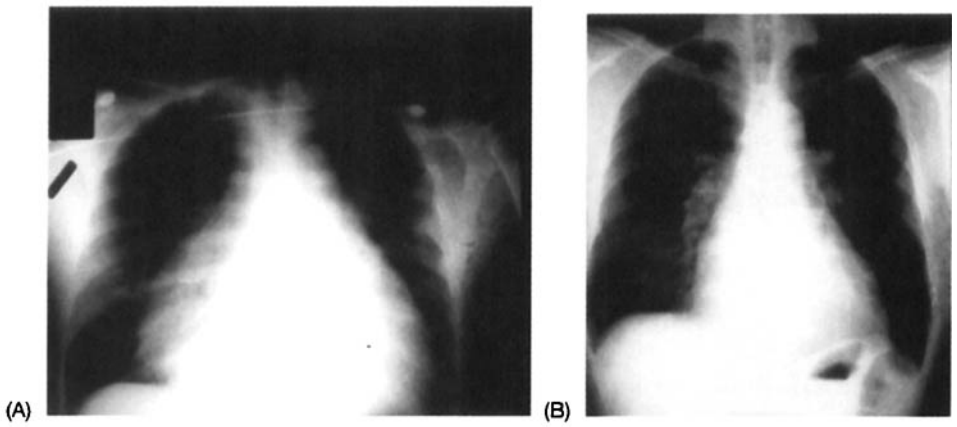

FIG. 1 Posteroanterior chest x-ray before (A) and four months after (B) surgical repair of aorto-caval fistula.

years ago. His past medical history was otherwise negative except for the exploratory laparotomy done at the time of the injury. No official report was available but the patient recalled having been told that the bullet had penetrated a blood vessel, which was apparently repaired.

On physical examination he was afebrile, his pulse was irregularly irregular at 110 beats/min, blood pressure was 120/66, and respiratory rate was 22/min. His neck veins were markedly distended and were seen to pulsate irregularly. The cardiac examination revealed a large, hyperdynamic, and leftwardly displaced apical impulse. The first and second heart sounds were unremarkable with a loud third heart sound. A crescendo-decrescendo 3/6 systolic ejection murmur was heard over the entire precordium. There were bibasilar inspiratory rales without wheezing on lung exam. His abdomen was not distended but there was a diffuse, pulsatile, nontender supraumbilical mass with a faint systolic bruit. There was no pedal edema, and he had good distal pulses without cyanosis.

The patient was initially treated with oxygen via nasal cannula, intravenous lasix, and digoxin. His shortness of breath improved and his heart rate was partially controlled. He continued to have evidence of CHF despite diuresis and heart rate control. He was then transferred to the cardiology telemetry floor with a diagnosis of new onset atrial fibrillation and CHF.

Laboratory test results including serum electrolytes, liver function tests, coagulation studies, thyroid function tests, and complete blood cell count were all normal. Chest x-ray demonstrated significant cardiomegaly (Fig. 1) and interstitial pulmonary edema while his electrocardiogram showed atrial fibrillation with rapid ventricular response and nonspecific ST-T-wave abnormalities. Transthoracic echocardiography revealed a hyperdynamic left ventricular systolic function with an estimated ejection fraction of 75% and clearly evident four-chamber dilatation. There was no significant valvular disease. Subcostal views showed a markedly dilated inferior vena cava (6.4 cm diameter), with color Doppler indicating turbulent flow from the abdominal aorta to the inferior vena cava (Fig. 2). Computed tomography scan with contrast of the abdominal aorta showed a 3.5 cm diameter pseudoaneurysm with peripheral calcification arising out of the aorta at the level of bullet fragments lodged in the vertebral body (Fig. 2). Right heart cardiac catheterization demonstrated right atrial pressure of 14 mmHg, right ventricular pressure of 55/14 mmHg, pulmonary capillary wedge pres-

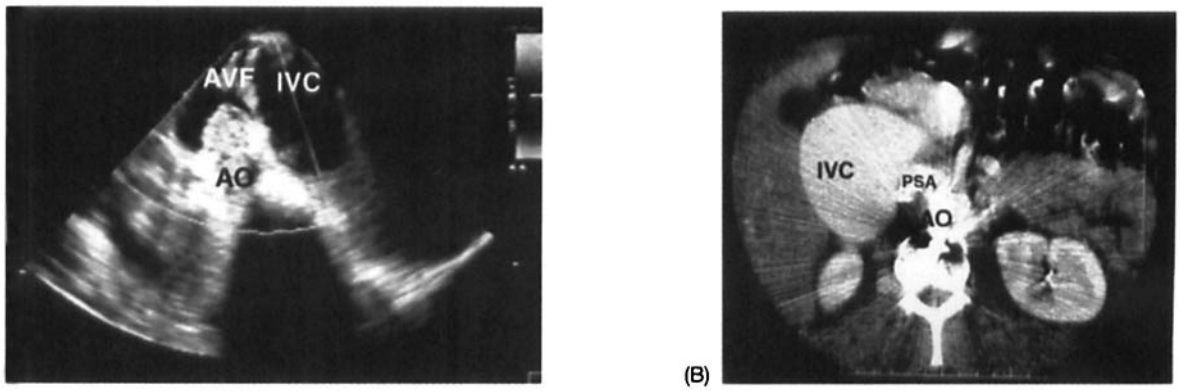

FIG. 2 Subcostal view by two-dimensional transthoracic echocardiography (A). Contrast computed tomography angiogram of abdomen (B). IVC = inferior vena cava, AVF = color Doppler jet of arterial-venous (aorto-caval) fistula, Ao = descending aorta, PSA = pseudoaneurysm.

sure of 22 mmHg, with a pulmonary artery pressure of 55/24. Cardiac output by thermodilution was 13.3 l/min with a systemic vascular resistance of 476 dynes/cm and an inferior vena caval oxygen saturation of 92%.

The patient then had successful corrective repair of the aorto-caval fistula and 4 months later is asymptomatic with excellent functional capacity in normal sinus rhythm. Repeat chest x-ray is shown (Fig. 1).

Discussion

High-output cardiac failure is known to occur in a number of systemic illnesses, including severe anemia, polycythemia vera, hyperthyroidism, severe liver disease, Paget's disease of bone, and systemic A-V fistula.[2] A hemodynamically significant A-V fistula can result from blunt and penetrating trauma, malignant disease, inflammation, and various invasive procedures such as partial nephrectomies, renal artery angioplasties, as well as percutaneous renal biopsies.[4] Rarely does peripheral A-V fistula arising from a remote peripheral injury also result in high output failure.[6]

Systemic A-V fistulas increase cardiac output by two mechanisms: (1) a decrease in systemic vascular resistance and (2) an increase in venous return. Clinical signs of the hyperdynamic state are usually absent unless the fistula involves large arteries and veins. The shunt from an artery to the vein decreases oxygenated blood to the tissue beyond the shunt and raises venous pressure distal to the fistula. However, venous pressure proximal to the fistula is usually normal, as is right atrial pressure, unless there is complicating CHF. Because of the decreased systemic vascular resistance, heart rate and stroke volume increase through baroreceptor mechanisms. Diastolic blood pressure is reduced and systolic arterial pressure and cardiac output rise while plasma volume tends to be increased. Our patient demonstrated several of these features, such as a decreased systemic vascular resistance and resting tachycardia, but this case was challenging and unique because CHF did not develop in this patient for 22 years. In addition, the presentation of atrial fibrillation and pulmonary edema with signs suggestive of an abdominal aortic aneurysm led us to hypothesize that there may be other etiologies responsible for the patient's findings. The most important diagnostic clue was turbulent vascular flow seen with color Doppler in the subcostal view during transthoracic echocardiography and the enlarged structures seen with two-dimensional imaging confirmed the presence of the aorto-caval fistula.

Conclusions

It is well accepted that A-V fistulas may be a cause of high-output heart failure. The speed of development of CHF is a function of the size of the fistula and the underlying cardiac condition. In this patient with a structurally normal heart, high-output heart failure developed 22 years after the establishment of the fistula. Once heart failure was apparent, it responded dramatically to surgical correction and would not have been amenable to medical therapy. The diagnosis of high-output failure due to aorto-caval fistula was confirmed by the echocardiographic findings evident in the subcostal views. This further emphasizes the role of echocardiography in the assessment of patients with CHF. It is not only helpful in monitoring these patients and guiding the appropriate timing of therapy but also in prompt diagnosis of potentially reversible etiologies of CHF. The past history and careful directed physical examination must consider the possibility of an A-V fistula. In particular, palpation and auscultation should be carried out over all scars in search of the characteristic bruit and thrill irrespective of the interval duration from a traumatic or surgical event.

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