Cardiac arrest and myocardial bridging

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Myocardial bridging, a systolic compression of an intramyocardial segment of an epicardial coronary artery, may be an incidental finding during coronary angiography or autopsy. However, some investigators reported a variety of acute coronary syndromes associated with this condition. We describe the case of a relatively young male patient (43 years old) referred to our emergency department for cardiac arrest and subsequent evidence of significant myocardial bridging at coronary angiography. The patient has been treated with an implantable cardioverter-defibrillator to prevent possible subsequent arrhythmic events associated with the myocardial bridging.

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Introduction

Myocardial bridging occurs when a band of cardiac muscle overlies an intramyocardial segment of an epicardial coronary artery. The prognostic significance of this entity is controversial with some investigators suggesting that it is a benign condition[1,2] and others reporting a variety of acute coronary syndromes associated with it[3,4]. Less often myocardial bridging has been associated with malignant arrhythmias[5,6].

We describe the case of a male patient with severe myocardial bridging referred to our emergency department for cardiac arrest.

Case report

A 43-year-old male, a maintenance employee in our hospital, was referred to our emergency department because of a cardiac arrest while at work. The patient was a smoker (20 cigarettes/day), had no history of drug abuse and had no other conventional risk factors for ischemic heart disease (diabetes, obesity, hypertension, dyslipidemia or a family history of coronary artery disease). His colleagues said that while he was working in the hospital, he suddenly lost consciousness and fell down. They immediately referred him to the emergency department. The patient did not refer any prior symptoms. The ECG showed ventricular fibrillation and after DC-shock, delivered within 5 min of the arrest, it showed T-wave inversion in the antero-lateral leads (Fig. 1). Repeated blood testing for cardiac enzymes was negative for myocardial infarction and the ECG normalized within 3 days. The QT segment was normal and an echocardiogram, performed the same day of the arrest, did not show valvular or structural heart disease. The serum electrolytes were normal. After 4 days, the patient was referred for a coronary angiography which showed no atherosclerotic coronary disease, but a significant myocardial bridge of the left anterior descending coronary artery with severe systolic compression (Fig. 2). The ventriculogram was normal. An ergonovine stress test was negative for coronary spasm and exercise myocardial Thallium single-photon emission computed tomographic scintigraphy was negative for reversible ischemia. The patient was treated with an implantable cardioverter-defibrillator (ICD) and beta-blockers and, 18 months after the cardiac arrest is asymptomatic.

Discussion

Myocardial bridging is characterized by a systolic compression of an intramyocardial segment of an epicardial coronary artery. It was initially recognized by Reyman in 1737 at *post-mortem* analysis but only in 1976 Noble et al.[7] described the transient narrowing of the left anterior descending coronary artery during systole in patients who underwent selective coronary angiography. The prevalence of myocardial

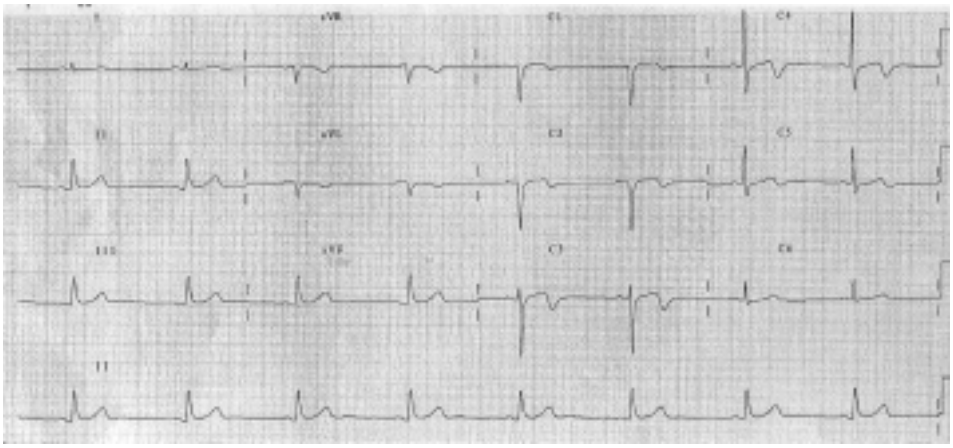

Figure 1. *ECG performed after DC-shock. The ECG showed a sinus bradycardia (50 b/min) with T-wave inversion in the antero-lateral leads.*

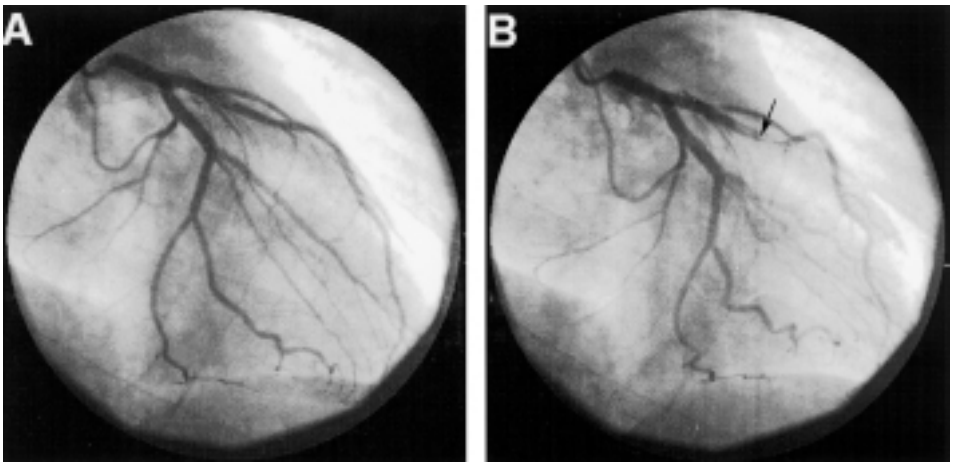

Figure 2. *Coronary angiogram showing the left anterior descending coronary artery during diastole* (A) *and systole* (B). *During systole, a significant reduction in the coronary diameter* (arrow *in* B) *with a "milking effect" may be noted.*

bridging varies among studies with a higher prevalence in *post-mortem* studies. The rate of angiographic bridging is < 5% and the length of myocardial bridging varies widely (from 4 to 40 mm)[8,9]. Among patients with isolated myocardial bridging documented at angiography, only two thirds exhibit a > 50%[7] narrowing of the vessel during systole and only in a few cases has a ≥ 75% narrowing been observed. Many case series showed that myocardial bridging may be an incidental finding at the time of coronary angiography and that it is a benign condition[1,2]. However, previous case reports showed that long bridges or bridges with ≥ 75% diameter reduction are often symptomatic[3,4] probably because of transient myocardial ischemia. Indeed, angiographic and intravascular studies[10,11] demonstrated that vessel compression is not limited to systole: in a case series of 20 patients, 85% of them with systolic compression had an extension of the obstruction into diastole with impaired coronary flow and reduced vasodilator reserve[8].

The present case refers to a relatively young patient with severe myocardial bridging resuscitated from a cardiac arrest. The patient was a smoker but had no prior history of ischemic heart disease or of chest pain. Other possible causes of cardiac arrest such as myocardial infarction (normal myocardial enzymes), coronary spasm (negative ergonovine test), electrolyte disorders, ECG alterations (long QT syndrome or Brugada syndrome) or structural and valvular heart disease (normal echocardiogram) were excluded. The only anomaly detected in this patient was the myocardial bridging. Previous reports documented cases of ventricular tachycardia[5], or ventricular fibrillation[12,13] associated with severe myocardial bridging, in the absence of myocardial infarction or structural heart disease, confirming our hypothesis. In one case[5], a subsequent electrophysiological study demonstrated an inducible sustained ventricular tachycardia.

The mechanism by which the myocardial bridging has probably induced the arrhythmia may be a transient

myocardial ischemia, due to the reduced coronary flow during diastole. Since long bridges with significant compression (≥ 75% diameter reduction) are often observed in symptomatic patients with impaired diastolic flow, it may be that in our patient the length of the bridging and the severity of compression may have impaired the coronary flow sufficiently to induce the arrhythmia.

We did not accurately screen our patient for prothrombotic risk factors and we cannot exclude the possibility of a transient clot in the coronary arteries that may have induced myocardial ischemia with subsequent ventricular fibrillation. However, the absence of a prior familial and individual history of thrombotic disease and the absence of myocardial damage (normal myocardial enzyme levels after the event) suggest that this was not the case.

The treatment of myocardial bridging is restricted to symptomatic patients[9]. Beta-blockers[10,14] or calcium channel blockers[5] are often the first-line therapy: the inotropic negative properties of these drugs might explain the decreased bridge-induced systolic coronary compression. Volume loading may also reduce compression of myocardial bridging, whereas the administration of nitrates may be detrimental because of increased compression and ischemia[15]. Other therapeutic approaches consist of coronary angioplasty with stent implantation and surgical myectomy. Intracoronary stents[16] prevent external compression of the bridged segments but the incidence of intrastent stenosis necessitating further target vessel revascularization is high. Surgical myectomy is more invasive and associated with a higher risk of periprocedural complications and is restricted to patients who remain symptomatic despite other therapies. No specific antiarrhythmic therapies have yet been proposed.

In our patient we proposed the implantation of an ICD together with beta-blockers to prevent the arrhythmic consequences of the bridging. Other approaches were excluded because at scintigraphy there was no evidence of myocardial ischemia and the patient was asymptomatic for angina pectoris. In this case, further evaluation of the arrhythmic risk was not taken into consideration because the patient had an "idiopathic" ventricular fibrillation. Besides, the implantation of an ICD is mandatory[17]. For those patients with severe myocardial bridging in the absence of documented myocardial ischemia, a more aggressive arrhythmic evaluation may be helpful to evaluate the arrhythmogenicity of this condition.

In conclusion we cannot consider "severe" myocardial bridging a benign condition, particularly when the bridging is long and associated with a systolic compression resulting in a diameter reduction ≥ 75%. These patients should be evaluated for the risk of life-threatening arrhythmias but the role of prophylactic implantation of an ICD needs to be evaluated further.

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