

# An Unusual Presentation of Aorto-caval Fistula

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## Abstract

We report a case of a 77 year-old male who presented with symptoms and signs consistent with congestive cardiac failure and concomitant acute lower respiratory tract infection. He failed to respond to conventional medical management and became anuric prompting further investigation. Radiological imaging confirmed an infra-renal abdominal aortic aneurysm (AAA). Prior to surgery delayed onset of anaesthesia was noted at induction and the patient required large doses of inducing agents and muscle relaxants to achieve adequate anaesthesia. This was thought to indicate the presence of an aorto-caval fistula (ACF). Indeed at operation an inflammatory AAA with a concomitant ACF was noted. We suggest that the delayed onset of anaesthesia during induction may indicate the presence of an ACF in patients with an AAA.

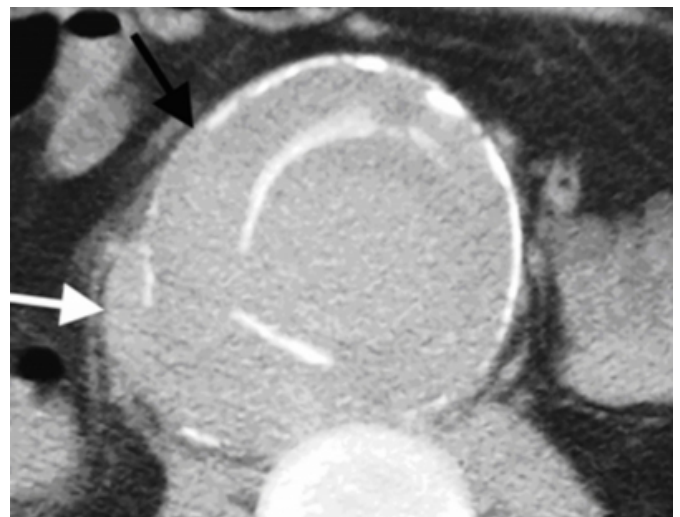
## CASE HISTORY

A 77-year-old male presented with a 3 day history of a productive cough and fever. He had no significant past medical history and had had no previous surgery. He had a weight of 70 kg. Examination revealed a temperature 37.1°C, pulse 110/min regular, respiratory rate 20/min, blood pressure 90/50mmHg and SpO<sub>2</sub> 92% on air. The patient had dry mucosal membranes and reduced skin turgor. Cardio-respiratory examination revealed bibasal inspiratory crepitations more prominent at the right base and bilateral pitting pedal oedema. Abdominal examination revealed no abnormalities. All peripheral pulses were present and were normal in character. There was no lower limb venous congestion noted. Haematological investigations showed a WCC 19.5x10<sup>9</sup>/l. Blood biochemistry was within normal parameters but C-Reactive Protein (CRP) was raised at 223mg/l. Urinalysis revealed no abnormality. Electrocardiogram (ECG) showed no acute changes. Plain chest radiography revealed bilateral basal pulmonary shadowing more prominent the right lung base. A provisional diagnosis of congestive cardiac failure with concomitant right basal pneumonia was made. The patient was commenced upon diuretics and intravenous antibiotics. Subsequent sputum cultures were negative. 24 hours after admission the patient became anuric and renal function deteriorated (urea 17.2mM/l and creatinine 177uM/l). Emergent abdominal ultrasound revealed an 8 cm abdominal aortic aneurysm (AAA). Haemodynamically the patient had remained stable (pulse 109/min, BP 139/79 and SpO<sub>2</sub> 96%

on air) and so a non-contrast CT scan of the abdomen was performed. This confirmed an 11 cm infra-renal AAA with intra-peritoneal fluid suggesting that it had ruptured (Figure 1). The patient was consented and prepared for emergency laparotomy.

## Figure 1

Figure 1: Non-enhanced abdominal CT showing an 11 cm infra-renal inflammatory AAA with the inferior vena cava adherent to its medial wall (arrow).



At induction, 20 mg etomidate and 40 mg rocuronium were administered. This failed to achieve adequate anaesthesia or muscle relaxation; the patient continued to move making intubation impossible. He required further 100 mg propofol and 80 mg rocuronium for successful intubation and ventilation. Central venous pressure (CVP) was recorded at

>25cm H<sub>2</sub>O during induction. Arterial line reading showed a blood pressure of 140/29. At laparotomy a non-leaking infra-renal inflammatory AAA was noted. On opening the aneurysmal sac, a 2.5 cm proximal aorto-caval fistula (ACF) was found. Venous bleeding was controlled with digital pressure and the fistula repaired. CVP returned to normal immediately after closure of ACF. The AAA was repaired with straight dacron graft. Renal function returned to normal after post-operative diuresis. The patient made an uneventful post-operative recovery and was discharged home after five days. At one year follow up he is well and symptom free.

### DISCUSSION

The incidence of ACF within an inflammatory AAA is estimated to be 17% [1]. Surgery is indicated in all cases as survival without operation is <2 months [2]. The formation of ACF is attributed to intense peri-aortic inflammation leading to adhesions with the adjacent inferior vena cava (IVC) and subsequent pressure necrosis of the caval wall [3]. Rupture into the IVC remains a rare event [4]. The precise presentation of ACF depends upon the rapidity of fistulisation and the size of fistula.

Due to its rarity and elusive clinical presentation the diagnosis of ACF can be easily missed. The characteristic triad of clinical findings in an ACF are a palpable AAA, machinery abdominal murmur and bruit is diagnostic [5]. However as in the reported patient the murmur may be absent if the fistula has been closed by intra-mural thrombus. There is a variable association of ACF with congestive cardiac failure, lower limb venous hypertension and haematuria [6,7]. Half of patients with an ACF present with high output hyperdynamic circulation with a widened pulse pressure and relatively low diastolic pressure [4]. In our patient this characteristic blood pressure was seen only at anaesthetic induction. In retrospect the significance of this finding was missed at the time.

In ACF fluctuations in haemodynamic status are as a result of the arterio-venous (AV) fistula diverting blood flow from the high resistance arterial circuit to the low resistance and high capacitance venous circuit. The resultant decrease in total peripheral resistance and subsequent increase in venous pressure, resistance and volume leads to acute pulmonary oedema as seen in the reported patient. This was further complicated in our patient by the development of a right basal pneumonia. There is therefore a resultant increase in heart rate, stroke volume and cardiac output. If the ACF persists the myocardium hypertrophies and can dilate and

lead to irreversible hyperdynamic cardiac failure. It is important to note as in the reported case, cardiac failure secondary to ACF is refractory to medical treatment [4]. As blood is diverted through the fistula, arterial perfusion distal to the fistula is reduced. There is an increase in renal venous pressure and decreased renal arterial perfusion pressure. The rennin-angiotensin system is activated by decreased distal perfusion and reduced renal arterial perfusion pressure. There is an increase in aldosterone secretion leading to plasma expansion and increased perfusion. A Swan-Ganz catheter may show a high cardiac index, increased stroke volume, decreased systemic vascular resistance and increased concentration of oxygen in the IVC at this stage [4].

Decompensated cardiac failure due to increased venous return occurs in 35% of patients [5]. In such patients, haematuria, acute renal failure may occur as a result of a renal infarction due to renal arterial problems, or of renal congestion due to a perforation of an AAA into the renal vein [5]. Furthermore pre-renal failure due to heart failure and lowered arterial blood pressure lead to lowered renal pressure [5]. Our patient was anuric and renal function impaired due to the above mechanisms. The resultant oliguria and/or anuria seen in patients is not due to hypovolemia but the re-distribution of circulating blood volume. The large proximal central AV fistula shunts the cardiac output back to the heart and this explains the delayed onset of anaesthesia in our patient because the drugs were circulating in a closed circuit with little peripheral distribution.

Both clinical examination and non-enhanced CT scan were unable to diagnose ACF prior to surgery. Contrast CT and/or aortography can show early flush of contrast in the IVC from the adjacent AAA indicating ACF [9]. However in our patient the use of contrast is limited due to renal dysfunction making diagnosis difficult. We suggest that in patients where the IVC is found adherent to an inflammatory AAA, either radiologically or at operation, and delayed onset of anaesthesia is noted the possibility of an ACF should be considered.

### SUMMARY

A high degree of suspicion is required for preoperative diagnosis of inflammatory AAA with concomitant ACF. The delayed onset of anaesthesia during induction using increased doses of inducing agents in a patient with an AAA should alert the anaesthesiologist and vascular surgeon to the

possibility of ACF.

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