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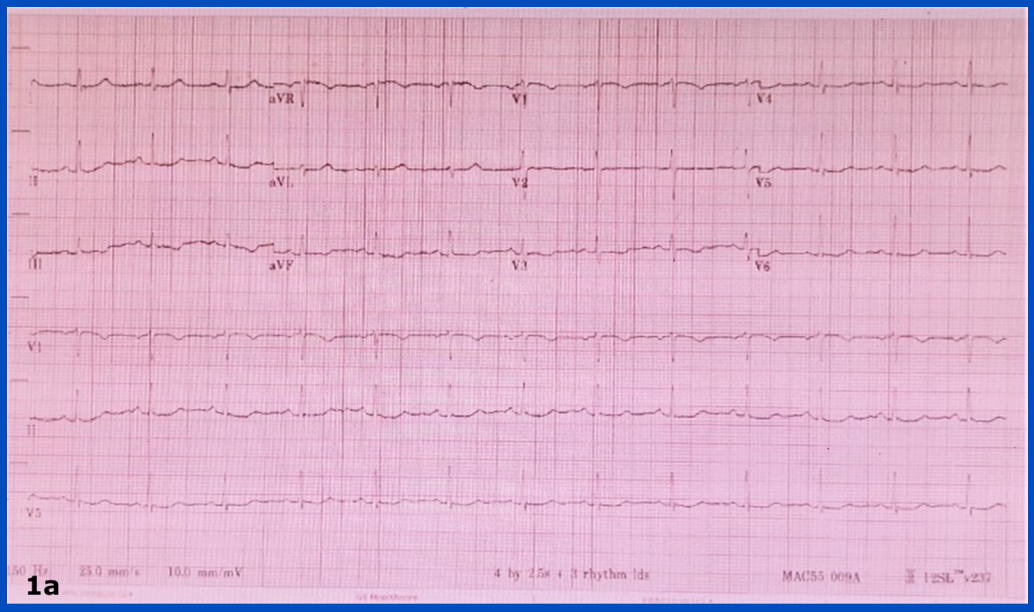

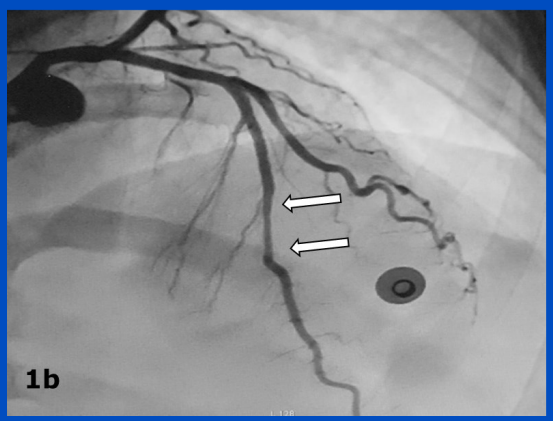

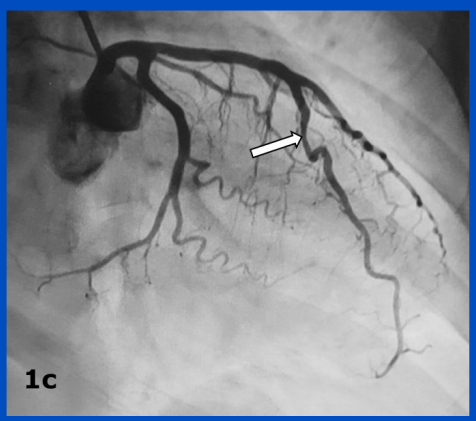

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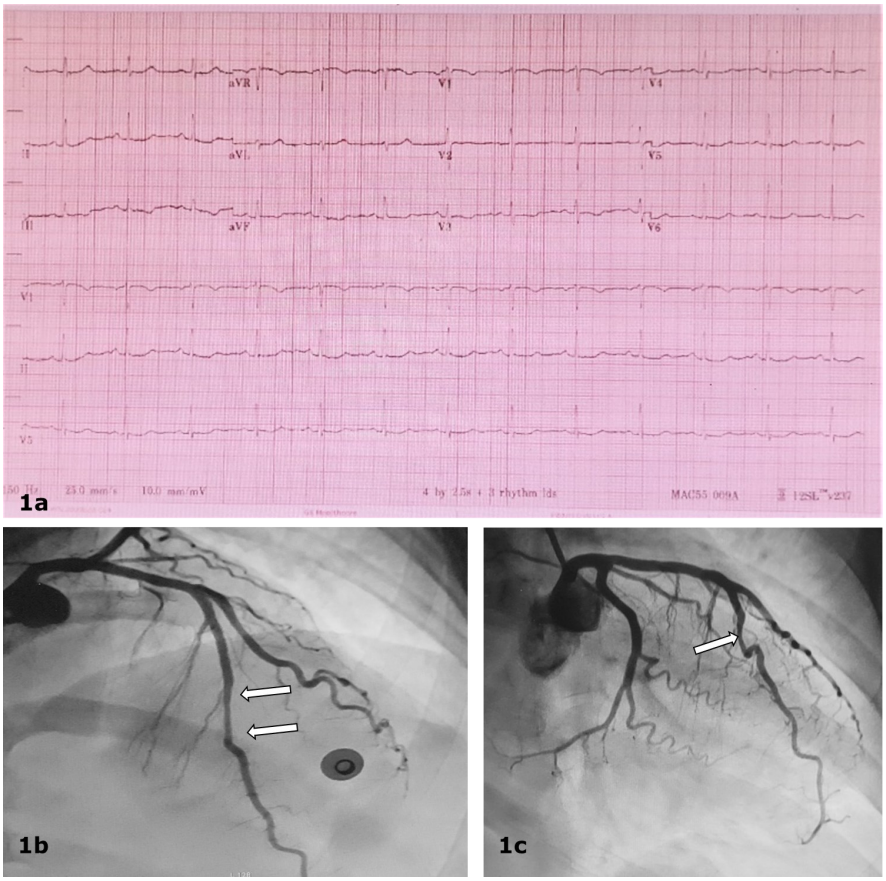

Figure 1

A 48-year-old lady with a history hypertension, iron deficiency anemia, ex-smoker and family history of coronary artery disease presented with sudden onset of epigastric pain. This was associated with radiation to the back and left shoulder, numbness along the left upper limb, giddiness and nausea. 12 leads ECG showed ST segment sagging in the inferior leads (III and aVF) and lead V3 (Figure 1a). Troponin I initially measured was mildly elevated at 32ng/L (normal range <30ng/L) but later increased to 836ng/L consistent with non-ST segment elevation myocardial infarction (NSTEMI). She was given aspirin, plavix and statin. Urgent coronary angiography was arranged (Figure 1b and 1c) which showed a smooth narrowed long segment of the mid left anterior descending artery (LAD).

What is the abnormality depicted in the coronary angiogram and what is the diagnosis?

***Answer**: refer to page 91*

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(Refer to page 90)

ANSWER: MYOCARDIAL BRIDGING OF MID LAD ARTERY RESULTING IN NSTEMI.

The coronary angiogram showed a narrowed (black and white arrows) segment of the mid left anterior descending (LAD) that is located a slightly lower plane due to myocardial bridging. The tunneling of the artery located at a lower plane is more obvious (black and white arrow) in Panel B. Black arrow in Figure A indicate the metal component of the ECG lead.

Myocardial bridge is a congenital abnormality where a segment of a major epicardial coronary artery runs intra-murally (tunneled artery) through the myocardium as depicted by angiogram in Figure 1c.[1,2] The incidence of myocardial bridging from an autopsy study of 90 hearts from patients who did not have a history of established heart disease or cardiac-related death was about 55% and the majority exclusively involved the Left anterior descending artery (LAD) in 70%, 40% involved the left circumflex artery and only 36% involved the right coronary artery.[1]

Intracoronary flow studies using quantitative coronary angiography and intracoronary Doppler imaging showed that intracoronary haemodynamic flow was characterized by phasic systolic compression with a localized peak pressure, persistent decrease in diastolic coronary diameter with a delay relaxation , an increase in blood flow velocities and retrograde flow, and a reduced flow reserve.[1] These haemodynamic flow abnormalities which are worsen with tachycardia, can lead to endothelial dysfunction secondary to turbulent blood flow with subsequent plaque formation just proximal and distal to the myocardial bridge.[1,2]

Patients with myocardial bridging, although present at birth, do not develop symptoms until after the third decade.[1] In 40% of patients, symptoms of angina can be brought on after administration of nitroglycerin or beta agonists.[1] Reported complications include angina pectoris, myocardial infarction, life threatening arrhythmias, and even sudden cardiac death.[1]

Diagnosis of myocardial bridging are usually confirmed by coronary angiogram, although non-invasive modality like multiple-slice computed tomography (MSCT) has also been used.[2] Intravascular ultrasound has also been utilized to provide physiological flow data and imaging of myocardial bridging coronary segment, which is typically characterized by the "half-moon sign".[2]

In most cases, myocardial bridge is non-significant and does not require any intervention. Treatment is mostly medical (first choice beta blockers).[3] Use of nitrates may worsen the symptoms and should be avoided. Percutaneous coronary intervention with intracoronary stents or surgical myotomy are alternative options should medical therapy failed.[3]

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