Inferolateral myocardial infarction? Which vessel is occluded and where?

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Answer to the rhythm puzzle

This is an acute myopericarditis due to parvovirus B19 infection. The most important electrocardiographically distinct features are the PTa segment depression (lead I, II, aVL), the PTa segment elevation (lead aVR) and the absence of ST-segment depression over the anterior precordial leads. The coronary angiogram was normal and the cardiac magnetic resonance images showed inferolateral and septal Gadolinium contrast enhancement in the mid- and epi-cardial regions and the absence of subendocardial involvement compatible with myocarditis.

Fig. 1 Twelve-lead electrocardiogram, at presentation, showing ST-segment elevations in leads I, II, III, aVF and V4–6

Atrial repolarisation starts after the atrial depolarisation (P wave) and continues into the QRS complex. The repolarisation potentials of the atria are much smaller than of the ventricle and usually not noticeable on the surface ECG. Because of this, the interval between the end of the P wave and the Q (or R) wave is usually isoelectric. However, atrial repolarisation events can be detected in some patients with AV block [1]. Atrial repolarisation spreads in the opposite direction of the depolarisation and lasts at least twice as long. [1]. This atrial T wave (Ta) increased when the P wave amplitude is increased. Other causes of atrial repolarisation abnormal-

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Fig. 2 Twelve-lead electrocardiogram a few days after admission showing negative T-waves in leads I, II, III, aVF and V4–6

ities (PTa segment deviation) are pericarditis, atrial infarction or ischaemia, rupture of the atrial wall, atrial aneurysm, and atrial tumour [1].

Acute myocarditis has been reported in up to 12% of young adults presenting with sudden death [2] and can be an important underlying cause of acute heart failure or a chronic dilated cardiomyopathy [3]. There are various infectious causes of acute myocarditis, but viral agents, such as enteroviruses, parvovirus B19 and human herpes virus-6, are the most frequent [4]. The pathophysiological process of acute myocarditis can be summarised as direct myocardial invasion of cardiotropic viruses or other infectious agents rapidly progressing to immunological activation and activation of CD4⁺ with clonal expansion of B cells, resulting in local inflammation and further myocytolysis [5].

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