Myocardial Bridging in a Young Patient with Sudden Death

DAVID CUTLER, M.D., AND JOHN M. WALLACE, M.D.

University of Texas Medical Branch at Galveston, Division of Cardiology, John Sealy Hospital, Galveston, Texas, USA

Summary: Systolic compression of a coronary artery is considered to be a benign phenomenon, although numerous case reports have suggested an association between bridging and sudden death or ischemia in certain patients without other abnormalities on cardiovascular evaluation. We present the case of a young patient with two episodes of spontaneous ventricular fibrillation and electrocardiographic evidence of ischemia, whose only primary abnormality on extensive workup was a long segment of left anterior descending systolic compression. This case adds to the growing body of anecdotal evidence that myocardial bridging may be associated with significant cardiac events.

Key words: systolic compression, myocardial bridging, sudden death

Introduction

Systolic compression of a segment of coronary artery by an intramyocardial bridge is not infrequent, the incidence ranging from 0.5 to 9% in reviews of coronary angiography and from 5 to 80% in autopsy studies.¹ The prevalence in angiographic studies is higher when systolic compression has been specifically sought retrospectively. Bridging is almost always an incidental benign finding and only rarely is cited as the cause of ischemic or myocardial dysfunction. Because nearly all coronary flow to the left ventricle occurs during diastole, it is postulated that the phenomenon of systolic compression of a coronary artery is usually not of clinical significance.² We report the case of a young man with two episodes of sudden death and electrocardiographic (ECG) evidence of ischemia in

whom a long segment of left anterior descending (LAD) artery bridging was discovered on extensive cardiac evaluation.

Case Report

The patient is a previously healthy 29-year-old man whose illness began 4 months prior to evaluation at this hospital. While relaxing at home he suddenly became unresponsive and cardiopulmonary resuscitation (CPR) was performed by a family member. Subsequent monitoring in the ambulance revealed polymorphic ventricular tachycardia (Fig. 1A). Upon arrival at the hospital, the rhythm spontaneously converted to sinus tachycardia with marked ST-segment elevation anterolaterally and inferiorly consistent with an acute anteroapical and lateral infarct (Fig. 1B). Initial laboratory data obtained just after sinus rhythm returned were remarkable for metabolic acidosis, pH = 7.18, consistent with the lactic acidosis often seen after cardiac arrest. The serum potassium was low at 2.8 meq/l. Drug screening was negative. Although tissue plasminogen activator (TPA) was promptly given, ventricular fibrillation requiring cardioversion occurred multiple times on the day of admission but did not recur after the first 24 h. He was intubated and comatose for a number of days, but he slowly improved and was weaned from the ventilator. An echocardiogram revealed severe diffuse hypokinesis with an ejection fraction of 25% and an apical thrombus. The maximum creatine phosphokinase (CPK) level was 8000 IU, but the MB portion only rose to 40 ng/ml, suggesting that the majority of enzyme leaks was due to skeletal muscle injury from the electrical cardioversions. Following extubation, further history revealed that he abused tobacco and alcohol, but there was no other history of drug use, additional cardiac risk factors, prior heart disease, or a family history of heart disease or sudden death. A cardiac catheterization performed 3 weeks after admission was reported to show normal coronary arteries and overall normal left ventricular function. No thrombus was seen. His ECG at this time was completely normal. He was treated with anticoagulants and nitroglycerin (NTG) and was discharged.

The patient did well until 1 week before admission to this hospital. While seated in a chair he became suddenly unresponsive. Subsequent monitoring revealed ventricular fibrillation and he was cardioverted a number of times before sinus

Address for reprints:

David Cutler, M.D.
400 Wabash Avenue, Suite 220
Akron, OH 44307, USA

Received: May 21, 1996

Accepted with revision: November 4, 1996

FIG. 1 (A) Rhythm strip with polymorphic ventricular tachycardia. (B) Initial electrocardiogram showing diffuse injury pattern.

rhythm was restored. The ECG revealed diffuse nonspecific ST-T changes. Lidocaine was administered and no further ventricular arrhythmias occurred. A drug screen again revealed no evidence of cocaine or amphetamines. The maximum CPK was 590 IU with an MB fraction of 71 ng/ml.

The patient was transferred to this institution for further evaluation. All medicines including lidocaine were discontinued. By continuous monitoring, ventricular arrhythmias and ventricular ectopy were known to be completely absent during the entire 2-week hospital stay. An echocardiogram initially showed mild anterior hypokinesis and an ejection fraction of 45%; no clot was seen. A repeat study 1 week later was completely normal. A signal-averaged ECG done on two occasions was consistently positive for low-voltage afterpotentials. Right and left heart catheterization revealed normal pressures without evidence of oxygen step-up. Both left and right ven-

triculography were normal. Initial coronary angiography revealed normal arteries, although the LAD was somewhat small. Testing with intravenous methylergonovine to a total dose of 0.4 mg provoked no chest pain, ECG changes, arrhythmia, or focal spasm. Standard repeat angiography after intracoronary NTG revealed a 20 mm segment of mid-LAD bridging that was not previously evident (Fig. 2A). Systolic compression caused an 80% reduction in diameter of the bridged segment compared with diastole (Fig. 2B). A right ventricular biopsy was normal. An electrophysiologic study (EPS) demonstrated normal conduction system, and aggressive stimulation using up to three extra stimuli at four different cycle lengths in the right ventricular (RV) apex, RV outflow tract, and in the left ventricle induced neither ventricular fibrillation nor ventricular tachycardia, even after the addition of isoproterenol. Magnetic resonance imaging (MRI) of the heart

FIG. 2 (A) Systolic compression (bridging) of the left anterior descending (LAD) (see arrow). (B) Diastolic frame with normalization of the LAD (see arrow).

was unremarkable. Thus, there was no evidence of arrhythmogenic RV dysplasia by biopsy, EPS, or MRI. An exercise-sesamibi test was normal.

Based on the malignant history and the initial ECGs showing both ventricular tachycardia and fibrillation, implantation of a defibrillator and surgical unroofing of the LAD bridge was recommended. The patient refused any surgical treatment and was discharged on empiric amiodarone therapy. He has been asymptomatic during a 2-month follow-up.

Discussion

Kramer *et al.* observed 81 patients with myocardial bridging who had otherwise normal coronary arteries and found no incidence of myocardial infarction or sudden death over a 5-year follow-up, even among a subgroup with >50% systolic compression.³ Sudden death was not the indication for catheterization in any of these patients, however. A review of 707 cases of bridging by Angelini *et al.*, including 61 of their own cases, revealed no evidence for definitive ischemia in association with bridging. Conflicting results with both radionuclide and coronary sinus lactate studies and the lack of a consistent clinical syndrome in these patients were cited as evidence against a true ischemic phenomenon.²

Nevertheless, small series and case reports have offered compelling evidence for an association of bridging with ischemia and sudden death. Faruqui reported two cases of symptomatic bridging, one of which involved a patient with ventricular fibrillation and an evolving anterior myocardial infarction,⁴ similar to our patient's initial presentation. Kracoff *et al.* noted a patient with recurrent ventricular tachycardia and an LAD bridge.⁵ They suggested that focal scarring in the ventricular septum due to recurrent systolic compression of the LAD might serve as a substrate for the arrhythmia. They also noted that the tachycardia recurred during an exercise test when the sinus rate exceeded 160, suggesting an ischemic etiology. Noble *et al.* used coronary sinus lactate levels to demonstrate heart rate-dependent ischemia in patients with bridging and hypothesized that shortened diastolic perfusion during tachycardia may allow systolic compression to become clinically important.⁶ This theory is supported by an autopsy report from Morales *et al.* that revealed LAD bridging in three patients with exercise-associated sudden death.⁷ Areas of septal fibrosis and reduced posteroseptal circulation were noted in all three patients. Of interest, the resting ECG in all patients was thought to demonstrate septal fibrosis by the loss of small normal "septal" Q waves in the lateral leads, a finding not present in our patient. Chambers *et al.* recently reported a case of myocardial stunning in a patient with an LAD bridge.⁸ The transient wall motion abnormalities described parallel those seen in this case. Recently, intravascular ultrasound (IVUS), quantitative coronary angiography (QCA), and Doppler flow analysis have been used to characterize symptomatic myocar-

dial bridging.^{9,10} IVUS and QCA have demonstrated that systolic compression is accompanied by delayed diastolic relaxation and residual mild diastolic narrowing. Intracoronary Doppler flow analysis has revealed elevated diastolic flow velocities within the bridging segment and impaired coronary flow reserve. Rapid atrial pacing was shown to elevate the diastolic flow velocity further, and to worsen flow reserve as well as to induce angina and ECG changes. Intravenous beta blockade was able to reverse much of the abnormality even when atrial pacing was continued, indicating that the negative inotropic effect may minimize systolic compression and improve resultant diastolic flow characteristics.

Our patient initially presented with ventricular fibrillation associated with an evolving anteroapical infarct, a scenario most consistent with LAD ischemia. In the cases cited, such events were usually related to exertion, although several did occur at rest, as in this report. Our patient also had a second resting episode of ventricular fibrillation accompanied by a significant enzyme leak, again indicating a probable ischemic etiology. An extensive evaluation revealed a long segment of LAD bridging as the only primary abnormality, reemphasizing that myocardial bridging may be associated with significant cardiac events.

References

1. Irvin R: The angiographic prevalence of myocardial bridging in man. *Chest* 1982;81:2:198-202
2. Angelini P, Trivellato M, Donis J, Leachman R: Myocardial bridges: A review. *Prog Cardiovasc Dis* 1983;26:1:75-87
3. Kramer J, Kitazume H, Proudfit W, Sones M: Clinical significance of isolated coronary bridges: Benign and frequent condition involving the left anterior descending artery. *Am Heart J* 1982;103:2:283-288
4. Faruqui A, Maloy W, Felner J, Schlant R, Logan W, Symbas P: Symptomatic myocardial bridging of coronary artery. *Am J Cardiol* 1978;41:1305-1310
5. Kracoff O, Ovsyshcher I, Gueron M: Malignant course of a benign anomaly: Myocardial bridging. *Chest* 1987;92:6:1113-1115
6. Noble J, Bourassa MG, Petitclerc R, Dyrda I: Myocardial bridging and milking effect of the left anterior descending coronary artery: Normal variant or obstruction? *Am J Cardiol* 1976;37:993-999
7. Morales A, Romanelli R, Boucek R: The mural left anterior descending coronary artery, strenuous exercise and sudden death. *Circulation* 1980;62:2:230-236
8. Chambers J, Johns J, Berndt T, Davee T: Myocardial stunning resulting from systolic coronary artery compression by myocardial bridging. *Am Heart J* 1994;128:5:1036-1038
9. Schwarz E, Klues H, vomDahl J, Klein I, Krebs W, Hanrath P: Functional, angiographic and intracoronary Doppler flow characteristics in symptomatic patients with myocardial bridging: Effective short-term intravenous beta blocker medication. *J Am Coll Cardiol* 1996;27:7:1637-1645
10. Ge J, Erbel R, Rupprecht H, Koch L, Kearney P, Gorge G, Haude M, Meyer J: Comparison of intravascular ultrasound and angiography in the assessment of myocardial bridging. *Circulation* 1994;89:4:1725-1731