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Unstable Angina Associated with Myocardial Bridging and Hypertrophic Cardiomyopathy

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Abstract

A 48-year-old man was admitted due to persistent chest tightness associated with cold sweats, hypotension and tachycardia. An electrocardiogram showed a sinus tachycardia with non-specific ST-T changes. Serial cardiac isoenzymes were not elevated. Unstable angina was diagnosed based on the clinical manifestations. Coronary angiography revealed no evidence of atherosclerosis but severe myocardial bridging in the middle third of the left anterior descending coronary artery which resulted in nearly total occlusion of the vessel in systole. Meanwhile, an echocardiogram revealed hypertrophic obstructive cardiomyopathy. The patient did well under medical treatment with bisoprolol and verapamil. In conclusion, both myocardial bridging and hypertrophic cardiomyopathy can cause myocardial ischemia and can be treated by calcium antagonists and/or beta-blocking agents. Coronary stenting can be performed to relieve the myocardial bridging but restenosis rate is high. Surgical myotomy or coronary artery bypass graft and transaortic subvalvular myectomy should be reserved when patients are refractory to medical treatment. (J Intern Med Taiwan 2008; 19: 260-265)

Key Words : Unstable angina, Myocardial bridging, Hypertrophic obstructive cardiomyopathy

Introduction

Myocardial bridging of coronary arteries is a frequent congenital anomaly. The incidence of myocardial bridging in angiographic studies is 0.5 to 12%[1,2], and a prevalence of 30 to 80% in adults with hypertrophic cardiomyopathy[3,4]. While some reports have suggested that myocardial bridging may be associated with myocardial ischemia or infarction[5,6], unstable angina in a patient with myocardial bridging and hypertrophic obstructive cardiomyopathy (HOCM) is not commonly reported. Herein, a patient with HOCM displaying unstable angina associated with nearly total systolic occlusion of the left anterior de-

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scending coronary artery (LAD) by myocardial bridging is presented and discussed.

Case Report

A 48-year-old male was admitted because of persistent severe chest tightness for few hours, associated with cold sweats. The patient was a non-smoker with pre-existing and poorly managed diabetes mellitus, hypertension, and hyperlipidemia. On admission, the blood pressure was 88/48 mmHg with a regular rapid heart rate of 116 beats per min. A grade 3/6 systolic murmur was noted at the aortic area. An electrocardiogram identified a sinus tachycardia with non-specific ST-T changes, with no evidence of left ventricular hypertrophy (Fig. 1). Cardiac isoenzymes were not elevated.

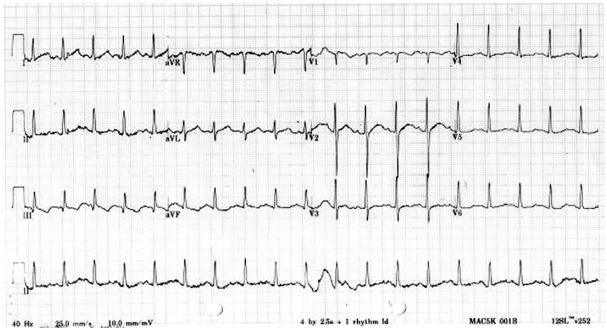

Fig.1. Electrocardiogram showing sinus tachycardia and non-specific ST-T changes.

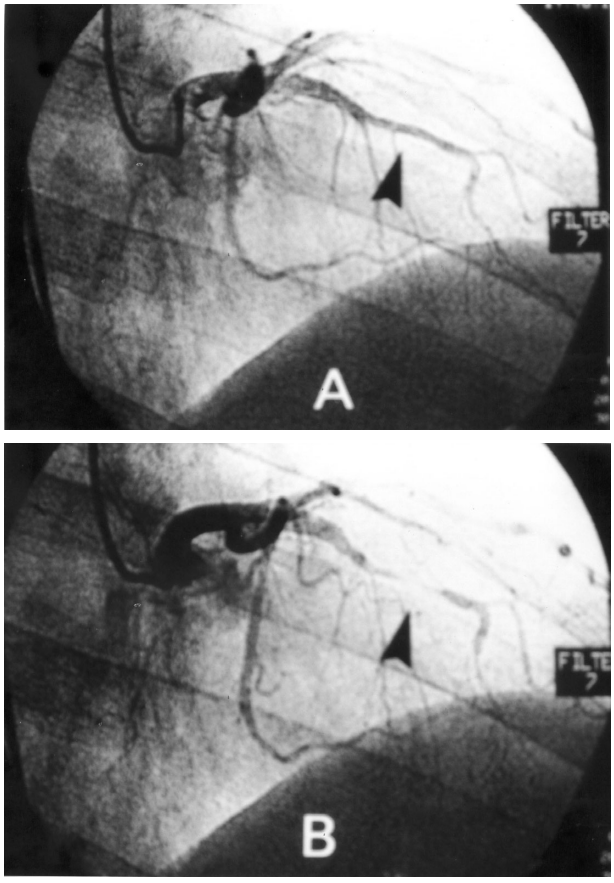

Fig.2. Coronary angiography (right anterior oblique projection) showing severe myocardial bridging of the middle left anterior descending artery (arrow) in diastole (A) and systole (B).

The patient was transferred directly to the cardiac catheterization laboratory with the presumptive diagnosis of acute coronary syndrome. Coronary angiography revealed no evidence of atherosclerosis in any coronary arteries. Instead, a myocardial bridge in the mid-LAD involving approximately 1.2 cm (in length) resulted in nearly total occlusion of the vessel in systole (Fig. 2). Left ventriculography showed hypercontractility of the left ventricle (ejection fraction = 93%) with mild mitral regurgitation. An echocardiogram revealed asymmetric septal hypertrophy where the thickness of the interventricular septum was 19 mm and the thickness of the left ventricular posterior wall was 12 mm. The left ventricle outflow gradient was 36 mmHg. In addition, systolic anterior motion (SAM) of the mitral valves, impaired left ventricle relaxation, and a mild to moderate mitral regurgitation were also noted (Fig. 3A, 3B). The ejection fraction was 80%, and no regional wall motion abnormalities were found.

Initially, dopamine was used to maintain systolic blood pressure, but was soon discontinued. Verapamil (80 mg) three times daily and bisoprolol (2.5 mg) one time daily were initiated after the patient's blood pressure stabilized. After heart rate slowed to 80 beats per min, the heart murmur was inaudible. A follow-up echocardiogram 2 weeks later showed disappearance of the left ventricular outflow gradient and SAM (Fig. 3C, 3D). The degree of mitral regurgitation also decreased. There were no further symptoms of chest tightness or dyspnea during the follow-up period.

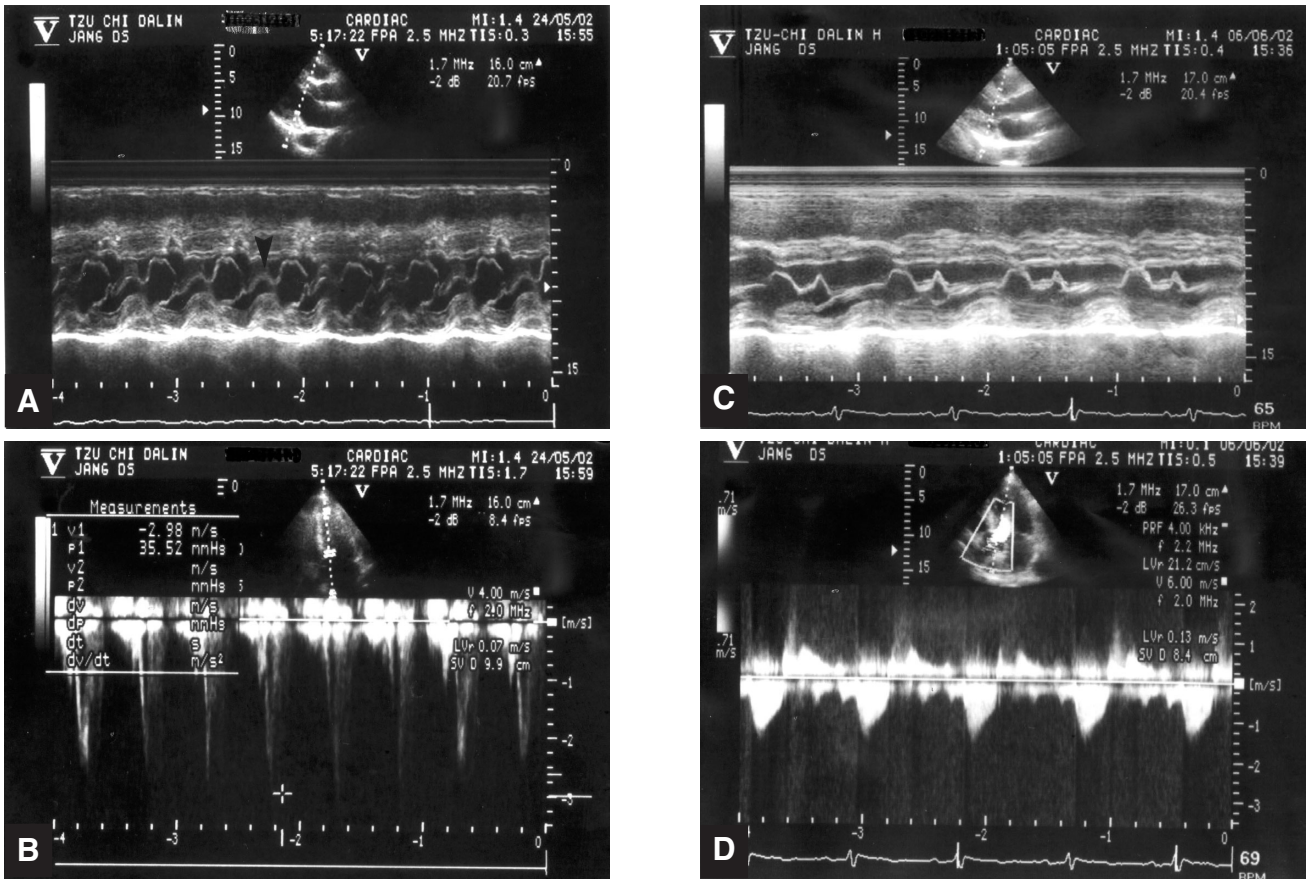

Fig.3.(A) M-mode echocardiogram (parastenal long axis view) showing systolic anterior motion of the mitral valves (arrow). (B) Continuous wave Doppler echocardiogram (apical five chamber view) showing a pressure gradient through the left ventricular outflow tract of 36 mmHg. (C) Follow up M-mode echocardiogram (parastenal long axis) showing no systolic anterior motion of the mitral valves. (D) Continuous wave Doppler echocardiogram (apical five chamber view) showing no pressure gradient through the left ventricular outflow tract.

Discussion

Myocardial bridging, a common anatomical finding, involves systolic compression of the epicardial coronary arterial segment by the overlying myocardium. Systolic coronary artery narrowing can exist in any coronary vessel, although the LAD is most commonly involved. Myocardial bridging has often been associated with hypertrophic cardiomyopathy (HCM). The incidence of myocardial bridging in HCM is high. The cause is obscure. Similarly, Harikrishnan et al. also reported that the incidence of HCM in myocardial bridging patients is also high: up to 33%[7].

Herein, we reported a patient with myocardial bridging and HOCM presented with unstable angina. While this patient had a history of diabetes mellitus, hyperlipidemia, and hypertension, a coronary angiogram revealed no evidence of atherosclerosis. Instead, severe myocardial bridging of the middle LAD was noted, which resulted in nearly total occlusion of the affected vessel in systole. In addition, an echocardiogram revealed concurrent HOCM.

Both myocardial bridging and HOCM can induce myocardial ischemia[1-7]. At least 3 factors may contribute to the development of myocardial ischemia in patients with myocardial bridging. These include length of the tunneled coronary segment, degree of systolic compression, and heart rate[8]. In cases with longer tunneled segments, more severe systolic narrowing of the bridged segment, and tachycardia, myocardial ischemia may be more common.

This theory may partially explain why our patient's symptom subsided after his heart rate decreased from 116 to 80 beats per minute.

The ausculatory hallmark of HOCM is a systolic murmur which usually reflects both outflow tract turbulence and concomitant mitral regurgitation[9]. In the patient described here, the systolic murmur, SAM, and left ventricular outflow gradient each disappeared after heart rate reached 80 beats per minute following administration of verapamil and concor. The degree of mitral regurgitation and the patient's chest tightness also improved. These findings suggest that HOCM may have played an important role in myocardial ischemia in this patient.

Medical treatment for HCM with myocardial bridging was recommended as the first line therapy. Beta-blocking agents and calcium antagonists can each decrease heart rate and myocardial dp/dt resulting in an improvement in the systolic obstruction by the muscle bridging. These drugs are also capable of decreasing the interventricular pressure gradient in HOCM patients.

Coronary stenting has been performed to treat patients with myocardial bridging with favorable result[10]. Nonetheless, coronary perforation has been reported during stenting in a patient with myocardial bridging[11]. Qian et al. determined that that vessel area in the myocardial bridge segment was smaller than in adjacent both segments proximal and distal to myocardial bridging[12]. Therefore, careful selection of stent size by intravascular ultrasound is recommended prior to performing coronary intervention to prevent coronary perforation.

In contrast to the findings of Haager et al., Kursaklioglu et al. reported an in-stent restenosis rate of patients with myocardial bridging after stenting was high (67%)[13]. This group concluded that despite the apparent immediate favorable results, stent implantation in myocardial bridging may not be realistic due to the high in-stent restenosis rate. Drug-eluting stenting is now possible in some patients with myocardial bridging. Whether drug-eluting stents could decrease the restenosis rate in such patients will require analysis via a prospective, randomized, controlled clinical trial.

Surgical resection of the bridge (myotomy) or coronary artery bypass grafting is also a surgical possibility; however, these techniques are typically reserved for patients who fail to respond to more conservative measures. Wu and Xu followed myocardial bridging cases treated via myotomy (15 patients) or coronary artery bypass grafting (16 patients) for 3 - 115 months (mean was 31 months)[14]. All patients survived and recovered uneventfully and were symptom-free during follow-up period, suggesting that both these techniques are appropriate.

Walters et al. reported a young women with HCM and severe myocardial bridging receiving percutaneous intracoronary stenting. Severe and diffuse restenosis developed within 30 days of the procedure. They concluded myotomy may provide a more effective treatment option for HCM patients with symptomatic myocardial bridging[15]. But whether the effect of stenting in myocardial bridging patients with HCM is worse than those without HCM deserves further observation.

Patients with myocardial bridging have a good long-term prognosis[7,16], although several reports have associated myocardial bridging with an adverse prognosis in pediatric HCM patients[17]. To investigate the prognosis of myocardial bridging in adult patients with HCM, Sorajja et al. followed 425 adult patients with HCM, including 64 patients with myocardial bridging for 6.8 $\pm$ 5.4 years[18]. This group reported no difference in survival free of all-cause mortality (5-year estimate, bridging vs. no bridging, 91% vs. 85%; P = 0.42), all cardiac death (93% vs. 89%; p=0.60), and sudden cardiac death (95% vs. 97%; p=0.72). Univariate and multivariate proportional hazards models also did not identify the presence of bridging or specific characteristics of the degree or extent of bridging with a poor outcome. As such, Sorajja et al.

concluded that no increased risk of death (including sudden cardiac death) among adult patients with HCM who had myocardial bridging existed.

Finally, in patients with HOCM, transcoronary ablation of septal hypertrophy or transaortic subvalvular myectomy is also possible in the event of failure to respond to medical management.

In conclusion, myocardial ischemia can be caused by myocardial bridging, and HCM can aggravate the ischemic symptoms. Both myocardial bridging and HCM are clinical entities that can be treated by calcium antagonists and/or beta-blocking agents. Alternatively, coronary stenting can be performed to relieve the myocardial bridging, but restenosis may occur. Surgical myotomy or coronary artery bypass graft and transaortic subvalvular myectomy for patients with myocardial bridging and HOCM should be reserved when patients are refractory to medical treatment.

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不穩定性心絞痛伴隨心肌橋及肥厚性心肌病變

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摘　要

一位48 歲男性病患因持續性胸悶，伴隨有冒冷汗、低血壓及心博過速的情形而住院。心電圖顯示實性心博過速及非特異性ST-T 段變化。一連串的心肌酵素測量並沒有升高。臨床上根據病人的臨床表現診斷爲不穩定性心絞痛。冠狀動脈血管攝影顯示冠狀動脈本身並無粥狀硬化的情形，但是在左前降支動脈中段有嚴重心肌橋，導致左前降支動脈在心臟收縮期時幾乎完全阻塞。另外心臟超音波顯示病人合併有肥厚性阻塞型心肌病變。病人胸悶的症狀經過bisoprolol 及verapamil 治療後緩解。心肌橋和肥厚性心肌病變兩者皆可能引起心肌缺血並且都可以用鈣離子阻斷劑及乙形阻斷劑加以治療。冠狀動脈支架置放可以治療心肌橋但再狹窄率偏高。而心肌切開術或冠狀動脈繞道手術及經主動脈瓣膜下心肌切除術應保留給內科治療失敗的病人。