

Aortocaval Fistula Presenting as Type 2 Acute Myocardial Infarction in a Patient with Severe Arteriopathy

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Abstract

Aortocaval fistulas (ACFs) are rare with varied etiologies. Symptoms can be acute or delayed with predominant manifestations being high output cardiac failure. Acute coronary syndrome due to ACF has not been widely reported. We present a case of a 68-year-old male who presented with signs and symptoms suggestive of acute coronary syndrome. This was confirmed by electrocardiogram changes and a rise in cardiac enzymes. A large abdominal aortic aneurysm was diagnosed initially by imaging without evidence of leak or rupture. A coronary angiogram showed only mild diffuse disease. On further reviewing, the computerized tomography imaging revealed an ACF. This was subsequently repaired with rapid improvement in his condition. Acute coronary syndrome is an unusual presentation of ACF with inadequately understood pathophysiological mechanisms. Prompt diagnosis and surgical management of this fistula are paramount to reduce mortality and morbidity.

Keywords: Abdominal aortic aneurysms, aortocaval fistula, myocardial infarction

INTRODUCTION

Aortocaval fistula (ACF) is a rare presentation of abdominal aortic aneurysms (AAAs). Most of them are spontaneous and fistulization occur commonly with inferior vena cava, iliac, or renal veins. A majority of those affected are males, in their seventh or eight decades of life.

Although the common presentations are that of high output cardiac failure or venous hypertension, we present a unique and uncommon case of ACF which presented as Type 2 acute myocardial infarction.

CASE REPORT

A 68-year-old male presented to a country hospital with dizziness and diaphoresis. His past medical history included hypertension, hypercholesterolemia, an extensive smoking history, and mild thrombocytopenia of uncertain etiology. Electrocardiogram (ECG) showed widespread ST depression in leads II, III, aVF, and V3 to V6 with ST elevation in aVR [Figure 1]. Initial Troponin T value was <40 ng/L; however, repeat assay 6 h later was elevated at 1313 ng/L. He was hypotensive at presentation, necessitating the use of vasoactive supports for transfer to our quaternary hospital

for the management of presumed acute coronary syndrome complicated by cardiogenic shock.

Shortly following retrieval to our center, he reported acute back pain. He had a heart rate of 109/min, oxygen saturations of 94% on 10 l oxygen through a nonrebreather mask, blood pressure of 108/62 mmHg on vasopressors, and his peripheries were cool. A focused ultrasound scan was suggestive of an AAA. Urgent computerized tomography (CT) scan with contrast subsequently confirmed the presence of an infrarenal AAA involving the iliac arteries, 95 mm at maximal axial diameter [Figure 2]. There was no evidence of leak. Bedside urgent echocardiography showed mild-to-moderate left ventricular (LV) dysfunction without any valve abnormalities.

An urgent coronary angiogram through right radial artery approach showed only mild diffuse disease in the mid-left anterior descending and right coronary arteries. The patient's clinical condition remained unchanged. At further review of the CT imaging, a fistulous communication between the inferior

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vena cava and the aneurysmal dilatation of the aorta at the L5 level was identified [Figure 3]. This prompted transfer to the operating theater for urgent open repair of the ACF.

Following midline laparotomy, a large nonruptured AAA was identified. The aorta was atheromatous and extensively calcified. The intraoperative course was complicated by major blood loss necessitating massive blood transfusion. The patient suffered hypovolemic arrest at the time of sac incision, with return of spontaneous circulation after five cycles of cardiopulmonary resuscitation.

Following successful repair of the ACF, the patient's hemodynamic status rapidly improved. Vasopressors were ceased by about 4 h, and he was extubated 6 h later without evidence of neurological deficit related to the period of hypoperfusion. ECG changes reverted to normal [Figure 4]. The patient developed acute kidney injury; however, urine output was maintained. The patient was discharged from the Intensive Care Unit after a 24 h period of observation. He was discharged from the hospital 1 week later. Follow-up investigation showed resolution of the acute kidney injury [Table 1]. The pre-morbidly noted thrombocytopenia also resolved. Three months following his acute presentation,

completion of the SF-30 questionnaire suggested a reduction in physical functioning to 70% of baseline.

DISCUSSION

ACF is a widely reported but uncommon complication of AAA, with series suggesting an incidence between 0.2% and 10%.^[1,2] They are often diagnosed intraoperatively,^[3] where repair is associated with higher mortality compared with uncomplicated AAA.^[4] This is possibly due to the well-recognized association with intraoperative cardiac arrest.^[5] While rupture of AAA is the most common cause of ACF (80%), they may also occur following penetrating trauma (15%) or complicate mycotic aneurysms, large vessel vasculitis, or develop iatrogenically, for example, as a complication of lumbar disc surgery.^[6]

Classical clinical findings include pulsatile abdominal mass with an audible bruit, high output cardiac failure, and regional venous hypertension.^[7-9] Chest pain, lower extremity edema, priapism, rectal bleeding and hematuria, renal failure, intestinal angina, and intermittent claudication have also been reported as presenting features.^[3]

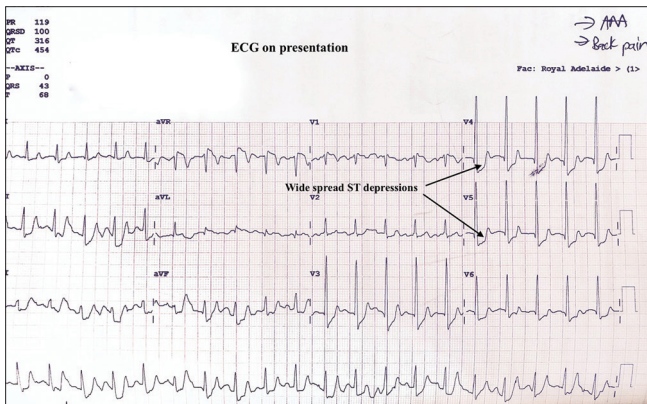


Figure 1: Electrocardiogram on presentation

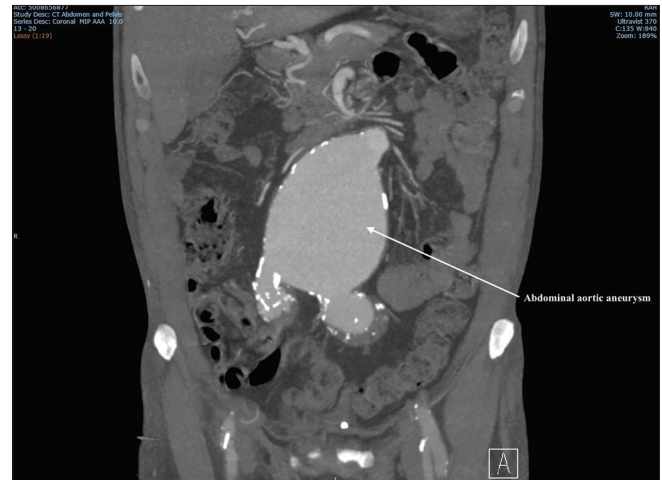


Figure 2: Coronal section of computerized tomography abdomen showing the abdominal aortic aneurysm

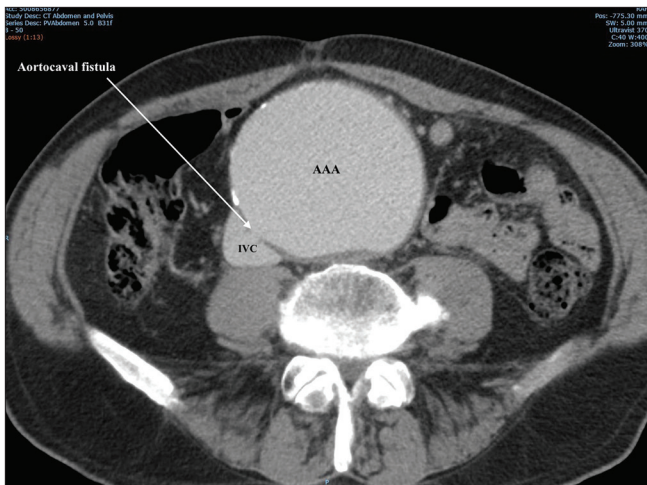


Figure 3: Axial section of computerized tomography abdomen showing the aortocaval fistula

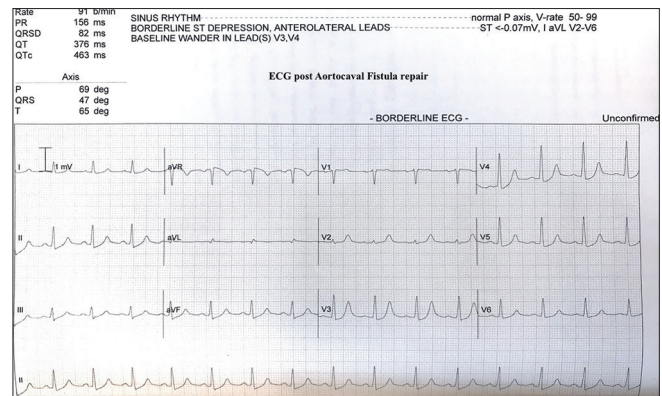


Figure 4: Electrocardiogram postfistula repair showing resolution of changes

Table 1: Trend in key variables around the time of presentation

	Premorbid	Presentation	6	12 h (PEA arrest in OT)	18 h	Day 2	Day 7
Troponin T (<30 ng/L)	-	<40	1313	1439	2505	-	-
Creatinine (50-120 umol/L)	105	-	232	231	220	169	74

PEA: Pulseless electrical activity; OT: operation theatre

Acute coronary syndrome due to ACF is very rare; to the authors' knowledge, this is only the second case that has been described in the literature. Acute volume overload of the left ventricle could explain such a presentation.^[10] Animal studies have shown low systemic blood pressure and elevated LV end-diastolic pressure in the setting of ACF.^[11] The resulting shunt to the low-pressure caval system reduces coronary driving pressures during diastole, precipitating LV systolic and diastolic dysfunction.^[11] In other animal models, subendocardial fibrosis was seen in the infarcted and noninfarcted myocardium as a result of low coronary driving pressure.^[12] The presence of LV hypertrophy and mild coronary artery disease could aggravate the ischemic myocardial state precipitated by ACF.

Early diagnosis of ACF is challenging due to the wide scope of clinical presentations. Surgery, or endovascular treatment in selected cases, is the only chance of survival for these patients.^[13]

CONCLUSION

This case highlights the value of careful review of imaging where incongruous objective findings (such as the normal coronary angiogram) come into conflict with the clinical picture. We often rely on the maxim, "common things occur commonly." In this instance, the very common clinical entity of non-ST elevation myocardial infarction was precipitated by ACF through a very uncommon pathophysiological mechanism.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that name and initial will not be published, and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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