Case Reports

Mycotic Aneurysm of the Left Anterior Descending Coronary Artery after Aortic Endocarditis

A Case Report and Brief Review of the Literature

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This report concerns a 29-year-old man with recent Streptococcus viridans *endocarditis on a bicuspid aortic valve who was found to have a mycotic aneurysm of the left anterior descending coronary artery and infective erosion and thinning of the posterior wall of the ascending aorta 1.5 to 3.5 cm above the origin of the left coronary artery, a combination of lesions not previously reported.*

Mycotic aneurysm of the coronary arteries affects less than 1% of patients with infective endocarditis, and there are few reports of the management of these rare lesions.

The surgical management of this patient is presented with a brief review of the available literature. ***(Texas Heart Institute Journal 1994;21:231-5)***

Mycotic aneurysm is found in life or at autopsy in 3% to 15% of patients with infective endocarditis.[1] However, the great majority of such lesions involve the aortic root or sinuses of Valsalva (25%), visceral arteries (24%), arteries to the extremities (22%), and cerebral arteries (15%).[1,2] This indicates that coronary artery mycotic aneurysm occurs in less than 10% of such patients (less than 0.5% of all infective endocarditis patients) and is in all probability extremely rare. (It must be added that many mycotic aneurysms remain undiagnosed because they most often are asymptomatic and clinically silent.[3]) As a consequence, there are few reports of value in the operative management of these lesions.

This report concerns a patient with recent endocarditis on a bicuspid aortic valve who was found to have a mycotic aneurysm of the left anterior descending coronary artery and infective erosion of the posterior wall of the ascending aorta above the origin of the left coronary artery, a combination of lesions not previously reported.

Key words: *Aneurysm, infected; coronary aneurysm; endocarditis, bacterial; streptococcal infections/ complications*

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Case Report

History

A 29-year-old man of Indian origin presented at another hospital with a 4-day history of fever, rigors, dizziness, dyspnea on exertion, and palpitation. Until this episode, he had been very fit and free of cardiac symptoms, although he did relate that 5 years earlier he had been rejected for army service due to "a weakness of a heart valve." At that time he had been advised to take regular injections of penicillin, but had failed to comply. There was no recent history of dental treatment or bacterial infection.

Examination revealed a thin, pale, ill-looking man with a pyrexia of 38.4 °C, blood pressure of 104/80 mmHg, and pulse rate of 120 beats per minute. There was a coarse systolic thrill over the left precordium, and a grade 4/6 ejection murmur was audible over the entire precordium, radiating to the left carotid artery. Palpation showed left ventricular dilatation, but normal jugular venous pressure. The spleen was palpable 2 cm below the costal margin, but there were no cutaneous or ungual stigmata of endocarditis.

The hemoglobin concentration was 11.8 g/dL, the white blood cell count 10.6 x 10^9/L, and the erythrocyte sedimentation rate 60 seconds. Three blood cultures taken over a 24-hour period all grew *Streptococcus viridans*. Liver and renal function tests were within normal limits. Intravenous gentamicin and penicillin were started, and the patient became afebrile within 72 hours.

Echocardiographic assessment showed the following: a thickened, bicuspid aortic valve with a transvalvular gradient of 100 mmHg, trivial aortic regurgitation, and a small vegetation present on the "anterior" leaflet. The mitral and tricuspid valves were normal. There was concentric left ventricular hypertrophy with an ejection fraction of 55%. Electrocardiography showed left ventricular hypertrophy, with strain and sinus rhythm.

Gentamicin treatment was discontinued after 2 weeks, and penicillin after 3 weeks. The only complication was left basal pulmonary collapse associated with pleuritic chest pain, which resolved without additional treatment. The patient was discharged in good condition 28 days after presentation, with no signs of residual infection. Prescribed medications were acetyl salicylic acid (300 mg per day) and cloxacillin sodium (500 mg taken orally 4 times per day), for 1 week.

Our Experience with the Patient

Eleven days later, the patient presented himself at our institution, and was admitted for assessment. The physical findings were as before, although the tachycardia had settled and the blood pressure was higher, at 110/80 mmHg. Investigations revealed a raised white blood cell count of 11.3 x 10^9/L, an elevated alkaline phosphatase level of 128 (normal, <117), and an elevated serum creatinine level of 106 (normal, <97). Blood cultures and other laboratory investigations were normal, apart from a persistently raised erythrocyte sedimentation rate. Further echocardiographic assessment confirmed that the vegetation on the aortic valve was still present.

Cardiac catheterization showed normal right-sided pressures, apart from a moderately elevated mean pulmonary wedge pressure of 16 mmHg. The transaortic gradient was 100 mmHg. The left ventricular systolic pressure was 204 mmHg and the end-diastolic pressure 24 mmHg. The cardiac output was 6.4 L/min, and the cardiac index 3.31 L/min/m^2.

Coronary arteriography (performed because of the patient's history of high cigarette consumption and our knowledge of a high rate of coronary artery disease among local natives of India) showed a 1.5-cm diameter aneurysm of the left anterior descending coronary artery (LAD) at the junction of its middle and distal thirds, with restricted flow into the distal LAD and compromise of the origin of a small 2nd diagonal branch (Figs. 1 and 2). Upon contrast injection, the aneurysm was seen to fill rapidly and to empty slowly.

In view of the presence of aortic valve vegetation, a significant aortic valve gradient, and restricted blood flow into the distal LAD associated with the aneurysm, surgery was advised.

The operation was performed using cardiopulmonary bypass, with moderate hypothermia (28 °C) and cold antegrade blood cardioplegia for myocardial protection. The mycotic aneurysm was visible on the epicardial surface of the heart (Fig. 3A). The aortic valve was bicuspid and thickened, with ulcer-

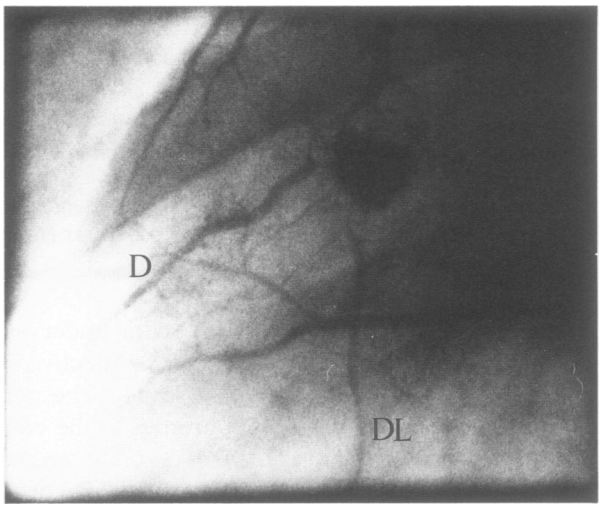

Fig. 1 *Left coronary arteriogram, showing impaired flow into both the distal left anterior descending coronary artery (LAD) and the 2nd diagonal branch.*

DL = distal LAD; D = 2nd diagonal coronary artery

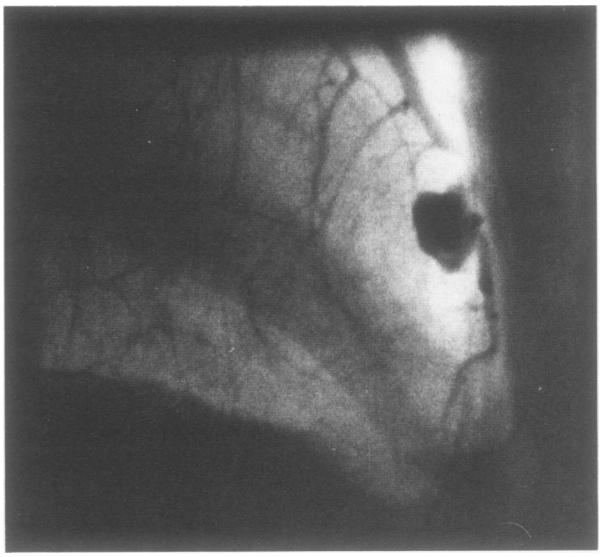

Fig. 2 *Lateral view of vessels seen in Figure 1. Note the extent of the aneurysm and the impaired flow from it.*

ation and vegetations of the anterior leaflet. On the posterior wall of the ascending aorta, 1.5 cm above the origin of the left coronary artery, there was an oval area, 2 cm in diameter, which was denuded of endothelium, was thinned by 1 to 2 mm, and had fronded vegetations on its proximal rim.

The aortic valve was excised. The LAD aneurysm was opened in the line of the artery and was found to contain fresh mural thrombus. This was removed. Neither the walls of the aneurysm nor the LAD above and below appeared to be acutely inflamed. The origin of the small diagonal vessel was oversewn in the wall of the aneurysm, and a short segment of reversed saphenous vein was sewn into place between the proximal and distal ends of the LAD, which were approximately 3.5 cm apart (Figs. 3B and C).

The aortic valve was then replaced, using interrupted horizontal mattress sutures, with a 24-mm Jyros bileaflet prosthesis (Manley-Western [UK] Ltd.; Chichester, West Sussex, England), which had been soaking in vancomycin. A double-velour Dacron patch was sewn to the margins of the defect in the posterior aortic wall, after removal of the vegetations. Recovery was uneventful. Cultures of the aortic valve, vegetations, aneurysm thrombus, and aneurysm wall were sterile. Prophylaxis with cefamandole and intravenous vancomycin (1 g twice daily) was discontinued after 4 days, and intravenous penicillin was discontinued after 1 week. Angiography of the LAD was performed on the 24th postoperative day (Fig. 4) and showed patency of the vein graft, with normal flow into the distal portion of the vessel.

Comment

Coronary artery aneurysm may be congenital, affecting males and the right coronary artery more frequently.[4] Most acquired aneurysms are secondary to Kawasaki disease in childhood,[5] and to coronary atherosclerosis in adulthood.[4,6]

Other documented causes are dissection, trauma following percutaneous coronary angioplasty, vasculitis, and syphilis.[4,6] Many of these aneurysms are clinically silent, and therefore remain undetected

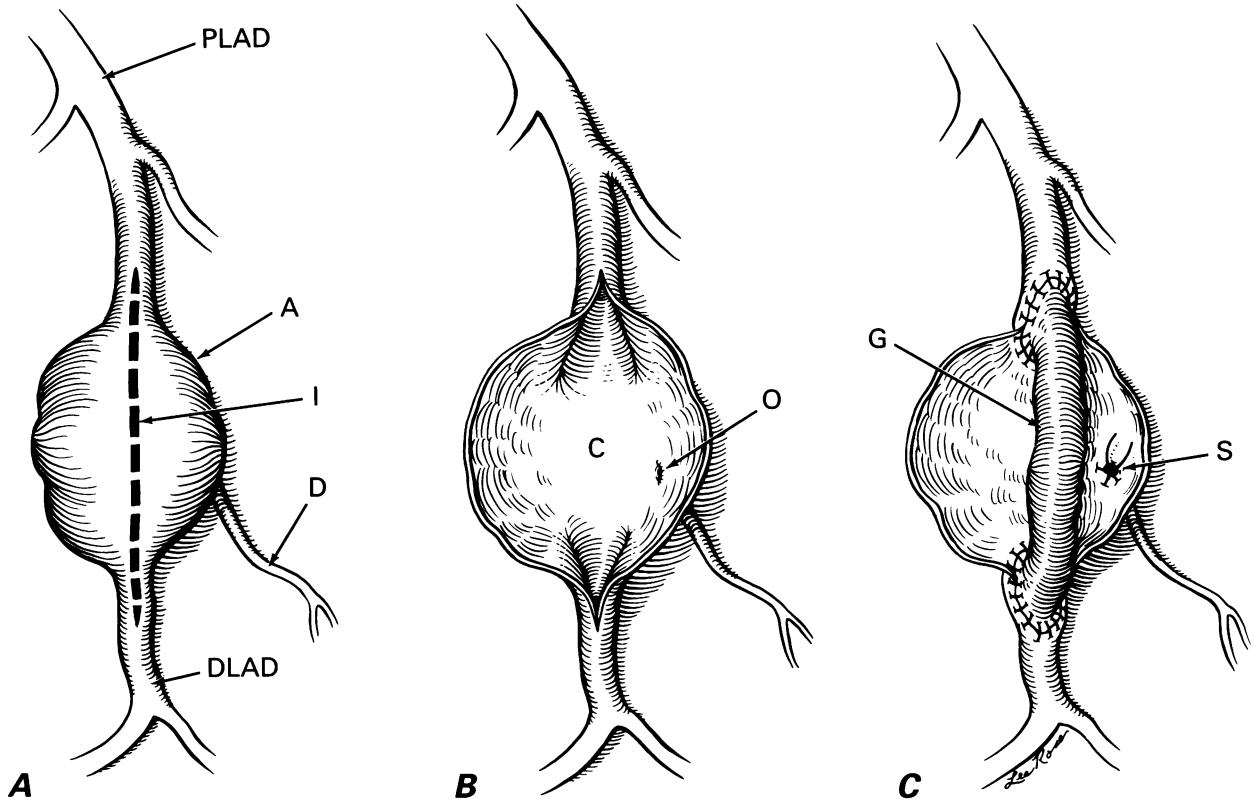

Fig. 3 ***A)*** *As seen before the aneurysm was opened.* ***B)*** *As seen after the aneurysm was opened and the thrombus removed. Note the origin of the 2nd diagonal coronary artery in the fibrous wall.* ***C)*** *Interposition graft in place.*

A = aneurysm; C = cavity of aneurysm; D = 2nd diagonal coronary artery; DLAD = distal left anterior descending coronary artery; I = line of incision into aneurysmal cavity; O = origin; PLAD = proximal left anterior descending coronary artery; S = suture closure of the origin of the 2nd diagonal coronary artery

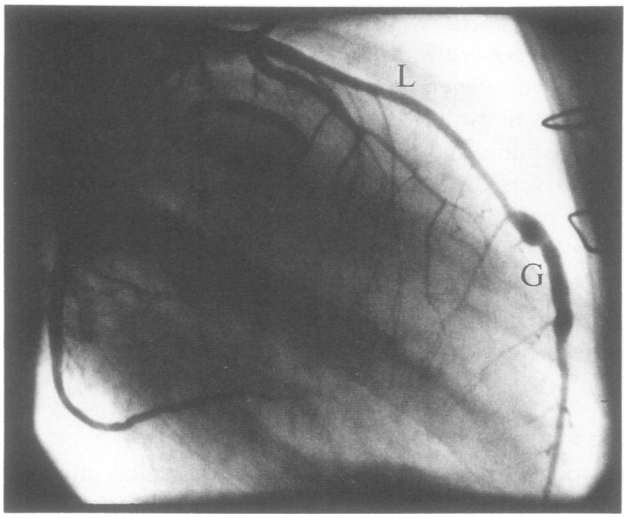

Fig. 4 *Left coronary arteriogram showing patent interposition graft. Note the improved flow in the distal left anterior descending coronary artery.*

G = graft; L = proximal left anterior descending coronary artery

unless seen at autopsy. Some patients present with angina or infarction from distal embolism or occlusion.[6,7]

The theoretical dangers of any aneurysm in the larger coronary arteries are those of local thrombosis with occlusion, distal embolism, and rupture.[1] Certainly angina and infarction occur, but the frequency of rupture cannot be ascertained from the literature for any cases grouped by causation, indicating that such occurrence must be exceedingly rare.

Mycotic aneurysm may be caused by microembolization of the vasa vasorum, by the impaction of macroscopic emboli from the infected site (followed by direct bacterial invasion of the arterial wall), or by medial injury from the deposition of immune complexes.[3,8]

Mycotic coronary aneurysm associated with infective endocarditis is believed to be caused by microemboli to the vasa vasorum or by local spread from impacted macroemboli from the primary site of infection.[3,8] Either could have been the precipitating factor in our patient.

Durack[1] suggests that small mycotic aneurysms resolve with appropriate antibiotic therapy, a view supported by Karp[9] in regard to cerebral arteries. However, Durack further suggests that aneurysms greater than 1 to 2 cm in diameter are unlikely to resolve, and can enlarge and eventually rupture, even when the lesions have been rendered sterile with antibiotic therapy. There are no other clinical reports to support this contention in relation to the coronary arteries.

In our patient, the aneurysm had a thick (2 to 3 mm) fibrotic wall except on the epicardial surface, and certainly had the potential for enlargement and rupture in that region. However, the primary reason for direct intervention was the risk of occlusion of the distal portion of the LAD and the consequent loss of myocardium in the presence of left ventricular hypertrophy. This risk is real: fatal, late thrombosis of a sterile mycotic aneurysm has been reported.[7]

Restoration of vascular continuity with an interposed graft was considered safe in the absence of active acute inflammation. In our patient, the interval from onset of symptoms to operation was 7 weeks, and the infection was under control. In the face of active, acute inflammation in uncontrolled endocarditis requiring surgery, interposition grafting close to the lesion would be dangerous. In this situation, ligation or excision, in combination with distal aortocoronary or internal mammary bypass, would be appropriate.

However, when operation is required in the active, acute phase of endocarditis, it is likely that there has been insufficient time for a mature mycotic coronary aneurysm to develop. The rarity of reports of later complications of mycotic coronary aneurysm—after surgical eradication of the primary focus and intensive antibiotic therapy[7]—suggest that such treatment may indeed abort the dilatory process, as suggested by Durack.[1]

Because coronary arteriography is often avoided in the presence of acute aortic valve endocarditis, especially when vegetations are present, it is also likely that mycotic coronary aneurysms are simply not detected. Indeed, some of the late deaths following valve replacement for endocarditis may be due to aneurysmal thrombosis, as reported by Davidson and colleagues,[7] or to rupture.

The lesion seen in the posterior ascending aortic wall probably is a jet lesion secondary to the process described by Rodbard,[10] in which bacteria are deposited in the vascular wall in the low-pressure sink downstream from a stenotic orifice. Our patient was unusual in that the aortic wall was significantly thinned and damaged by active local infection, giving the potential for further thinning and dilatation, for which reason the lesion was patched. Either this lesion, which was just above the origin of the left coronary artery, or the ulcerated anterior aortic leaflet, could have been the site of origin of macroembolism to the LAD and the cause of mycotic aneurysm formation.

For distal coronary mycotic aneurysm, simple ligation or excision is probably all that is required. For more proximal lesions in the major vessels, distal flow must be restored with a bypass graft after ligation or excision.

Because of the potential for late thrombosis,[7] it would seem logical to recommend coronary arteri-

ography in all patients with aortic valve endocarditis in order to detect the presence of such aneurysms. This would also assist considerably in ascertaining the true incidence of the lesion and its complications. However, the risk of dislodging infectious material from the valve or aortic root has to be considered. Because mycotic coronary aneurysm appears to be so rare, the risk seems unjustified in the acute situation; but coronary arteriography should certainly be part of the assessment of all patients undergoing operation for hemodynamic reasons, after successful medical treatment has rendered their lesions sterile.

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