A 71-year-old man was referred to our intensive care unit for short-term dyspnea associated with hemodynamic instability (systolic blood pressure, 75 mm Hg; heart rate, 115 bpm). He had a history of hypertension, hypercholesterolemia, and intermittent claudication with a maximal walking distance of 50 m and had been smoking 20 cigarettes daily for 30 years.

Immediate clinical examination revealed hypotension, hypoxemia, warm extremities, and normothermia. Pulmonary auscultation revealed end-inspiratory crackles. Abdominal physical examination, difficult because of morbid obesity, did not reveal any abnormality. A chest x-ray performed before any fluid resuscitation depicted a pulmonary edema and pleural effusion. Initial white cell count and C-reactive protein level were both elevated (17 000/mm$^3$ and 74 mg/L). Other blood tests revealed sudden renal failure and metabolic acidosis. Troponin level was elevated (3.24 mmol/L), but the ECG was normal. These findings led the physicians to suspect septic shock of pulmonary origin. Echocardiography revealed high cardiac output (10 L/min) with preserved left ventricular ejection fraction (65%) and pulmonary hypertension (45 mm Hg). After intubation, the patient underwent fluid resuscitation and received continuous intravenous epinephrin infusion. However, this treatment failed to correct hypotension.

Six hours later, the patient presented a critical left leg ischemia. An urgent computed tomography revealed a 90-mm left common iliac artery aneurysm (Figure [A and B, solid arrow]), which had ruptured into the left common iliac vein (Figure [B, black arrow]). This diagnosis was suggested by simultaneous enhancement of the inferior vena cava (Figure [A and B, dotted arrow]).

The diagnosis of septic shock was rectified to high-output heart failure because of cardiogenic pulmonary edema associated with warm extremities, preserved left ventricular function, high cardiac index, and arteriovenous fistula. The latter was excluded by deploying a right aorto-uni-iliac stent graft associated with an embolization of the left internal iliac and the common left iliac artery. A right-to-left femoral-femoral bypass was then realized (Figure [C]). By the end of the intervention, cardiac output returned to normal (6.4 L/min). The patient remains stable at 3 months.

Spontaneous rupture of an abdominal aortic or iliac aneurysm into the inferior vena cava or the iliac vein is a rare complication with a reported overall prevalence of 3% to 6% of all ruptured aortic aneurysms. It is the most common cause of major abdominal arteriovenous fistula. Iatrogenic injury after lumbar disk surgery and trauma have been responsible for <20% of the cases.

The mortality for open surgical repair is 5% to 66%. Significant postoperative morbidities occur in 11% to 39% of surgical corrections of iliac arteriovenous fistulas. Associated local complications include limb ischemia, gangrene, and limb loss; systemic complications include pulmonary embolus, caval thrombosis, venous stasis, and severe bleeding. Endovascular approaches may be preferred over open operation.

Disclosures

None.

References

Figure. A, Computed tomography angiogram shows early and unusual simultaneous enhancement of the vena cava (dotted arrow) above the aneurysm (solid arrow) that had ruptured into the left common iliac vein. There is no rupture into the abdominal cavity. B, CT scan showing communication between the aneurysm (solid arrow) and the left common iliac vein (black arrow). Dotted arrow indicates the vena cava. C, Reconstruction of the computed tomography angiogram after surgery showing total exclusion of the iliac aneurysm by the endograft and coil. The left leg is vascularized by the femoral-femoral bypass.
High-Output Heart Failure
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