


**CLINICAL VIGNETTE**

**A dangerous bridge: myocardial infarction due to myocardial bridging in left ventricular hypertrophy**

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A 63-year-old woman with a history of hypertension presented to the coronary care unit with a subacute anterior wall myocardial infarction (MI). She reported of initial left-sided chest pain, dyspnoea, and weakness 18 h before hospital admission, during a long-distance ride in a rental car with air condition on a hot midsummer day after a minor car crash. The initial electrocardiogram (Panel C) revealed normal sinus rhythm, left axis deviation, left anterior hemiblock, inversion of the terminal T-waves in the anterolateral leads, ST-segment-depression in I, aVL, V5–V6, ST-segment-elevation in III and aVR, and obvious signs of marked left ventricular hypertrophy. Serum chemistry revealed elevated levels of cardiac troponin T (1.02 μg/L), creatine kinase (938 U/L), and lactate dehydrogenase (348 U/L), also suggestive for a subacute MI. In view of these diagnostic findings, the patient was referred for emergency cardiac catheterization, after receiving aspirin, heparin, a loading-dose of clopidogrel, and a beta-blocker.

Left ventricular angiography demonstrated a moderately depressed left ventricular systolic function with akinesia of the anterolateral and apical anterior wall (Panels A and B). The left ventricular end-diastolic pressure was mildly increased (18–20 mmHg). Coronary angiography exhibited ‘corkscrew appearance’ of all coronary arteries without significant atherosclerosis. Furthermore, myocardial bridging in the distal segment of the left anterior descending (LAD) artery with total systolic compression and almost complete resolution in the diastole was present (Panels A and B). There was normal antegrade flow (TIMI III) in the LAD distal to the bridging segment without evidence of thrombus. Because of the primarily benign prognosis of myocardial bridging, we decided upon a conservative treatment. The patient was discharged after an uncomplicated hospital course with a medication comprising a beta-blocker, a calcium antagonist, an ACE-inhibitor, and aspirin.

The present case illustrates that MI may be a specific complication of myocardial bridging. Particularly, the presence of left ventricular hypertrophy, additional catecholaminergic triggers, and/or increased thrombocyte activation, may contribute to the genesis of ischaemia in this predominantly angio-graphic diagnosis of a congenital coronary abnormality.

Panel A: End-diastolic left ventricular angiogram and angiogram of the left coronary artery in RAO 45° and LAO 90°.

Panel B: End-systolic left ventricular angiogram and angiogram of the left coronary artery in RAO 45° and LAO 90°. Compression of the distal LAD with total disappearance (arrows) in the systole.

Panel C: 12-channel-surface-ECG on admission, exhibiting a susacute anterior MI in the presence of left ventricular hypertrophy.

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