Myocardial bridging: A 'forgotten' cause of acute coronary syndrome – a case report

Chiara Ripa MD, Maria Cristina Melatini MD, Fabiola Olivieri BS PhD, Roberto Antonicelli MD FESC

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During a stress test, an asymptomatic 40-year-old man showed an ST depression above 4 mm and a horizontal ST depression above 2 mm in the V3 to V6 precordial leads during the recovery phase, without symptoms related to myocardial ischemia. After several days, he experienced recurrent episodes of oppressive retrosternal pain with radiation to the interscapular region, associated with stress dyspnea. Stress myocardial scintigraphy using technetium sestamibi was performed, which showed a modest push-pull deficit of perfusion in the septal-anterior basal area associated with a small deficit of perfusion in the apical region.

The patient was admitted to hospital with a diagnosis of unstable angina. Repeated episodes of chest pain appeared during this period, which were partially relieved with the administration of sublingual

nitrate. There were no significant changes in the electrocardiogram or cardiac enzyme levels. Coronary angiography showed initial parietal hypertrophy with normal segmentary kinesis and global systolic function and, most importantly, the presence of a systolic narrowing (myocardial bridging) of the middle one-third of the left anterior descending artery from the likely intramyocardial route. There was no significant stenosis of the remaining coronary tracts.

Myocardial bridges have traditionally been considered a benign condition, but recent studies have demonstrated that the clinical complications can be dangerous; these complications include acute coronary syndromes, arrhythmias (including supraventricular tachycardia and ventricular tachycardia), exercise-induced atrioventricular conduction blocks, transient ventricular dysfunction and sudden death.

Key Words: *Acute coronary syndrome; Chest pain; Myocardial bridging*

The myocardial bridge is an anomaly characterized by a typical intramyocardial route of a segment of one of the major coronary arteries. This anomaly is more frequent than previously thought, and its reported incidence varies from 1.5% to 16% (1).

Myocardial bridges have traditionally been considered to be a benign condition, but several recent studies have demonstrated that their clinical complications can be dangerous; these complications include ischemia and acute coronary syndromes (2-9), coronary spasm (10), ventricular septal rupture (11), arrhythmias (including supraventricular tachycardia and ventricular tachycardia) (12), exercise-induced atrioventricular conduction blocks (13), transient ventricular dysfunction (14) and sudden death (15). The prognosis of patients with myocardial bridges, therefore, is not as benign as it was believed to be in the past.

The present case report is a good example of the clinical effects of a myocardial bridge. It is therefore interesting not only because of the rarity of the case, but also because it brings the attention of cardiologists to an anomaly that is often neglected.

Considering the epidemiological prevalence of this anomaly, clinical suspicion of a myocardial bridge would be warranted in all cases of typical or atypical chest pain in subjects who have a low probability of atherosclerosis because they are free from the traditional cardiovascular risk factors, particularly in the young.

CASE PRESENTATION

During a stress test to evaluate sporting ability, a healthy 40-year-old man in optimal general condition, who was a member of a recreational cycling team, showed an ST depression above 4 mm

in V3 to V6 precordial leads and a horizontal ST depression above 2 mm in the same leads during the recovery phase, without symptoms related to myocardial ischemia. After several days, he experienced recurrent episodes of oppressive retrosternal pain with radiation to the interscapular region, associated with stress dyspnea. Even though the patient had a low cardiovascular risk (mild hypercholesterolemia was the only cardiovascular risk factor recorded), it was decided to perform stress myocardial scintigraphy using technetium sestamibi, which showed a modest reversible deficit of perfusion in the septal-anterior basal area associated with a reversible small deficit of perfusion in the apical region (Figure 1).

Because the typical chest pain remained, and due to the suspicion of unstable angina (blood tests did not show an increase in cardiac enzymes levels), the patient was admitted to the coronary care unit.

The patient had repeated episodes of chest pain while in the coronary care unit, confirming the hypothesis of unstable angina. The pain was partially relieved by administering nitrate sublingually, with aspecific alterations of electrocardiography and without increase of cardiac enzyme levels.

The echocardiography examination results were unremarkable, with a normal left ventricle size and global systolic function, and without obvious kinetic alterations. The aortic root was of normal size, while the left atrium had dimensions near the upper limit of normal. Doppler examination did not show a gradient of either regurgitations or relief.

Radiological examination of the thorax was negative for pleural-parenchymal lesions, and a small hiatal hernia was found on gastroscopy.

The patient was treated with the following medications: oral acetylsalicylic acid, 100 mg/day; clopidogrel, 75 mg/day;

UO di Cardiologia-UTIC, Istituto Scientifico INRCA, Ancona, Italy

Correspondence: Dr Roberto Antonicelli, UO Cardiologia-UTIC, Istituto Scientifico INRCA, Via della Montagnola 81, 60100 Ancona, Italy.

Telephone 39-071-800-3202, fax 39-071-800-3203, e-mail r.antonicelli@inrca.it

Figure 1 Hypoperfusion areas during stress (top) and rest (bottom) phases of technetium sestamibi myocardial scintigraphy. ANT Anterior; INF Inferior

sodium enoxaparin, 6000 IU twice daily; metoprolol, 5 mg twice daily; atorvastatin, 40 mg/day; esomeprazole, 40 mg/day; and ramipril, 2.5 mg/day.

Coronary angiography and left ventriculography showed the beginnings of parietal hypertrophy with conserved segmentary kinesis and global systolic function but, more significantly, it revealed the presence of a systolic narrowing (myocardial bridging) of the mid one-third of the left anterior descending artery, likely from the intramyocardial route. There was no stenosis of the remaining coronary tracts (Figure 2).

After the diagnostic procedures were complete, the patient was treated with beta-blockers in addition to hypocholesterolemic dietetic therapy for control of mild hypercholesterolemia.

After one-year follow-up, the patient was in good health, and using only a low dose of beta-blocker therapy. He continues recreational cycling without major functional limitations.

DISCUSSION

The myocardial bridge is an anomaly characterized by an intramyocardial route of a segment of one of the major coronary arteries.

It was recognized at autopsy by Reyman in 1737, and first described angiographically by Portmann and Igwig in 1960.

This anomaly is more frequent than previously thought; its reported incidence varies from 1.5% to 16% when assessed by coronary angiography, but can reach much higher percentages (from 40% to 80%) if estimated in the course of an autopsy series (1).

Myocardial bridging is generally confined to the mid left anterior descending artery (16); it is less frequently located in the circumflex artery, and is occasionally seen in the right coronary artery.

Figure 2 Systolic coronary reduction flow (arrow) in myocardial bridging during coronary angiography

Even though myocardial bridging has often been considered a simple variant of the normal anatomy of coronary arteries, previous reports have demonstrated that it can potentially have clinical relevance (16,17).

The pathophysiological mechanisms of clinical manifestations of a myocardial bridge are believed to be related to the tendency in these patients to develop atherosclerosis.

It has been demonstrated that the intima of the tunnelled artery is significantly thinner than that of the proximal segment of the artery (18). Morphological changes of the endothelial cells have also been shown, which suggests that the intima beneath the myocardial bridge may be protected by hemodynamic factors. Some studies (19) have reported that the expression of vasoactive agents (endothelin-1, endothelial nitric oxide synthase, angiotensin-converting enzyme) and the extension of the atherosclerotic process are diminished in the myocardial bridge compared with the proximal and subsequent artery segments. The preliminary data obtained up to now therefore suggest that the myocardial bridge is associated with a greater development of atherosclerosis in the proximal artery segment compared with the tunnelled artery.

The typical angiographic feature of a myocardial bridge is systolic narrowing of an epicardial artery, which is often completely resolved during the diastolic phase of the cardiac cycle. Because only 15% of coronary flow normally occurs during systole and because the myocardial bridge is a systolic angiographic event, it acquires clinical relevance only in particular conditions, such as tachycardia. Tachycardia can provoke an ischemic effect on the myocardial bridge by shortening the diastolic phase and increasing the importance of systolic blood flow (1).

Intracoronary ultrasonography and Doppler studies have made it possible to determine some characteristics of myocardial bridges that were not revealed by traditional diagnostic

instruments. In a study by Ge et al (20), the authors used intravascular ultrasonography to study 62 patients with angiographically positive signs of systolic compression of a tunnelled artery and intracoronary Doppler imaging to study 48 patients with myocardial bridging. In all the subjects analyzed through ultrasonography, the typical 'half-moon' phenomenon (echolucent area surrounding the myocardial bridge) was visible during both the systolic and diastolic phases, demonstrating that the extrinsic compression of a tunnelled artery is not only a systolic event, but it also persists during a significant portion of the diastolic phase. Moreover, in the majority of subjects examined through intracoronary Doppler, a particular flow pattern was observed at the level of the myocardial bridge, characterized by a marked increase in blood flow velocity during the early diastolic phase, followed by a sharp deceleration and a subsequent 'plateau' phase. Anterograde flow did not appear during the systolic phase of the cardiac cycle. This particular behaviour, known as the 'fingertip' phenomenon, can be explained by the fact that during the early diastolic phase, a sudden reduction in parietal tension and resistance occurs at the myocardial bridge with a consequent increase in blood flow volume. At the same time, a partial extrinsic compression persists in the tunnelled artery, with a persistent decrease in vessel diameter during early diastole, which together explain the typical marked increase of blood flow velocity during this phase of the cardiac cycle. On the contrary, during the systolic phase of the cardiac cycle, because of the extrinsic compression of the tunnelled artery, anterograde flow is not produced or is very reduced, and retrograde flow at the proximal segment of the myocardial bridge is observed.

The therapeutic approaches suggested currently for these patients are: beta-blockers (21), calcium channel blockers, coronary stents (22), minimally invasive coronary artery bypass grafting (23) and surgical myotomy (24).

Nitrates should generally be avoided because they increase the angiographic degree of systolic narrowing and can lead to a worsening of clinical conditions (25). Beta-blockers, on the contrary, reduce the cardiac frequency and increase the diastolic time, with a decrease in cardiac contractility and compression of the tunnelled artery. Thus, these pharmacological agents seem to offer a better therapeutic effect in these patients, although randomized controlled trials have not yet been conducted. Stenting of the myocardial bridge has, until now, given discordant results. Some studies (26) demonstrate that stenting can abolish hemodynamic alterations and improve symptoms, although no studies demonstrate normalization of myocardial perfusion when a perfusion defect was present before stenting. Moreover, high inflation pressures may be required for optimal stent implantation, which presents a higher risk of coronary perforation (27). The data collected until now demonstrate, therefore, that the treatment of myocardial bridges with stent implantation is not free from potential complications.

Surgical treatment with a dissection of the myocardial fibres that surround the myocardial bridge should be limited to patients with symptoms that persist despite medical treatment (28). Good clinical results have been reported in a small series of these patients, although this type of procedure is extremely delicate due to the possible appearance of dangerous complications, including right and left ventricular perforation. Minimally invasive coronary artery bypass grafting has been reported for myocardial bridges (23).

CONCLUSION

Myocardial bridges are generally expressed clinically in young adult men, in whom typical or atypical chest pain appears, induced by physical exercise and appearing at rest (29). Although these anomalies are present at birth, they generally do not become clinically visible before the third decade of life, although the reasons for this are still not clear.

Traditionally, myocardial bridges have been considered to be a benign condition, but several recent studies have demonstrated that their clinical complications can be dangerous, including ischemia and acute coronary syndromes (2-9), coronary spasm (10), ventricular septal rupture (11), arrhythmias (including supraventricular tachycardia and ventricular tachycardia) (12), exercise-induced atrioventricular conduction blocks (13), transient ventricular dysfunction (14) and sudden death (15). Therefore, the prognosis of patients with myocardial bridges is not benign as it was believed to be in the past.

Considering the epidemiological prevalence of these conditions, clinical suspicion of a myocardial bridge should be considered in all cases of typical or atypical chest pain, particularly in young patients with a low probability of atherosclerosis who are free from traditional cardiovascular risk factors.

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