High output heart failure in a young woman secondary to massive arteriovenous malformations from a uterine tumor

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DESCRIPTION

A 30-year-old woman with no significant medical history, presented with subacute onset dyspnea for 1 month. At presentation, her physical examination revealed tachycardia, bilateral lung crackles and a faint S3 on chest examination. Abdominal examination showed lower abdominal distention with an audible bruit. Laboratory tests showed a normal complete blood cell count, basic metabolic panel and thyroid stimulating hormone. Liver function tests showed a total bilirubin of 2.1 mg/dL, with normal transaminases. Brain natriuretic peptide was >2000 pg/mL. An ECG revealed sinus tachycardia. CT of the chest revealed cardiomegaly and bilateral pleural effusions ([figure 1](#)). The patient was given intravenous lasix which improved her symptoms initially. She had an echocardiogram that showed a left ventricular ejection fraction of 60%. An MRI of the abdomen was performed due to the presence of an abdominal bruit. It showed massively dilated blood vessels in the uterus that resulted in large uterine arteriovenous malformations and high output heart failure ([figure 2](#)). The patient subsequently had a total abdominal hysterectomy after failed endovascular embolisation of the uterine vessels. Pathology was positive for gestational trophoblastic disease. She subsequently received chemotherapy and immunotherapy. Her heart failure completely resolved after the hysterectomy.

High output cardiac failure is an uncommon cause of heart failure. Various physiologic, pathologic and iatrogenic aetiologies can lead to this.

Figure 2 MRI abdomen showed abnormal serpentine uterine and parametrial vessels (arrow) that mostly replaced the uterus. These enhance in both the arterial and venous segments in the arterial phase. This is consistent with massive uterine arteriovenous malformations.

Physiological causes include pregnancy, fever and illness. Pathological causes include morbid obesity, liver disease, arteriovenous shunts, Lung diseases and myeloproliferative disorders.¹ Iatrogenic causes include iatrogenic AV fistulas, pulmonary vasodilators and inotropic medications. High output cardiac failure from AV fistulas results from the shunting of blood directly to low-pressure veins bypassing the capillary beds. There is a compensatory increase in the stroke volume, heart rate and plasma volume.¹ This may result in sympathetic

Figure 1 CT chest showed effusions and cardiomegaly.

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Learning points

- ▶ Arteriovenous malformations are a rare cause of heart failure. They should be considered as a differential especially if the patient appears otherwise healthy.
- ▶ High output heart failure can be managed easily with treating the underlying cause of the heart failure.

activation of the renin–angiotensin axis with resultant elevated renovascular resistance and chronic salt and water retention leading to ventricular enlargement or dilatation in the long term.² The diagnosis is based on clinical features of heart failure and identification of an AV fistula. Management includes volume status optimisation and correcting the fistula.

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