Severe left anterior descending artery stenosis with proximal arteriovenous malformation presenting as acute myocardial infarction

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ABSTRACT

Congenital coronary artery anomalies are rare, with an incidence of about 0.06–1.3 percent of all patients undergoing cardiac catheterisation. They are commonly asymptomatic, but potentially serious lesions may lead to myocardial ischaemia, infarction and/or sudden cardiac death. The occurrence of a concomitant stenotic lesion is exceedingly rare. We report an 80-year-old man who presented with acute anterior myocardial infarction. Coronary angiography revealed severe proximal left anterior descending (LAD) and arteriovenous malformation (AVM) from the first septal branch of the LAD. The LAD stenosis and the AVM were successfully treated with two Jomed® covered stents.

Keywords: acute anterior myocardial infarction, arteriovenous malformation, congenital coronary artery anomalies, left anterior descending artery, myocardial infarct

INTRODUCTION

Coronary artery anomalies are rare, occurring in 0.06%–1.3% of patients undergoing cardiac angiography.[1,2] These anomalies are predominantly asymptomatic, but may lead to myocardial ischaemia, infarction and sudden cardiac death if the lesions are progressive. The outcome is dependent upon the origin, course and termination site of these anomalous vessels.[2] Communications between the coronary arteries and other vessels are often due to deviations from normal embryological development. They may occur as a result of trauma, invasive cardiac procedures such as pacemaker implantation, endomyocardial biopsy or coronary angiography.[3] Most coronary artery fistulae are small with no myocardial blood flow compromise and therefore, patients remain asymptomatic.

However, there may be coronary artery steal, causing ischaemia of the segment of myocardium perfused by the coronary artery distal to the fistula. The presence of a coronary artery stenosis distal to the arteriovenous
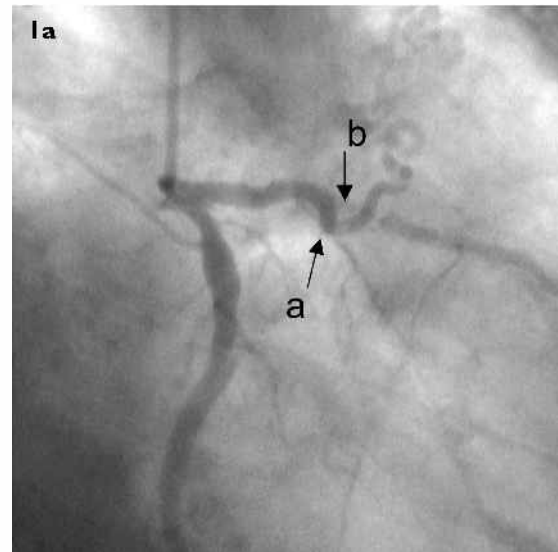
Fig. Ia RAO caudal view coronary angiogram shows an AVM arising from the first septal branch of the LAD associated with severe proximal LAD stenosis (a), and just distal to the AVM (b).
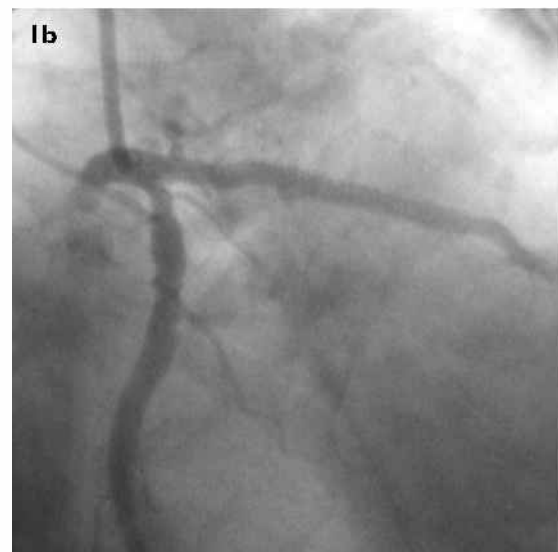
Fig. Ib RAO caudal view coronary angiogram following deployment of two Jomed® covered stents shows obliteration of the first septal branch and AVM.

malformation (AVM) will exacerbate the coronary artery steal. We present an 80-year-old man who was found to have an AVM proximal to a severe left anterior descending (LAD) stenosis successfully treated with covered stent placement across both lesions.

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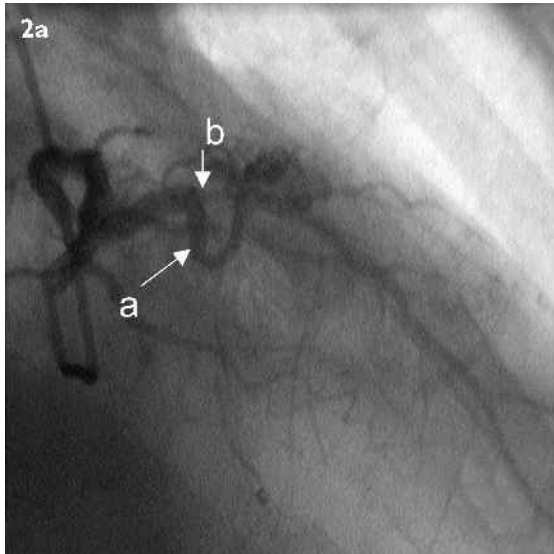
Fig. 2a AP cranial view coronary angiogram shows an AVM arising from the first septal branch of the LAD associated with severe proximal LAD stenosis (a), and just distal to the AVM (b).

Fig 2b AP cranial view coronary angiogram following deployment of two Jomed® covered stents shows obliteration of the first septal branch and AVM.

CASE REPORT

Our patient is an 80-year-old man who initially presented to a peripheral hospital with recurrent chest discomfort and exertional dyspnoea. He had a history of gouty arthritis and gastritis under the peripheral hospital follow-up. He was a frequent user of over-the-counter analgesics which he readily had access to at pharmacies. Although there was no previous history of chest pain, syncopal or presyncopal attacks, he had reduced effort tolerance on walking up to 20 metres and orthopnoea for the past one year. He was an ex-smoker who quit 20 years ago. There was no family history of ischaemic heart disease or sudden death. Physical examination revealed that he was alert and not tachypnoeic. The jugular venous pressure was not raised but he had bibasal lung crepitations. His

blood pressure was 100/50 mmHg and heart rate was 70/ min. Peripheral pulses were equal. Precordial examination revealed dual rhythm with no murmur. Per abdomen was soft, non-tender with no organomegaly.

Blood investigation showed renal impairment with sodium 135 mmol/L, potassium 5.6 umol/L, urea 9.2 mmol/L and creatinine of 123 mmol/L. This could be due to underlying analgesic nephropathy. He was mildly anaemic with haemoglobin of 11.2 g/dL and MCV 88 /fl which could be explained by underlying chronic kidney disease. He had mild dyslipidaemia with total cholesterol of 5.2 mmol/L, LDL 3.2 mmol/L, HDL 1.2 mmol/L and triglycerides 1.0 mmol/L. His sugar profile was normal with a random blood glucose of 6.9 mmol/L. Creatinine kinase was 1,247 u/L and Troponin T was 0.49 ng/ml. Electrocadiogram showed a sinus rhythm, with poor R wave progression at anterior leads. Echocardiogram revealed left ventricular hypokinesia with an ejection fraction of 35%–40%. There was moderate LV diastolic dysfunction and a dilated left atrium and ventricle.

Coronary angiogram showed normal left main stem (LMS), severe proximal LAD 80% stenosis after the first septal branch (Figs. 1a & 2a) and AVM from the first septal branch of LAD. The circumflex and right coronary arteries were only mildly diseased, with no flow-limiting stenoses. The patient subsequently underwent percutaneous coronary stenting of the LAD. The LAD lesion was stented with two Jomed® (Jomed International AB, Helsingborg, Sweden) covered stents (3.5 mm × 18 mm distally and 4.0 mm × 9 mm proximally). Post-stenting angiography showed disappearance of the AVM with stent coverage over both the proximal septal branch and proximal LAD stenosis (Figs. 2a & b). The patient was completely asymptomatic with good effort tolerance on review three months later.

DISCUSSION

Communications between the coronary arteries and the cardiac chambers (coronary-cameral fistulae) or other vessels (coronary artery arteriovenous malformations) are often due to deviations from normal embryological development.[3] The coronary artery proximal to the fistula enlarges due to a compensatory mechanism. This is related to the diastolic pressure gradient and run-off from the coronary arteries to a low-pressure receiving cavity such as the right atrium.[4] If the fistula is large, the intracoronary diastolic perfusion pressure reduces progressively. Over time, progressive dilation of the fistula tract may progress to frank aneurysm formation, intimal ulceration, medial degeneration, intimal rupture, atherosclerotic deposition, calcification, side-branch obstruction, mural thrombosis and rupture.[4]

The outcome depends on the site of origin and termination of the abnormal connection and the size of

the connection.[3,4] Common sites of origin are the right coronary artery (55%), the left coronary artery system (35%) and both coronary arteries (5%).[5] The right side of the heart is the most common termination site, occurring in 90% of cases.[4] Less frequently, it may drain into the superior vena cava or coronary sinus, and least often to the left atrium or left ventricle. If left untreated, haemodynamically significant fistulae may cause symptoms or sequelae in approximately 11% of patients under the age of 20 years, and 35% of those over the age of 20 years.[4]

Surgical closures are associated with low mortality and morbidity; the long-term outcome is excellent and most patients remain asymptomatic.[6] Despite the good results with surgery, closure percutaneously has become the method of choice. In view of the natural progression in larger fistulae to dilate over time, with a progressively increasing risk of thrombosis, endocarditis, or rupture, the general advice is to close all but the small fistulous connections.[4-6] Various percutaneous catheter techniques have been employed, including thrombogenic coils that promote vascular occlusion such as Gianturco coils and interlocking detachable coils, direct occlusion with detachable balloons and polyvinyl alcohol foam as well as more specific devices such as double umbrellas and the Amplatzer duct occluders.[7]

Another mode of treatment is percutaneous angioplasty with covered stent. Covered stent has been well-documented for the exclusion of various aneurysms, treatment in coronary artery rupture and treatment of coronary aneurysm.[8,9] The long-term outcome of patients treated with a covered stent for aneurysmal disease is not known. There are still risks of subacute stent thrombosis and stent re-stenosis. For our patient, stenting the proximal LAD stenotic lesion with either a bare metal or drug-eluting stent would have afforded the same outcome in terms of coronary revascularisation. However, we opted for two Jomed® covered stents as this would tackle both the proximal septal AVM and distally, the proximal LAD stenosis at the same time. Prognostically, successful revascularisation of the LAD either percutaneously or surgically would afford a good outcome. Whether the implantation of the covered stents would afford equivalent outcomes compared to more "conventional" stents remain to be seen and should be the subject of further trials.

In conclusion, coronary AVMs are rare and the symptoms depend on the site of origin/course of the vessel. If symptomatic, the standard management would be surgical or percutaneous closure. However, other factors also affect the course of management, for example as demonstrated in this case, the advanced age of the patient, acute presentation as myocardial infarction and concomitant severe proximal LAD stenosis. Good clinical judgment should always be exercised in dealing with such anomalies and evidence-based medicine (which is clearly lacking in this case) should never be a substitute for clinical acumen.

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