Editorial comment

Should myocardial bridging always be surgically correct in hypertrophic cardiomyopathy?

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Myocardial ischemia may be responsible for some of the most severe complications of hypertrophic cardiomyopathy (HCM), including ventricular arrhythmias, sudden death, progressive left ventricular remodeling, and systolic dysfunction[1]. A causal link between acute myocardial ischemia and life-threatening arrhythmias has only been rarely demonstrated[2]. Microvascular dysfunction is the most important substrate of myocardial ischemia, and is a major predictor of negative long-term outcome[3]. Coronary artery disease in patients with HCM may also induce myocardial ischemia or myocardial infarction and be associated with negative outcome in HCM patients, as compared with the general population[4].

The issue of the clinical and prognostic impact of myocardial bridging in patients with HCM is still controversial. Although a few reports suggested in the past that myocardial bridging was a negative prognostic indicator in young children[5], other studies have not confirmed this finding both in children[6] and in adult patients[7].

Myocardial bridging is a common finding in children, with a prevalence of up to 52%, mostly associated with the magnitude of hypertrophy[6]. In adults a prevalence of 15% has been reported in a large cohort of patients seen in a tertiary referral center[7]. Incidentally myocardial bridging may induce myocardial ischemia and even relevant chest pain. The pathological increase in coronary pressures and the flow reduction, due to the prolonged relaxation time of the hypertrophied wall segment, may induce a variable hypoperfusion and induce ischemia, angina and myocardial infarction[8].

Typical angina related to effort or meals is relatively rare, but about 40% of the HCM patients complain of prolonged episodes of atypical chest pain, usually occurring at rest, but instrumental evidence of myocardial ischemia is not easy to demonstrate[9]. Typical ST-T changes on the ECG may be detected, but the ECG is neither a sensitive nor a specific marker, mainly due to the usual marked basal ST-T alterations due to left ventricular hypertrophy[1]. Perfusion myocardial scan is also of limited value in these patients. Only a quantitative perfusion study using positron emission tomography after dipyridamole injection, could demonstrate the presence of reduced perfusion in one or more wall segments. Unfortunately it is often difficult to obtain.

The possibility of superimposed atherosclerotic coronary artery disease or a myocardial bridge inducing myocardial ischemia should always be considered in patients with HCM who complain of chest pain and have evidence of myocardial ischemia. However when studied at angiography, most patients with angina show normal epicardial coronary arteries. Nevertheless, a coronary angiogram is not warranted in all patients with HCM, but it is clearly indicated whenever a suspicion of myocardial ischemia is present.

Implications for management of patients with hypertrophic cardiomyopathy and myocardial ischemia

At present, while no specific treatment has been demonstrated to relieve microvascular dysfunction, calcium channel blockers and beta-blockers may improve symptoms through a reduction in oxygen consumption or a redistribution of myocardial flow and perfusion[10]. Angiotensin-convert-

ing enzyme inhibitors may improve transmural myocardial perfusion, but are limited to those patients without evidence of obstruction. In clinical practice pharmacological treatment may positively influence the status of myocardial perfusion in patients with HCM and reduce or abolish angina in most patients.

In the case report of Fiorani et al.[11], the patient complained of angina. Instrumental evidence of myocardial ischemia was reported on ECG monitoring, although hypokinesia of the anterior wall and a slightly reduced ejection fraction might also be considered secondary to myocardial ischemia. However, no evidence of improvement of wall motion or ECG abnormalities, at Holter monitoring, is given after the operation. The patient is only reported asymptomatic.

In conclusion, myocardial ischemia is an important pathophysiological feature of HCM. It should be suspected in all patients complaining of chest pain and arrhythmias. Evidence of myocardial ischemia and hypoperfusion should be searched for and a coronary angiogram obtained in order to exclude the presence of atherosclerotic disease, amenable to invasive procedures, or myocardial bridging. Only when clear evidence of myocardial ischemia associated with myocardial bridging is obtained, the bridge should be relieved either by coronary stenting or open-chest surgery.

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