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A Young Indian presenting acute ST Elevetion Myocardial Infarction with unfixed obstruction of LAD culprit due to Myocardial Bridging- Case Report.

KEYWORDS

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ABSTRACT *A 36-year-old male patient presented one hour after the onset of severe, substernal chest pain at rest. ECG showed ST-segment elevations in leads V1 to V4 that resolved with nitrate, aspirin and beta-blocker therapy. He developed another episode of angina with ST-segment elevations on electrocardiogram despite therapy with aspirin, clopidogrel, low-molecular weight heparin, beta-blocker and nitroglycerin. Emergent catheterization revealed unfixed obstructive coronary artery disease (CAD) with myocardial bridging. Myocardial bridging (MB) caused a 70 % systolic compression in the 22 mm long middle segment of the left anterior descending artery. A 3.0 x 20 mm drug eluting stent was deployed in the tunneled artery at 12 atm. A repeat cath showed in-stent restenosis which was treated with a 3 x 12 mm Cutting Balloon at a maximal inflation pressure of 12 atm. Brachytherapy was performed using a 60 mm BetaCath device to deliver 23 Gy over 5 minutes. The patient remains asymptomatic at 2-year follow-up*

Introduction-

The major coronary arteries, which normally are distributed over the epicardial surface of the heart, occasionally have a segmental intramyocardial course. During systole, this segment of the vessel is compressed, a condition referred to as milking or systolic "myocardial bridging" . On angiography, bridging is recognized as compression of a segment of a coronary artery during systole, resulting in narrowing that reverses during diastole. The dynamic and phasic nature of the obstruction serves to differentiate bridging from fixed coronary stenosis .It has been thought that most instances of bridging are of little clinical significance. However, there are reports suggesting that severe bridging of the major coronary arteries can produce myocardial ischemia, coronary thrombosis, and myocardial infarction, as well as predispose the patient to atherosclerosis or sudden death .

Case Report.

A 36-year-old male patient presented one hour after the onset of severe, substernal chest pain at rest. Physical examination revealed moderate distress secondary to chest pain. His blood pressure was 128/82 mmHg, his heart rate was 116 beats per minute and he had a respiratory rate of 22 per minute. The patient's lungs were clear bilaterally, and there was no murmur, rub or gallop. The initial electrocardiogram showed ST-segment elevations in leads V1 to V4 (See figure-1) that resolved with nitrate, aspirin and beta-blocker therapy.

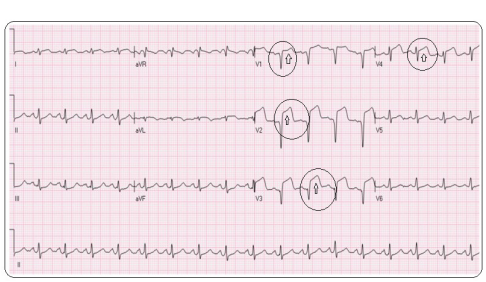
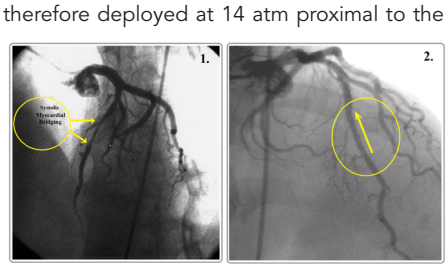


Figure-1(electrocardiogram showed ST-segment elevations in leads V1 to V4)

Eight hours after the onset of pain, his creatine kinase was 766 mg/dL and troponin I was 6.8 ng/mL. The patient developed another episode of angina with ST-segment elevations on electrocardiogram despite therapy with aspirin, clopidogrel, low-molecular weight heparin, beta-blocker and nitroglycerin. Emergent catheterization revealed unfixed obstructive coronary artery disease (CAD). Myocardial bridging (MB) caused a 70% systolic compression in the 22 mm long middle segment of the left anterior descending artery (Figure 2.1). A ventriculogram showed anteroapical hypokinesis, with an overall left ventricular ejection fraction of 45%. A 3.0 x 20 mm everolimus drug eluting stent was deployed in the tunneled artery at 12 atm. This closed-cell, slotted tube, stainless steel stent with uniform strut cell design was selected due to its strong radial stiffness and equal distribution of wall stress induced by MB and stent deployment forces.

Residual mild milking effect at the proximal edge of the first stent persisted and worsened after intracoronary therapy with 90 mg of nitroglycerin. A second 3.5 x 8 mm everolimus drug eluting stent was therefore deployed at 14 atm proximal to the first stent, with approximately 3 mm of overlap (Figure 2.2). The final angiogram showed complete disappearance of the milking effect. The patient presented 8 weeks later with crescendo angina.

(Figure 2.1)-Unfixed obstructive coronary artery disease (CAD). Myocardial bridging (MB) caused a 70% systolic compression in the 22 mm long middle segment of the left anterior descending artery. **(Figure 2.2)-** A second 3.5 x 8 mm bare metal Multi-LinkPenta coronary stent was therefore deployed at 14 atm proximal to the first stent.



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A repeat catheterization showed Mehran type 2 in-stent restenosis which was treated with a 3 x 12 mm Cutting Balloon at a maximal inflation pressure of 12 atm. Brachytherapy was performed using a 60 mm BetaCath *device* to deliver 23 Gy over 5 minutes. The patient remains asymptomatic at 2-year follow-up on beta-blocker and aspirin therapy, having fully returned to his previous activities.

Discussion-

A myocardial bridge is a band of myocardial tissue overlying a segment of epicardial coronary artery — tunneled artery. MB is a congenital abnormality that is almost exclusively localized in the mid-segment of the left coronary artery. The incidence of MB ranges between 15–85% in autopsy studies,and 0.5–16% in angiographic series.This usually benign condition4 has been associated with stable and unstable angina,myocardial infarction, myocardial stunning, atrioventricular nodal block, ventricular tachycardia and sudden death. Factors associated with increased risk of symptomatic MB include its length, thickness and location, severity of systolic arterial compression, presence of left ventricular hypertrophy, increased heart rate, low systolic blood pressure and concomitant coronary vasospasm.By an unclear mechanism, MB seems to protect the tunneled artery against atherosclerosis. Ge et al. found no atherosclerotic lesions in tunneled or distal coronary arteries in 8 patients, while 12 of 14 patients had atherosclerotic plaques proximal to a MB.

Diagnosis-

Atypical chest pain may be a presenting symptom in up to 45% of patients with MB. Sensitivity of exercise stress testing and perfusion imaging studies in patients with MB is comparable with that seen in patients with single-vessel coronary disease. MB with a minimal lumen diameter reduction of 70% during systole and 35% during diastole is considered to be hemodynamically significant. The intracoronary Doppler flow velocity profile within MB shows early diastolic flow acceleration — the "finger-tip" phenomenon, followed by a plateau phase and early systolic flow reversal in the proximal tunneled artery.Coronary flow reserve is reduced in patients with MB to less than 3.However, coronary flow reserve is not site-specific and both concomitant atherosclerotic lesions, and microvascular disease can overestimate the severity of a MB. Intravascular ultrasound helps to delineate the extent of MB. Provocation tests with intracoronary nitroglycerin or intravenous dobutamine uncover angiographically silent MB if the characteristic intravascular ultrasound (IVUS) "half-moon" phenomenon is present. IVUS also identifies significant atherosclerotic stenoses at the edges of the tunneled artery and may be particularly useful in MB stenting.

Treatment-.

Therapeutic options in symptomatic patients with MB are similar to therapy for atherosclerotic coronary artery disease.Beta-blockers and calcium channel blockers improve myocardial perfusion by prolonging diastole. Rate-independent anti-ischemic properties of beta-blockers seem to be mediated by reduced systolic and diastolic vascular compression. Calcium channel blockers may be particularly useful in patients with MB-associated vasospasm. Nitrates may worsen symptoms of MB due to increased vessel compression and should be used with caution. Antiplatelet agents counteract possible enhanced platelet aggregation in patients with MB. Coronary artery bypass grafting in symptomatic MB is currently used if there are other indications for cardiac surgery, Patient failed medical therapy and prefers surgical treatment and Patient failed medical and percutaneous therapy (unsuccessful stenting, recurrent in-stent restenosis). Minimally invasive, supra-arterial myotomy may become the preferred surgical therapy if its safety can be documented. Magnetic resonance imaging or intraoperative epicardial echocardiography may then provide necessary information regarding the MB thickness and intramyocardial vessel course.

Percutaneous Intervention-

Treatment of a tunneled artery with balloon angioplasty is not effective. MB leads to almost complete early arterial recoil. Stent placement is currently the only effective percutaneous therapy. IVUS helps in stent size selection and guides stent deployment to achieve maximal luminal area to prevent clinical in-stent restenosis. The radial strength of balloon-expandable stents seems to be sufficient to overcome compressive forces of the MB. Self-expanding stents have not been used in percutaneous therapy of symptomatic MB, and are unlikely to be useful due to their relatively low radial strength and potential recoil during periods of increased MB contractions. Stents with thin struts seem to induce less vessel wall injury and lead to lower rates of restenosis. However, they also seem to have lower radial strength. New metal alloys such as cobalt chromium overcome this limitation. Closed-cell design stents provide better radial strength than those with open-cell design and are therefore likely better suited for stenting in symptomatic myocardial bridging. Recurrent symptoms after percutaneous intervention were usually related to in-stent restenosis due to neointimal proliferation rather then stent strut compression. Percutaneous therapy appears to be safe. Reported complications include tamponade requiring emergent thoracotomy with venous patching of the coronary perforation and coronary dissection leading to a coronary fistula into the right ventricular outflow tract that resolved spontaneously within 3 months. Recurrent symptoms due to in-stent restenosis after percutaneous therapy on a symptomatic MB have been reported, but its frequency remains unknown.

Conclusion-

Myocardial bridging is a common, usually benign coronary artery anomaly that can (rarely) cause angina, myocardial infarction, malignant arrhythmia and sudden death. Non-invasive stress testing, quantitative coronary angiography, intracoronary ultrasound and intracoronary Doppler can identify patients at risk. Both percutaneous stent therapy and bypass surgery appear to be safe and effective therapies for MB and should be considered in patients who fail medical therapy with aspirin, beta-blockers and/or calcium channel blockers. Both percutaneous stent therapy and bypass surgery have been utilized in the treatment of symptomatic MB and may be considered in patients who fail medical therapy with aspirin, beta-blocker and/or calcium channel blocker.

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