A case of myocardial infarction with conduction abnormalities

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Case presentation

A 56-year-old man with history of hypertension, diabetes mellitus type 2 and chronic kidney disease is evaluated in the emergency department because of nausea, abdominal pain, excessive sudation and light-headedness for the past 4 hours. Physical examination is unremarkable. The following ECGs (figs. 1 and 2) are obtained.

What is your diagnosis? How would you manage this patient?

Solution

See figures 3 and 4.

There is an irregular rhythm with a ventricular rate of 61 bpm. QRS complexes are narrow. Big amplitude T-waves are seen in V1 to V3 (circled). There is significant ST segment elevation in leads II–III, aVF and V1 (red arrowheads) with reciprocal ST segment depression in leads aVL, V5 and V6 (black arrowheads). An increased number of P waves (black and red arrows, corresponding to conducted and non-conducted P waves, respectively), compared to the number of QRS complexes (green arrows), can be counted. The PP interval is regular. An increasing PR interval (red lines with increasing length) is measured, with a resulting non-conducted P wave (second red arrow and terminated red line), compatible with a Wenckebach phenomenon. The non-conducted P wave is merged in the QRS complex and can be better visualised in V2 and V3 (leftward bending red arrows). The following QRS complex (green arrowhead) results from conduction with a very long PR interval or represents a junctional escape beat. The PR interval of the preceding P wave is indeed too short to result in atrioventricular (AV) conduction. Thereafter, a 2:1 block occurs for two cycles (underlined in black), followed by a repeat Wenckebach phenomenon (red lines with increasing length and finally terminated). The blocked P wave is hidden in the QRS complex and can...
be better spotted in V5 and V6 (rightward bending red arrows).

Based on these ECGs, an inferior ST segment elevation myocardial infarction (STEMI) with associated AV nodal conduction abnormalities is suspected. The ST-segment elevation in lead III>II, associated with an ST segment elevation in V1, suggests an associated right ventricular infarction [1, 2]. Additionally, a second degree Mobitz type I AV block with 2:1 ventricular conduction is present. An intranodal rather than infranodal AV block is suggested, due to the prolonged PR interval and documented Wenckebach phenomenon with 3:2 conduction [3]. A proximal-to-mid right coronary artery (RCA) lesion is probable, with resulting ischaemia of the right ventricle, as well as the AV nodal artery perfusion area.

The catheter lab should be immediately activated and further management should follow the STEMI guidelines. A right-sided 12-lead ECG should be obtained to confirm right ventricular infarction, and pre-load reducing drugs (opiates, glyceryl trinitrate) used with caution. Finally, due
to second degree AV block, use of AV blocking drugs (beta-blockers, calcium channel blockers) should be avoided. This is particularly relevant when the transradial approach is selected for percutaneous revascularisation.

Coronary angiography confirmed an inferior wall STEMI due to a thrombotic occlusion of the mid-RCA (Fig. 5A). Figure 5B shows the final angiographic result after balloon angioplasty and stenting with a single drug-eluting stent, with subsequent reperfusion of the AV nodal artery.

A further ECG (Fig. 6) obtained 1 hour after percutaneous coronary intervention shows a regular sinus rhythm with a heart rate of 87 bpm and first degree AV block (AV interval 232 ms). Early recovery from the ischaemia-related 2:1 AV block after successful recanalisation of the RCA is observed.

High-degree AV block can be a complication in up to 7% of patients with inferior STEMI, and about 9% of patients with STEMI require a permanent pacemaker implantation due to persistent or recurrent high-degree AV block [4]. In patients with second degree, Mobitz type I AV block, no specific therapy is needed other than ischaemia-related therapy. Atropine should be used in symptomatic patients with a ventricular rate that does not exceed 50 bpm, with premature ventricular complexes and/or associated bundle branch blocks or signs of heart failure [3]. Temporary pacing therapy is rarely needed.
An anteroseptal wall myocardial infarction can also cause AV conduction abnormalities due to ischemia in the perfusion zone of the septal perforators of the left anterior descending artery. However, in this case, ischaemia would involve the infranodal conduction system (the conduction system below the AV node and the bundle of His), which would predominantly result in a second degree, Mobitz type II AV block. As this situation increases the potential for progression to complete heart block, temporary pacing should be considered [3].

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