LESSON OF THE MONTH

Haematuria and an Abdominal Aortic Aneurysm – Warning of an Aortocaval Fistula

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Introduction

The incidence of atherosclerotic abdominal aortic aneurysm (AAA) as well as its associated complications is steadily increasing in Western populations. Although AAA may rupture spontaneously into adjacent tissues and viscera, the formation of aortocaval fistulas (ACF) is rare and has been reported to occur in only 0.2–1.3% of AAA. However, the mortality of this rare complication ranges from 30% to 60% in previous reviews.

Case Report

A 65-year-old man was hospitalised with persistent haematuria, urinary retention and oedema of the lower limbs. He was initially treated for urinary infection, but became progressively dyspnoeic and developed myocardial and renal insufficiency. Physical examination revealed blueish lower limbs with concomitant oedema. The abdomen was distended, with a palpable, pulsatile abdominal mass without murmur or thrill. The systolic blood pressure was 120 mmHg with a pulse rate of 110 bpm. A CT scan of the abdomen showed an AAA of 6 cm in diameter and retroperitoneal fluid. The inferior vena cava could not be differentiated from the aneurysmal sac (Fig. 1).

The patient underwent an emergency laparotomy. Intraoperative findings revealed an ACF of 2 cm in diameter, partially covered by an intra-aortic thrombus. The caval defect was sutured from inside the aneurysm sac and the aneurysm excluded with an interposition Dacron aortobi-iliac graft. Re-laparotomy for profuse bleeding from the right iliac artery had to be performed on the first postoperative day. The rest of the postoperative course was uneventful and the oedema of the legs and haematuria subsided rapidly. Renal and cardiac function also returned to normal. A follow-up CT scan two years later showed a patent inferior vena cava and aortobi-iliac graft (Fig. 2).

Discussion

ACF following AAA rupture into the vena cava causes extensive haemodynamic disturbances with an increase in venous volume, pressure and cardiac preload. The hyperdynamic circulation is characterised
by elevation of the stroke volume, the heart rate, and the cardiac output, inevitably leading to intractable cardiac failure. The reduction of renal arterial perfusion pressure, combined with an increase in venous pressure, causes a decrease in the glomerular filtration rate.\textsuperscript{1,2} The oedema and cyanosis of the lower limbs is due to a distal shunting of the ACF, which has been demonstrated experimentally in dogs.\textsuperscript{3} The “arterialisation” of the pelvic veins is responsible for venous congestion of the urinary bladder. These distended and fragile veins may finally rupture and cause massive haematuria.\textsuperscript{3}

The classic symptoms of ACF – abdominal or back pain, palpable aneurysm, abdominal bruit, congestive heart failure with high output state, shortness of breath, lower limb oedema and pelvic congestion with haematuria – is uncommon. In consequence, even in experienced centres a correct preoperative diagnosis is made only in 20–50% of patients with ACF.\textsuperscript{2,4}

Severe and persistent haematuria should be considered a significant diagnostic criterion for an ACF. In a literature review, Brewster \textit{et al.}\textsuperscript{3} found that haematuria is usually not associated with symptomatic AAA. In 1000 patients with AAA, he found only one case of haematuria caused by a ureteral tumour. Conversely, haematuria has been reported in 17–100\%\textsuperscript{2–4} of patients with an ACF due to a ruptured AAA. In consequence, haematuria in patients with AAA should be regarded as due to ACF until proved otherwise.

Fig. 2. Follow-up CT scan 2 years postoperatively showing a patent inferior vena cava and bifurcation graft.

References


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