

ANGINA PECTORIS DUE TO SEVERE MUSCULAR BRIDGE IN HYPERTROPHIC CARDIOMYOPATHY

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ABSTRACT

We report the case of a 39-year-old male with hypertrophic cardiomyopathy who complained of angina pectoris. The patient was treated with a beta blocker and a calcium antagonist without effect. Myocardial scintigraphy revealed anterior ischemia. Cardiac catheterization and ventriculography revealed severe systolic narrowing of the left anterior descending coronary artery and no significant pressure gradient across the left ventricular outflow tract. Myotomy was performed on a muscular bridge over the left anterior descending coronary artery and the patient's angina was relieved. In young patients with hypertrophic cardiomyopathy who develop angina refractory to medical therapy, a coexisting muscular bridge should be sought.

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INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is a primary hypertrophy of cardiac muscle characterized by increased left ventricular mass, a small left ventricular cavity, increased systolic function, and impaired diastolic function.¹ Angina pectoris is the most common symptom of HCM found in about three-quarters of symptomatic patients and may be related to a number of anatomic or physiologic variables.² These include imbalance between oxygen supply and demand as a consequence of the increased myocardial mass, inadequate capillary density, elevated diastolic filling pressures, impaired vasodilatory reserve, coronary vasospasm, septal perforator arteries compression, unsuspected coronary atherosclerosis, and

systolic compression of coronary arteries due to myocardial bridging. In patients over 45 years of age, atherosclerotic coronary artery disease is common although the ischemic pain is indistinguishable from that in patients with HCM.³ We present the case of a patient with muscular bridging and HCM who suffered angina pectoris due to phasic severe narrowing of the left anterior descending coronary artery during systole.

CASE REPORT

A 41-year-old male was admitted because of unstable angina pectoris. His symptoms started 2 months before admission and he was prescribed beta blockers, calcium antagonists, and monosorbide dinitrate. He had no risk factors for coronary artery disease. His blood pressure was 140/80 mm Hg and his heart rate was 76 beats per minute. On cardiac examination, the apical impulse was displaced laterally and it was forceful and enlarged. A 2/6 midsystolic ejection murmur was present at the left sternal border and radiated to the lower sternal border and apex. Chest radiography was normal. Electrocardiography showed left axis deviation, left ventricular hypertrophy, anterior T wave abnormalities, and abnormal prominent Q waves in leads II, III, and AVF, suggestive of inferior

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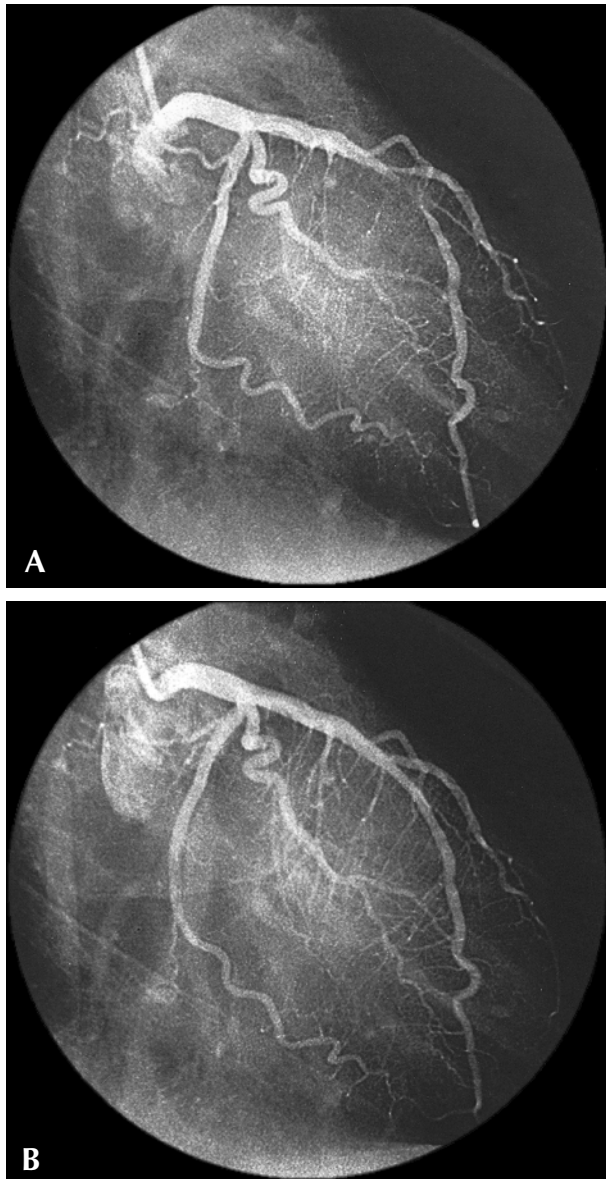


Figure 1. Coronary angiogram showing severe long-segmental stenosis of the left anterior descending coronary artery during systole (A) with normalization during diastole (B), suggesting a myocardial bridge.

myocardial infarction. Echocardiography showed left ventricular hypertrophy. Maximal hypertrophy was seen at the upper septum, reaching 23.3 mm in diastole. The left ventricular cavity was small and the left ventricular outflow tract was slightly narrowed but no significant pressure gradient was present. Systolic anterior motion of the anterior mitral leaflet was not observed. Although HCM was diagnosed, concomitant coronary artery disease was suspected because of the abnormal electrocardiographic findings and persisting angina pectoris in spite of medical treatment. Tetrafosmin myocardial scintigraphy showed ischemia of the left ventricular anterior wall. Left ventriculography revealed apical hypokinesia. A 15-mm Hg peak systolic gradient was measured at the left ventricular outflow tract. Coronary angiography showed a muscular bridge causing near occlusion of the left

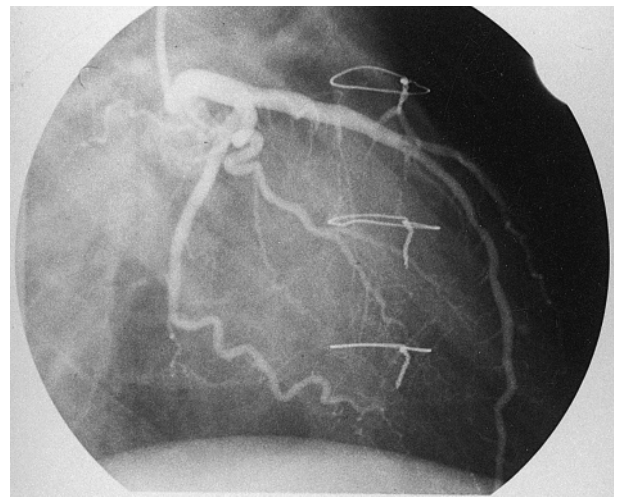


Figure 2. Coronary angiogram after resection of the myocardial bridge showing no narrowing of the left anterior descending coronary artery during systole.

anterior descending coronary artery (LAD) during systole (Figure 1A), which normalized during diastole (Figure 1B). The other coronary arteries were normal.

Myotomy was performed using cardiopulmonary bypass and moderate systemic hypothermia (28°C to 30°C). Myocardial protection consisted of topical hypothermia and cold blood cardioplegia. After the heart was arrested, the embedded segment of the LAD was freed from the myocardial bridge that was approximately 5 cm long and of 1 cm thickness. It was located over the proximal segment of the LAD in direct line with the thickened area of the ventricular septum. Because the ventricular gradient was not found to be severe during catheterization, septal myectomy was unnecessary. The patient's postoperative course was uneventful and he was discharged from the hospital 8 days after surgery. His angina disappeared after the surgical procedure. Angiography performed one month postoperatively revealed no LAD compression during systole (Figure 2).

DISCUSSION

The overall prevalence of HCM has been estimated as 0.02% to 0.2% of the population.⁴ Angina pectoris is the most common symptom in patients with HCM, which is due to several anatomic and physiologic factors including atherosclerotic coronary artery disease and muscular bridging.² Kitazume and colleagues⁵ reported the occurrence of significant muscular bridging over the LAD in approximately 15% of patients with HCM. Cokkinos and colleagues³ reported the coexistence of atherosclerotic coronary artery disease and HCM although this is rare in patients under 45 years of age. Beta blockers and the calcium antagonist verapamil can reduce angina pectoris and also prevent asymptomatic myocardial ischemia in HCM. In our patient who was receiving atenolol and verapamil for 2 months, there was no relief of angina

pectoris. His electrocardiogram revealed anterior ischemia and Q waves suggestive of an old inferior infarction. Therefore, myocardial scintigraphy was performed and myocardial ischemia was documented. Coronary angiography demonstrated the muscular bridge causing severe systolic narrowing of the LAD and the left ventricular outflow tract had no significant pressure gradient at the time of study. There was no evidence of atherosclerotic coronary artery disease. Even though Kitazume and colleagues⁵ did not recommend surgery for an isolated muscular bridge, surgical intervention may benefit patients with persistent ischemic symptoms refractory to medical therapy.^{6,7}

In our patient, freeing the LAD from the muscular bridge relieved his angina pectoris and the postoperative angiographic findings were normal. Our results are in accordance with previous studies reporting successful myotomy of muscular bridges.⁶⁻⁸ We recommend that even patients younger than 45 years with HCM and long-standing angina pectoris refractory to medical treatment should be considered for coronary angiography. Patients with severe narrowing of the coronary arteries due to muscular bridging can be successfully treated with surgery.

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