Acute Myocardial Infarction Associated with Myocardial Bridging in a Young Adult

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Abstract

A 28-year-old man was admitted because of chest pain. Emergency coronary angiography showed a massive thrombus in the proximal segment and another occlusive thrombus in the distal segment of the left anterior descending artery. He was treated with thrombolytic therapy. Repeat coronary angiography showed disappearance of the thrombi in the proximal and distal segments and obvious myocardial bridging in the mid segment. Intravascular ultrasound revealed an atherosclerotic plaque in the segment immediately proximal to the myocardial bridging, but did not reveal any plaque within or distal to the site. He was discharged 12 days later.

Key words: myocardial bridging, coronary angiography, intravascular ultrasound, smoking

Introduction

Myocardial bridging of the coronary arteries is a frequent congenital anomaly, detected in 15% to 85% of autopsy series and in 0.5% to 16% of angiographic studies (1–5). Although myocardial bridging is generally considered as a harmless clinical anomaly, several reports have showed an association of this condition with sudden cardiac death (6), myocardial infarction (7, 8), coronary artery spasm (9) or ventricular arrhythmia (10). We describe a young man with acute myocardial infarction (AMI) associated with myocardial bridging which was assessed by coronary angiography and intravascular ultrasound.

Case Report

A 28-year-old man with no history of hypertension, diabetes, hypercholesterolemia or Kawasaki disease experienced chest pain during smoking at 14:00 on June 11, 2003. Because his chest pain continued for 2 hours, he was admitted to our hospital. He was fully conscious, his pulse rate was 85 beats/min, and blood pressure was 110/66 mmHg. The peripheral pulses were normal and he did not have edema. His neurologic examination was negative. Electrocardiography on admission showed ST elevation in II, III and aVF leads, but did not show ST elevation in precordial leads (Fig. 1). Chest X-ray showed no pulmonary congestion. Emergency cardiac catheterization was performed via the right radial artery. Left ventriculography showed severe hypokinesis of the apical region. Coronary angiography showed a massive thrombus in the proximal segment which did not limit coronary blood flow and another occlusive thrombus in the distal segment of the left anterior descending artery (Fig. 2, arrows). He was treated with tissue plasminogen activator and heparin. The activities of protein C and protein S were normal, and peak creatine kinase was 547 IU/l. Anticoagulant treatment with warfarin was initiated and the prothrombin time was meticulously kept in the therapeutic range.

Repeat cardiac catheterization was performed 8 days later. Coronary angiography showed disappearance of the thrombi in the proximal and the distal segments and obvious myocardial bridging in the mid segment of the left anterior descending artery (Fig. 2, arrows). He was treated with tissue plasminogen activator and heparin. The activities of protein C and protein S were normal, and peak creatine kinase was 547 IU/l. Anticoagulant treatment with warfarin was initiated and the prothrombin time was meticulously kept in the therapeutic range.

Repeat cardiac catheterization was performed 8 days later. Coronary angiography showed disappearance of the thrombi in the proximal and the distal segments and obvious myocardial bridging in the mid segment of the left anterior descending artery. Systolic compression of the mid segment was seen with return to a normal caliber during diastole. Intravascular ultrasound revealed an atherosclerotic plaque in the segment immediately proximal to the myocardial bridging, but did not reveal any plaque within or distal to the site (Fig.
3). He was treated with warfarin, aspirin and diltiazem, and was discharged 12 days later.

**Discussion**

A few case reports have demonstrated an association of myocardial bridging with AMI. Ramos et al reported a 26-year-old man with AMI who died 2 hours after the onset and underwent autopsy (11). They reported that left anterior descending artery buried deep within the heart wall for a 4 cm extension, and that an occlusive thrombus was found in the proximal segment of the myocardial bridging at autopsy. Angiographic findings in the present patient are consistent with their results. In our patient, there was an occlusive thrombus in the distal segment, which might be isolated from the thrombus in the proximal segment. Ramos et al also observed a greater severity of atherosclerosis in the proximal segment of the myocardial bridging in comparison with other affected areas at autopsy. Moreover, Ge et al performed intravascular ultrasound in 14 patients with angiographic evidence of myocardial bridging in the left anterior descending artery (12). They reported that no atherosclerotic lesions were detected in the myocardial bridging or the distal segment in the 8 patients in whom the intravascular ultrasound catheter was successfully advanced through the entire myocardial bridging, but atherosclerotic plaques were found in the proximal segment in 12 of 14 patients. In accordance with their reports, the present patient also had an atherosclerotic plaque only proximal to the myocardial bridging, and this lesion was not detected by coronary angiography. Although it remains unclear why atherosclerosis is confined mainly in the proximal segment, previous studies have suggested that local wall stress and subsequent injury to the vessel wall may result in local atherosclerosis or thrombus formation proximal to the myocardial bridging (12, 13). Our patient had no previous history of chest pain, and experienced chest pain at 14:00 when spontaneous coronary spasm was not likely to occur. However, he experienced chest pain during smoking which was associated with coronary spasm, platelet aggregation and blood coagulability (14, 15). Thus, we thought that such adverse effects of smoking caused thrombus formation under the existence of myocardial bridging.

![Figure 1](image_url)
Although the infarct artery was the left anterior descending artery, electrocardiography on admission showed ST elevation in inferior leads, and not in precordial leads. In fact, Sasaki et al previously demonstrated that patients with wrapped left anterior descending artery had ST elevation in inferior leads more frequently than those without (16). As shown in Fig. 2 and Fig. 3, the present patient had an obvious wrapped left anterior descending artery. This anatomy and occlusion in the distal segment probably resulted in ST elevation only in the inferior leads.

Therapeutic strategies in symptomatic patients with a myocardial bridging have been varied, including surgical myotomy (17, 18), calcium channel antagonists (19) and beta-blockers (20, 21), due to a poor understanding of the underlying pathophysiologic mechanisms. The beneficial effect of beta-blockers is based on their negative inotropic and chronotropic effects, which should lead to a reduction in external muscular compression and a prolongation of diastolic perfusion intervals. Teragawa et al recently reported that patients with myocardial bridging experience acetylcholine-induced coronary spasm more frequently than those without (9). Their results suggested that coronary spasm might be the proximate etiology of cardiac events associated with myocardial bridging. Because we thought that beta-blockers might induce coronary spasm especially in Japanese patients with a cardiac event associated with myocardial bridging, we treated our patient with diltiazem which had favorable effects including negative inotropic and chronotropic effects and an antivasospastic effect. Further studies are necessary to evaluate the long-term efficacy of diltiazem therapy.

In summary, we observed an atherosclerotic plaque in the segment immediately proximal to the myocardial bridging, but not within or distal to the site by using intravascular ultrasound. We should recognize myocardial bridging as a cause of acute myocardial infarction in the young adult. Although myocardial bridging is generally considered as a harmless clinical anomaly, intensive medical treatment is required in patients with a cardiac event associated with myocardial bridging.
References

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