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Case Report

Atypical chest pain with non-specific ST changes: an incidental finding of myocardial bridge without flow limiting coronary lesion

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ABSTRACT

Myocardial bridging is a developmental anomaly characterized by intramural course of a short segment of epicardial coronary artery which mostly remains silent in the initial few decades of life. A 42 years old male non diabetic, hypertensive on single regime therapy presented with atypical chest pain for the last two hours. Serial 12 lead ECGs on an interval of half-hour showing nonspecific dynamic ST changes in lead II, III and aVF suggesting of inferior wall changes with negative bio markers like troponin-T and troponin-I. Coronary angiography reveals myocardial bridge in mid segment of left anterior descending artery (LAD). Considering superficial type of bridging in this case, a conservative management strategy was planned accordingly. Physicians should be more vigilant on possibilities of underlying myocardial bridging as a major differential in emergency scenarios with low suspicion of atherosclerosis but with a picture equivalent to acute coronary syndrome (ACS). Myocardial bridge carries a wide array of complications depending on variabilities like depth of tunnelling, collapsibility, loss of wall sheer stress and vasospastic changes. Hence it needs to be promptly diagnosed with immediate treatment.

Keywords: Myocardial bridge, Angiography, Wall sheer stress, Dynamic ST changes

INTRODUCTION

Depending upon the extent of myocardial grooving; the segment of epicardial vessel which remains deep seated from the surface, myocardial bridging can be classified into: (a) Superficial bridges (75% cases) crossing perpendicularly and (b) Deep (25% cases), in which muscle fibres arising from right ventricular apical trabeculae crossing LAD transversely or obliquely.[1] It is not quite uncommon to witness this vascular anomaly in cadaveric cases yet its functional incidence is relatively uncommon, ranging from 0.5-16% when assessed through coronary angiography.[2] Myocardial tunnelling may vary according to its depth from 1-10 mm with length of the bridge mostly within 10-30 mm.[3] Segment of the epicardial artery proximal and distal to the myocardial groove having less wall shear stress (WSS) in its course often results in increased release of vasoactive and pro-thrombotic agents due to endothelial injury, giving rise to majority of atherosclerotic plaque in the these segments with predominance in proximal region.[2]

CASE REPORT

A 42-year-old male presented with complaints of severe chest pain which radiated to the left hand. He is a known hypertensive on Cilnidipine for the last 5 years. On examination blood pressure was 130/90 mm Hg, pulse rate was 67/min and respiratory rate was 20/min. No pallor, cyanosis or edema was observed. Normal bilateral vesicular breath sound, S1 and S2 heart sounds were audible on auscultation. ECGs was done in an interval of 30 mins which showed dynamic ST changes in leads II, III and aVF suggesting inferior wall changes (Figure 1). He was started on nicorandil (2 mg/hr infusion), tab. aspirin (325 mg stat.), tab. ticagrelor (180 mg stat.) and

tab. atorvastatin (80 mg stat.). Then a coronary artery angiography was performed which revealed an intermittent collapsing of middle long segment (appx. 20-25 mm) of the left anterior descending coronary artery suggesting myocardial bridging of the LAD (Figure 2). The patient was kept under observation for 24 hrs. following angiography with post-procedural satisfactory recovery. Patient was advised to take nicorandil-5 mg (BD), ecosprin-75 mg (ODPC), cilnidipine-10 mg (OD) upon discharge following guidelines as per Schwartz criteria.[4]

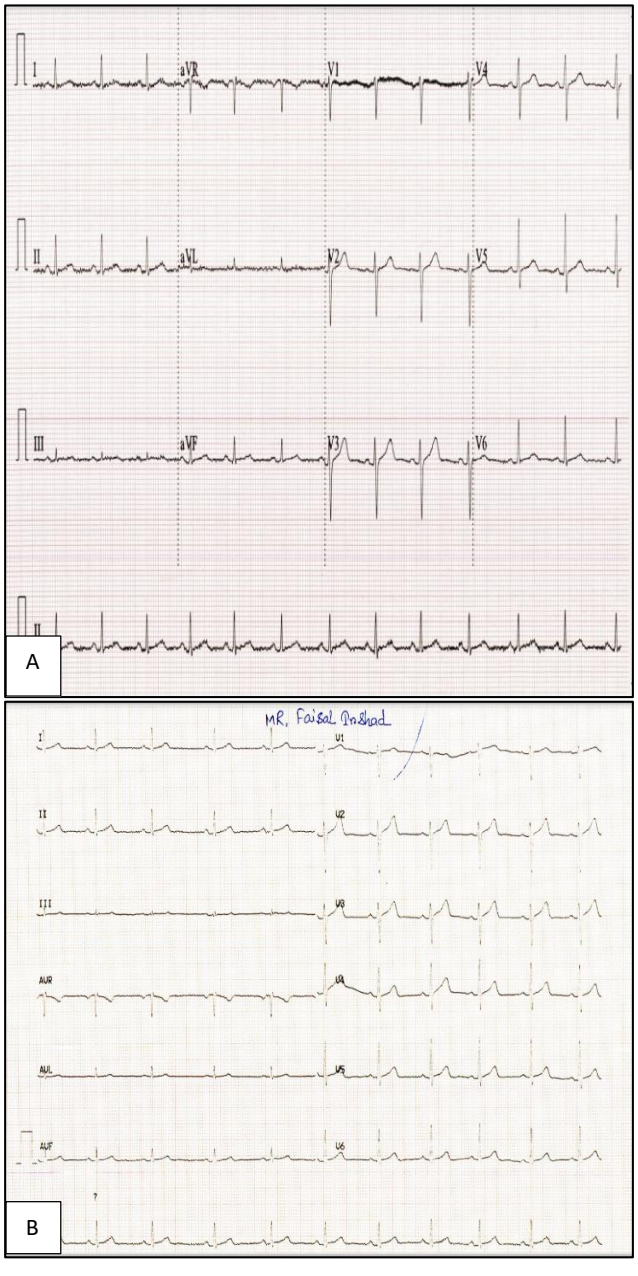

Figure 1 (A and B): Serial interval ECGs by 30 minutes reflects nonspecific dynamic ST changes predominantly over leads: ii, iii and a-VF suggesting inferior wall changes. ECG at presentation followed by repeat at 30 mins interval, showing dynamic ST changes.

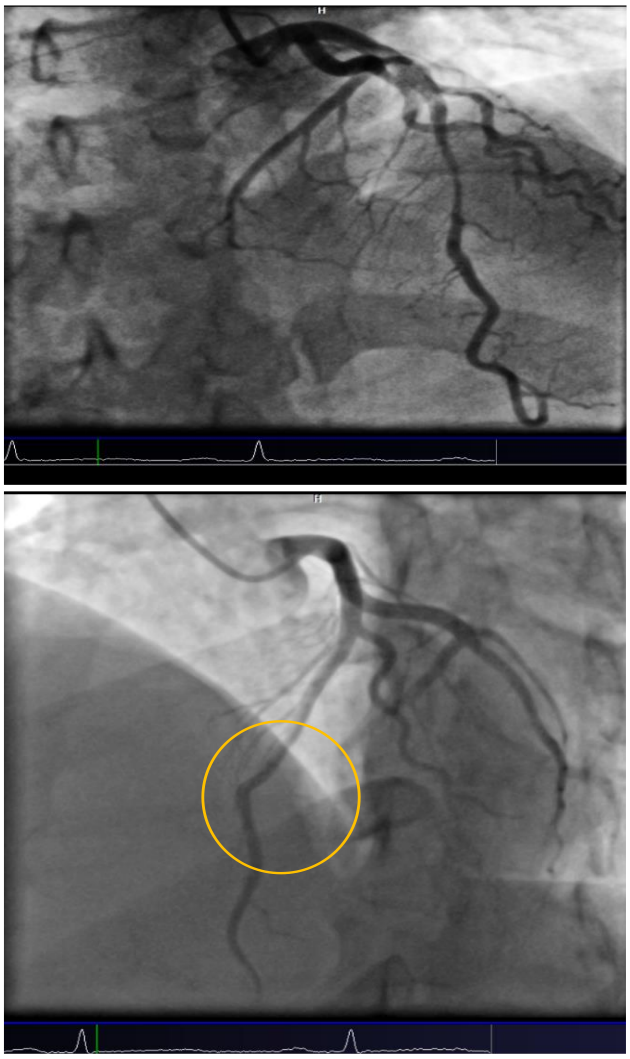

Figure 2: Coronary angiography in our patient displaying mid-segment compression of the left anterior descending artery (LAD), during systole (In circle).

DISCUSSION

The extent of cardiac complications and outcome in patients with myocardial bridging depend on the length and deeper grooving of the vessel into the myocardium as well as the involvement of arterial lesion which is most commonly found in the proximal segment due to decreased WSS intensifying endothelial injury.[5] Computational fluid dynamic study also revealed disruption in the antegrade systolic wave behind disturbance of blood flow pattern, amplifying further derangement in WSS and plaque formation.[6] Besides endothelial properties and alteration in surface molecular dynamics in the cadaveric studies of these cases have revealed significant changes with heightened endothelial adhesion molecular expression increased neutrophil recruitment which may trigger the destabilization of the atherosclerotic plaque, mostly in the proximal segment.[5] Interestingly histopathological examinations of bridging vessels in the cadaveric samples have also revealed the

inherent resistance towards the development of coronary atherosclerosis in the bridging segment, possibly due to epicardial perivascular adipose tissue associated production of proinflammatory cytokines and adipokines.[7] Such co-existence can be life threatening and can be associated with acute coronary syndrome (ACS), myocardial ischemia, arrythmias, atrioventricular (AV) conduction block, transient ventricular block; leading to sudden cardiac death.[8]

In our patient, without any remarkable cardiovascular history apart from long standing moderate hypertension on medication, was diagnosed with 25-30% lesion in the mid-LAD most likely from the myocardial bridging and loss of WSS. There no significant family history reflecting towards early Coronary disease to rationalise his coronary anomaly.

Although the patient did not show significant atherosclerotic changes on angiography but given his history of early cardiovascular disease in his siblings and his father, an inherent genetic polymorphic component or variance attributing towards the increased platelet and leukocytic activity cannot be completely ruled out.

CONCLUSION

So far, the clinical sequelae of myocardial bridging are significantly correlated with the extent of deeper grooving being affected by systole, gradient of loss of wall sheer stress (WSS) and percentage of atherosclerotic lesion in the proximal segment, as well as with the length of involvement. Hence any such presentation with atypical chest pain in absence of any sign in diagnostic parameters towards CAD needs an elective coronary angiography to identify the possibility of a bridging segment. Although the coronary angiography imaging cannot be interpreted to localise the depth of tunnelling, an adjunct non-invasive study such as multiple slices computed tomography (MSCT), cardiac-MRI, stress single-photon CT, intravascular ultrasound (IVUS) can be helpful to investigate the detailed nature of this segmental anomaly. Related literatures also suggest that a deeper invasion of more than 5 mm with a length more than 25 mm can hardly be managed by emergency surgical myotomy due to increased risk of bleeding, ventricular rupture and aneurysm formation. Rather a CABG is more beneficial in such extended cases of myocardial bridge. On this note it is equally important to include the possibilities of other non-atherosclerotic differentials of myocardial injury such as MINOCA (Myocardial infarction with non-obstructive coronary arteries), coronary artery dissection, spasmodic changes in coronary vasculature and microvascular angina. In this spectrum of non-atherosclerotic myocardial injury MINOCA manifests in patients with AMI but without any associated predilection of underlying coronary obstructive changes, not with any obstruction exceeding and got; 50%; devoid of any plausible explanation for this manifestation.

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