Acute posterolateral myocardial infarction diagnosed on contrast enhanced CT

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DESCRIPTION

A 66-year-old Caucasian woman was admitted to the emergency department at the weekend with sudden onset, central, crushing chest pain of 10/10 severity radiating to her left hand and neck, with associated nausea and vomiting. She reported that it was nothing like she had ever experienced before. She was an active smoker but otherwise her medical history included only hypothyroidism. Her initial ECG showed no changes to suggest myocardial ischaemia (figure 1). Baseline high-sensitivity cardiac troponin I (HscTnI) was elevated at 153 ng/L (0–16 ng/L).

Pain persisted despite sublingual nitrate and intravenous morphine administration and an intravenous nitrate infusion was commenced. She continued to deteriorate, developing pulmonary oedema and a new oxygen requirement of 15 L/min. HscTnI at 6 hours was 19810 ng/L. Repeated ECGs failed to demonstrate any changes suggestive of ischaemia aside from transient first degree heart block (figure 1). Treatment with dual antiplatelets (aspirin and clopidogrel) and fondaparinux was commenced for presumed non-ST-elevation myocardial infarction (MI). CT scan was performed and reported as showing no evidence of pulmonary embolism or acute aortic syndrome.

Chest pain remained persistent in absence of dynamic ECG change. On cardiology review the following day, CT images were revisited. An acute posterolateral myocardial perfusion defect was noted with preservation of the left ventricular myocardial wall thickness, supportive of a diagnosis of acute MI (figure 2). The patient was transferred to the regional cardiology tertiary centre for coronary angiography. This demonstrated a subtotal occlusion of the proximal left circumflex artery (LCx) which was treated with a drug-eluting stent.

She made good clinical progress and was discharged on the following day, CT images were revisited. An acute posterolateral myocardial perfusion defect was noted with preservation of the left ventricular myocardial wall thickness, supportive of a diagnosis of acute MI (figure 2). The patient was transferred to the regional cardiology tertiary centre for coronary angiography. This demonstrated a subtotal occlusion of the proximal left circumflex artery (LCx) which was treated with a drug-eluting stent.

She made good clinical progress and was discharged to the tertiary centre within the following few days. At last follow-up with the cardiac rehabilitation services 2 months after the index event, she remained well and reported good levels of exercise tolerance.

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The 12-lead ECG is commonly non-diagnostic in MI caused by a lesion in the LCx artery, and is regarded the least sensitive in LCx occlusions and infarcts of the interolateral wall compared with MIs in other coronary artery territories. Often, as was the case here, an LCx-territory STEMI may present without high lateral ST-elevation.

In the setting of ischaemic acute chest pain without ECG changes where CT thorax is performed to investigate the cause of pain, recognition of an acute myocardial perfusion deficit may help support the diagnosis of acute MI and bolster referral for early coronary angiography.
Images in…

Learning points

► Not all myocardial infarctions (MIs) have typical ischaemic changes on a standard 12-lead ECG. ECG changes are most commonly absent in MI caused by lesions of the left circumflex artery.
► Reduced myocardial perfusion on a non-cardiac CT can help support the diagnosis of MI.

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