CASE REPORTS

Left Ventricular Aneurysm Subsequent to Cleavage of Myocardial Bridging of a Coronary Artery

CHRIS de ZWAAN, MD, HEIN J. J. WELLENS, MD, FACC
Maastricht, The Netherlands

In two patients, an aneurysm of the left anterior wall developed at the site of periarterial muscle resection of a myocardial bridge over the proximal portion of the left anterior descending coronary artery. One patient had a severe atherosclerotic stenosis proximal to the bridge. He also received a bypass graft to the left anterior descending coronary artery distal to the bridge. In the other patient without signs of atherosclerotic disease, only cleavage of the myocardial bridge was performed. Observations in these two cases suggest the need to re-evaluate the risks of surgical treatment in symptomatic patients with myocardial bridging.

Myocardial bridging of coronary arteries was recognized and described by Black (1) in 1796. A myocardial bridge seems to be a congenital anomaly, apparently due to incomplete exteriorization of the primitive coronary intratubecular arterial network, and is usually located over the mid portion of the left anterior descending coronary artery and only exceptionally over the diagonal, obtuse marginal or posterior descending branches (2–8).

The incidence of this anomaly is higher in women than in men (8). It is found in 5 to 86% of patients in anatomic studies (2–4) and observed in 0.5 to 12% of patients undergoing coronary arteriography (7–12). Its significance in relation to myocardial perfusion is still a matter of debate. Evidence is increasing, however, that myocardial bridging is not an innocent finding and that some patients with this finding may suffer from myocardial ischemia (7,8,11,13), myocardial infarction (4,7,13–17), ventricular fibrillation (15), conduction disturbances (left anterior hemiblock, left bundle branch block, right bundle branch block and complete atrioventricular [AV] block) (7,8,18) and sudden death (19). The preferred surgical approach to the treatment of myocardial bridging is not clear, although successful cases of supra-arterial myotomy have been reported (20–24). That cleavage of the bridge may lead to a ventricular aneurysm is suggested by the following observations.

Case Reports

Case 1

A 54 year old man presented to the outpatient clinic because of atypical chest pain. The pain, which was not related to the typical triggers for angina, lasted less than 5 minutes and did not respond to treatment with a beta-adrenergic blocking agent or long-acting nitroglycerin. With the exception of a blood pressure of 180/110 mm Hg, the physical examination, the laboratory data, chest X-ray film and echocardiogram were normal.

During a treadmill exercise test the patient complained of chest pain during stage V of the Bruce protocol. No electrocardiographic changes diagnostic of myocardial ischemia were observed. A thallium-201 perfusion scan showed no abnormalities during exercise.

Cardiac catheterization. Because of continuing complaints, the patient was admitted to the hospital. Cardiac catheterization revealed myocardial bridging with a systolic narrowing of 85% in the left anterior descending coronary artery over a distance of about 3 cm at the site of the second and third septal branches. This was followed by poststenotic dilatation. No other abnormalities were observed in the coronary arteries. The anterolateral and anteroseptal segments of the left ventricle showed early relaxation. Because of the elevated blood pressure, the patient was discharged on a regimen of a beta-adrenergic blocking agent and a diuretic drug.

Clinical course. Ten months later the patient had an attack of chest pain on exertion that lasted 15 minutes and led to admission to our coronary care unit. For the first time, the electrocardiogram showed abnormal ST segments with
deeply inverted T waves in the precordial leads suggestive of ischemia of the anterior wall of the left ventricle. A calcium channel antagonist was added to the drug regimen. No rise in cardiac serum enzyme levels was observed. A new thallium-201 exercise study showed a perfusion defect in the ventricular septum that disappeared at rest. A second cardiac catheterization revealed the same findings as 10 months previously.

**Surgery.** Because of the abnormal ST segments on the electrocardiogram and the perfusion defect on the thallium study, we decided to operate on the patient. During operation, the myocardial bridge, which was 3.5 cm long and 1 cm thick, was cleaved stagewise. The postoperative course was without complications. One week postoperatively, the electrocardiogram showed complete right bundle branch block with loss of R wave voltage in precordial leads V₁ and V₂. The laboratory data showed a rise in serum enzyme values (U/liter): creatine kinase (CK) maximally 430, serum glutamic oxaloacetic transaminase (SGOT) maximally 85, lactic dehydrogenase (LDH) maximally 600, the upper limit of normal being, respectively, 200, 40 and 450.

**Postoperative findings.** After operation the patient was free of complaints. During a treadmill exercise test he had no complaints and the electrocardiogram showed slight elevation of the ST segment in lead V₄. The thallium-201 exercise study showed no perfusion defects. The echocardiogram revealed a dyskinetic area in the septal segment of the left ventricle compatible with an aneurysm. Cardiac catheterization revealed a 2 cm aneurysm at the site of the cleavage of the myocardial bridge (Fig. 1). The left anterior descending coronary artery was completely normal.

**Case 2**

A 44 year old man was admitted to the coronary care unit because of an “impending myocardial infarction.” For 1 week the patient had had attacks of typical anginal chest pain occurring mostly at rest and lasting less than 20 minutes. Physical examination was normal. The electrocardiogram on admission showed no abnormalities. Twenty hours after admission, however, deeply inverted T waves were present in precordial leads V₁ to V₄. Laboratory data showed a minimal rise in serum enzyme levels (U/liter): CK 225, SGOT 30, LDH 520.

**Cardiac catheterization.** A 90% narrowing was noted in the proximal part of the left anterior descending coronary artery before the first septal branch. This area of fixed narrowing was followed by a segment showing a systolic narrowing of 90% over a distance of approximately 3.5 cm in the area ranging from the first to the third septal branch. The apical and anteroseptal segments of the left ventricle showed a hypokinetic contraction pattern.

**Coronary angioplasty.** In view of these findings, coronary angioplasty was performed and resulted in a reduction of the narrowing of the proximal portion of the left anterior descending coronary artery to 50%. After this procedure the inverted T waves in the precordial leads persisted for 13 days, when the patient was readmitted because of an attack of chest pain at rest lasting 15 minutes and resulting in the development of positive T waves in the precordial leads. Because no enzyme level changes occurred, an impending occlusion of the left anterior descending coronary artery was suspected and the patient was operated on.

**Surgery.** During operation a myocardial bridge, 4 cm long and 0.5 cm thick, was dissected. A coronary bypass graft was placed on the left anterior descending coronary artery, distal to the site of cleavage. Postoperative laboratory data were normal. The electrocardiogram returned to normal and treadmill exercise test was negative.

**Postoperative findings.** Two months postoperatively, the patient had a new episode of anginal chest pain lasting 2 hours. On admission the electrocardiogram showed changes in the QRS complex and ST segment suggestive of a small anteroseptal infarction. The electrocardiographic changes were accompanied by a slight rise in enzyme serum levels (U/liter): CK 390, SGOT 70, LDH 1,200. A technetium scan did not demonstrate fresh myocardial infarction.

**Four days after admission cardiac catheterization was performed.** The left ventricular angiogram showed a prunesized aneurysm in the anteroseptal area (Fig. 2, right). The
bypass graft to the left anterior descending coronary artery was patent. The proximal portion of this artery was occluded at the site of insertion of the aortocoronary bypass graft.

Discussion

The two patients described had a severe systolic narrowing over a long bridged segment of the proximal portion of the left anterior descending coronary artery. One patient also had a severe organic stenosis in the proximal part of the same artery. Both patients were operated on because of chest pain accompanied by electrocardiographic changes suggesting a critical lesion high in the left anterior descending coronary artery. One patient also had a reversible perfusion defect during a thallium exercise study. In the other patient, a coronary angioplastic procedure had been performed previously.

Postoperative findings. The postoperative left ventricular angiogram of both patients showed an aneurysm and not a dyskinetic segment of the left ventricle at the site of cleavage of the bridge. Postoperatively, Patient 1 with normal coronary arteries had a rise in serum enzyme levels and an electrocardiographic pattern suggesting a small anteroseptal infarction. No abnormalities were found on the angiogram of the left anterior descending artery. Patient 2 with preexistent coronary artery disease did not develop complications immediately after operation, but had a long-lasting attack of chest pain 2 months later. This was accompanied by a slight rise in serum enzyme levels and an electrocardiographic pattern of a small anteroseptal infarction. In this patient, the proximal segment of the left anterior descending coronary artery was found to be occluded on the coronary angiogram 2 months postoperatively. In the first patient, the aneurysm apparently was the direct consequence of the cleavage of the myocardial bridge. In the second patient, we cannot completely exclude a relation with the occlusion of the proximal left anterior descending coronary artery, even though the bypass graft was patent.

Implications. Our observations suggest that cleavage of a myocardial bridge may result in development of a left ventricular aneurysm. They stress the need for careful evaluation of the different modes of surgical therapy in patients with symptomatic myocardial bridging of a coronary artery.

Figure 2. Case 2. Left ventricular angiogram taken in the 30° right anterior oblique position at the end of systole before (left) and after (right) operation. Postoperatively, an aneurysm is visible at the site of cleavage of the myocardial bridge.

We are indebted to Bruno Messmer, MD, Professor of Cardiac Surgery, RWTH Aachen, Federal Republic of Germany and to Jehia Mashhour, MD and Joseph Bredée, MD, Department of Cardiac Surgery, Catharina Hospital, Eindhoven, The Netherlands, who operated on the patients.
References


