Case reports

Myocardial bridging in hypertrophic cardiomyopathy: a plea for surgical correction

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Myocardial bridging may be associated with an unfavorable prognosis in patients with hypertrophic cardiomyopathy. We describe a case of a young symptomatic patient with myocardial bridging associated with hypertrophic cardiomyopathy successfully treated by surgical unroofing. Such a procedure should be strongly recommended in patients with hypertrophic cardiomyopathy.

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Introduction

An intramural coronary artery is defined as an arterial segment of variable length that courses deep in the myocardium and is covered by myocardial fibers (a "myocardial bridge")[1]. Myocardial bridging may cause compression of the coronary artery resulting in myocardial ischemia. The prevalence of myocardial bridging in the general population is 0.5-1.6%[2].

The clinical significance of myocardial bridging remains controversial since in the majority of cases there is no evidence of myocardial ischemia. Myocardial bridging may be associated with hypertrophic cardiomyopathy and in this context it may represent a risk factor for sudden death, particularly in children[2].

We report a case of myocardial bridging in a young patient affected by hypertrophic cardiomyopathy in whom surgery was indicated to relieve symptoms and improve prognosis.

Case report

A 29-year-old man was referred to our unit with the diagnosis of hypertrophic cardiomyopathy. He presented with chest pain, which developed with increasing frequency and intensity even at rest over a period of 2-3 years. The symptomatology had no benefits from medical therapy.

Holter ECG monitoring showed ventricular arrhythmias and ST-segment depression in leads V_3-V_5 during chest pain. Echocardiography confirmed the diagnosis of hypertrophic cardiomyopathy with marked septal hypertrophy (thickness of the septum 26 mm) and small-sized ventricular cavity, with clear evidence of diastolic dysfunction. The ejection fraction was 47% being the anterior wall hypokinetic. There was no evidence of left ventricular outflow tract obstruction. A small atrial septal defect was detected (Qp/Qs = 1.4).

Coronary angiography revealed complete systolic obstruction of a long segment (6 cm) of the left anterior descending coronary artery (LAD). The site of bridging was in the middle portion of the LAD, just distal to the origin of the first diagonal branch (Fig. 1). In diastole the LAD appeared of normal diameter without obstructive lesions (Fig. 2). The correlation with the echocardiographic findings revealed that the bridging portion of the LAD was in the area of the most prominent septal hypertrophy. The patient was operated on for surgical unroofing of the LAD in the bridging portion and the muscular compression was relieved. The operation was performed with midline sternotomy using extracorporeal circulation and cross-clamping the aorta; the atrial septal defect was closed at the same time. The bridging porting of the LAD was identified in the most hypertrophic area of the interventricular septum. Left ventricular myectomy was not performed as outflow obstruction was not significant. The postoperative course was uneventful and the patient was discharged 5

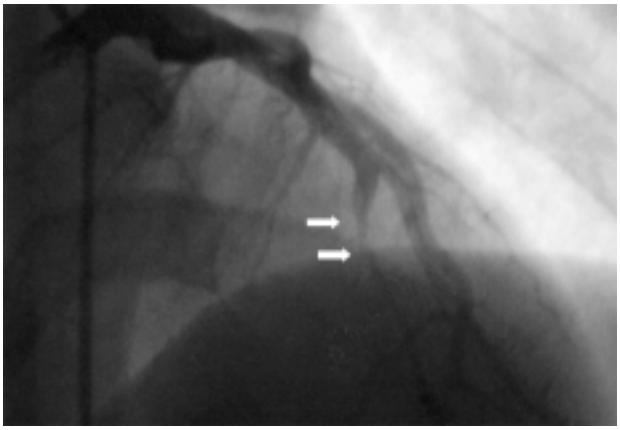

Figure 1. *Myocardial bridge of the left anterior descending coronary artery during systole.*

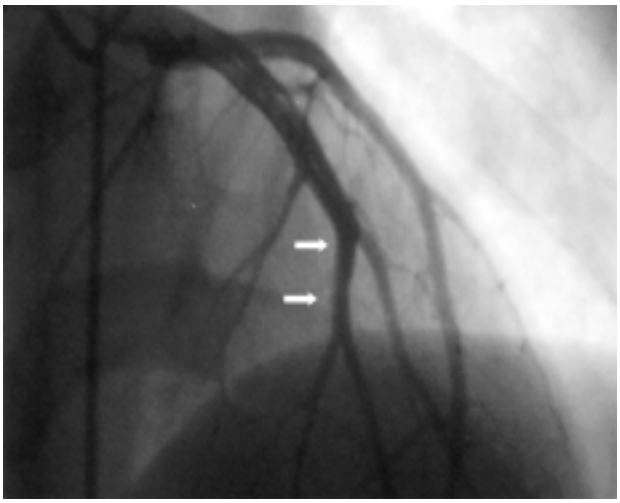

Figure 2. *Myocardial bridge of the left anterior descending coronary artery during diastole.*

days after the operation. Before discharge the patient underwent coronary angiography that revealed absence of systolic LAD occlusion or narrowing (Fig. 3).

At follow-up 1 year postoperatively the patient is asymptomatic.

Discussion

Myocardial bridging is commonly considered as an anatomic variation of the coronary arterial tree with little clinical implications. In the majority of cases patients are asymptomatic and bridging is occasionally discovered. Myocardial bridging has a typical appearance on coronary angiography. Compression of the coronary arteries by these bridges was first shown on angiography by Porstmann and Iwig[3] in 1960. The bridged segment is of normal caliber during diastole but narrows abruptly during systole in absence of atherosclerotic plaque. In a few cases myocardial bridging may cause angina, myocardial infarction or sudden death[4]. In symptomatic patients bridging causes a marked reduction in systolic blood flow. Also a reduction in diastolic flow has recently been documented by intracoronary Doppler ultrasound[5,6]. The association of bridging and hypertrophic cardiomyopathy in young patients is not rare. When hypertrophic cardiomyopathy is present, bridging is usually localized in the most hypertrophic area of the septum. The coronary artery more frequently involved is the LAD in the middle portion. The prognosis of patients with coronary bridging of the LAD in the context of hypertrophic cardiomyopathy is unfavorable[7]. There have been a number of reports of unselected patients and patients with hypertrophic cardiomyopathy that have associated myocardial bridging with cardiac morbidity and risk of death[8]. However, Sorajja et al.[9] reported that myocardial bridging was not associated with detrimental long-term outcome, including an increased risk of sudden cardiac death. Yetman et al.[2] report a high risk of sudden death in this group of patients and recommend surgical unroofing of the LAD. Probably the remarkable hypertrophy of the septum typically present in hypertrophic cardiomyopathy plays a role in determining a worse prognosis, compared to the bridging in the normal population. Different therapeutic options exist to treat myocardial bridging, such as stenting that is an easy and low-risk procedure, or surgical unroofing, that can be accomplished without cardiopulmonary bypass[10-12]. Several studies have specifically examined the hemodynamic consequences of myocardial bridging and have reported alleviation of cardiac ischemia and symptoms through surgical relief of myocardial bridging[13]. In our case the stenting procedure was not suitable for the length of the bridging tract and because of the origin of a main septal branch from the bridged LAD (Fig. 1). Moreover the presence of the septal defect precluded the off-pump unroofing.

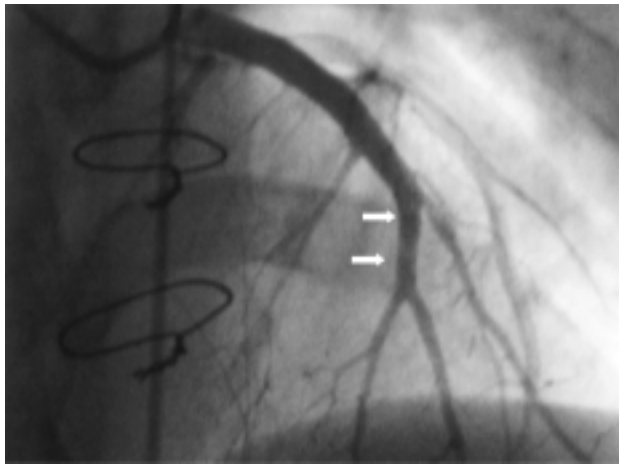

Figure 3. *Myocardial bridge of the left anterior descending coronary artery after surgical unroofing.*

On the basis of the above considerations, surgical treatment with dissection of the overlying myocardium should be limited to patients with symptoms that persist despite medical treatment. Downar et al.[14] described 5 symptomatic patients in whom symptoms resolved after supra-arterial myotomy. However, the decision is made on a case-by-case basis. Patients need to be evaluated with functional studies after the procedure to assess the surgical results. The experience in our case shows that the operation is simple and effective in relieving severe symptoms. On the basis of the available information also an improvement in prognosis can be expected. Additional studies are needed to assess which patients should be selected for invasive or surgical therapy.

References

1. Gori F, Basso C, Thiene G. Myocardial infarction in a patient with hypertrophic cardiomyopathy. N Engl J Med 2000; 342: 593-4.
2. Yetman AT, McCrindle BW, MacDonald C, Freedom RM, Gow R. Myocardial bridging in children with hypertrophic cardiomyopathy - a risk factor for sudden death. N Engl J Med 1998; 339: 1201-9.
3. Porstmann W, Iwig J. Intramural coronary vessels in the angiogram. Fortschr Geb Rontgenstr Nuklearmed 1960; 92: 129-33.
4. Frank RE Jr. Myocardial bridging. J Insur Med 1999; 31: 31-4.
5. Ge J, Jeremias A, Rupp A, et al. New signs characteristic of myocardial bridging demonstrated by intracoronary ultrasound and Doppler. Eur Heart J 1999; 20: 1707-16.
6. Schwarz ER, Klues HG, vom Dahl J, Klein I, Krebs W, Hanrath P. Functional characteristics of myocardial bridging. A combined angiographic and intracoronary Doppler flow study. Eur Heart J 1997; 18: 434-42.
7. Mohiddin SA, Begley D, Fananapazir L. Myocardial bridging in children with hypertrophic cardiomyopathy. N Engl J Med 1999; 341: 288-90.
8. Ishimori T, Raizner AE, Chahine RA, Awdeh M, Luchi RJ. Myocardial bridges in man: clinical correlations and angiographic accentuation with nitroglycerin. Cathet Cardiovasc Diagn 1977; 3: 59-65.
9. Sorajja P, Ommen SR, Nishimura RA, Gersh BJ, Tajik AJ, Holmes DR. Myocardial bridging in adult patients with hypertrophic cardiomyopathy. J Am Coll Cardiol 2003; 42: 889-94.
10. Haager PK, Schwarz ER, vom Dahl J, Klues HG, Reffelmann T, Hanrath P. Long-term angiographic and clinical follow-up in patients with stent implantation for symptomatic myocardial bridging. Heart 2000; 84: 403-8.
11. Akilli A, Kultursay H, Akin M, et al. Stenting of myocardial bridging. J Invasive Cardiol 1997; 9: 529-33.
12. Mahon NG, Sugrue DD. Treatment of a long segment of symptomatic myocardial bridging with multiple coronary stents. J Invasive Cardiol 1997; 9: 484-7.
13. Pey J, de Dios RM, Epeldegui A. Myocardial bridging and hypertrophic cardiomyopathy: relief of ischemia by surgery. Int J Cardiol 1985; 8: 327-30.
14. Downar J, Williams WG, McDonald C, Wigle ED, McCrindle BW. Outcomes after "unroofing" of a myocardial bridge of the left anterior descending coronary artery in children with hypertrophic cardiomyopathy. Pediatr Cardiol 2004; 25: 390-3.