

An inferior myocardial infarction with conduction abnormalities



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A 73-year-old man was admitted to hospital 20 hours after the onset of acute chest pain. The electrocardiogram (ECG) on admission is shown in Figure 1.

Questions

1. What is the location of different conduction abnormalities?
2. In which coronary artery and where is the occlusion located?

Discussion

1. A sinus tachycardia is present with a frequency of 112 beats/min (PP interval 540 ms). At that sinus rate, the PR interval measures 240 ms and is therefore definitely prolonged. In addition, as indicated under lead aVF, in the middle of the tracing there is sudden doubling of the PP interval (1080 ms), suggesting a 2:1 sinoatrial block. The P wave after the pause is conducted to the ventricle

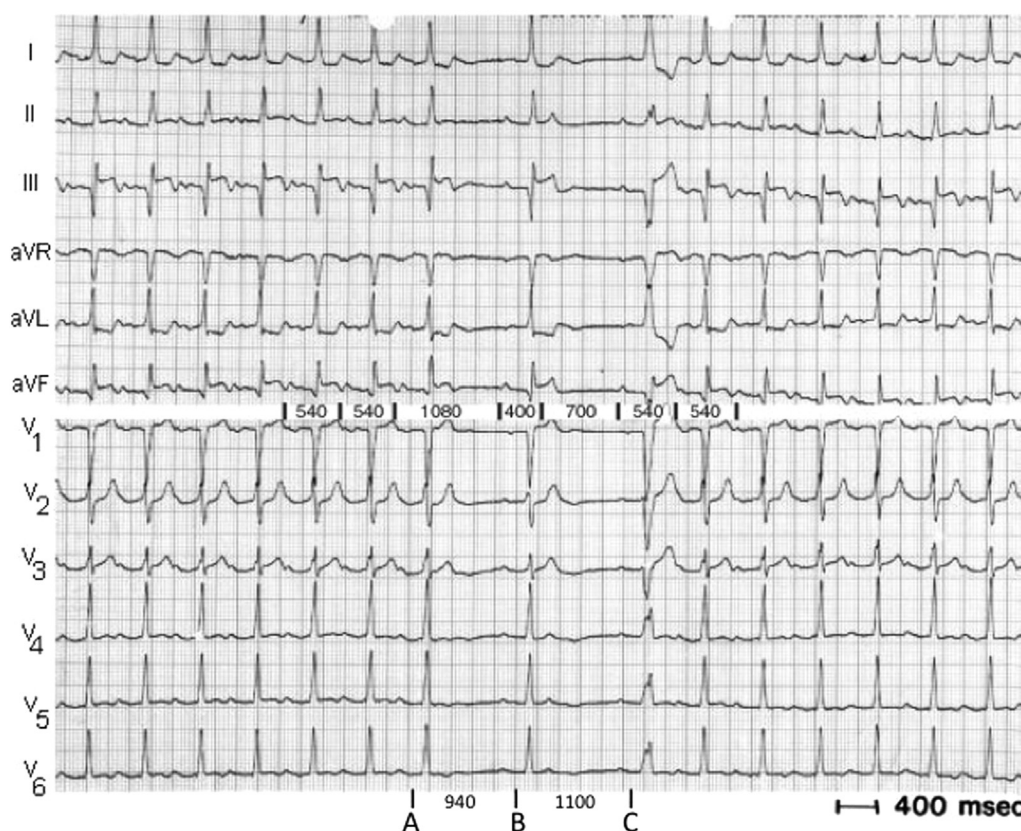


Figure 1 The electrocardiogram shows sinus tachycardia with sinoatrial and atrioventricular conduction disturbances in the setting of an inferior myocardial infarction. Also bradycardia-related phase 4 block in the left bundle branch is present. See text for analysis.

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and followed by an atrial premature beat after 400 ms, which is not conducted to the ventricle; 700 ms later, a sinus P wave is conducted to the ventricle but with a

QRS complex showing a left bundle branch block configuration. That QRS complex, labeled C in [Figure 1](#) under lead V₆, occurs 1100 ms after the previous narrow QRS complex, labeled B (the B-C interval). QRS complex B is preceded by a narrow QRS complex by 940 ms (the A-B interval). Therefore, the occurrence of left bundle branch block in QRS complex C can be explained by bradycardia-related phase 4 block in the left bundle branch (B-C interval > A-B interval). This is not an unusual finding during bradycardia in inferior myocardial infarction (IMI).¹

2. The Q waves in leads III and aVF and the ST-T segment findings indicate an IMI in the subacute phase. The culprit coronary artery (right or circumflex coronary artery?) and the location of the occlusion site in the artery should be based on ST-T-segment deviation changes and the type of conduction disturbances.

ST-segment elevation in lead III more than that in lead II as well as ST-segment depression in lead I indicate the right

coronary artery (RCA) as the culprit. The delay in atrioventricular nodal conduction and the episode of sinoatrial block tell us that the occlusion site must be proximal in the RCA.

In addition, the positive T waves in the precordial leads V₁–V₃ indicate the presence of a right ventricular infarction pointing to an RCA occlusion site before the takeoff of the right ventricular branch.

Although the clinical history and ECG findings indicate a subacute phase of IMI, the presence of sinus tachycardia pointing to poor hemodynamic tolerance and the pattern of ST-segment deviation was the reason to take the patient to the catheterization room immediately after admission. A proximal occlusion in a dominant RCA was found. This was followed by PTCA and stenting.

Reference

1. Lie KI, Wellens HJ, Schuilenburg RM, Durrer D. Mechanism and significance of widened QRS complexes during atrioventricular block in acute inferior myocardial infarction. *Am J Cardiol* 1974;33:833–839.