

OBSERVATIONS

The obvious abnormalities are the tachycardia and ST segment changes.

Systematic analysis reveals (Standard calibration)

■ Rate and rhythm

A tachycardia of 120 bpm originating from the sinus node giving positive P waves of normal shape in leads I, II and aVF.

■ PR interval

160ms (normal is 120-200ms) with no variation

■ QRS width

80ms (normal is up to 100ms or 2.5 little blocks)

■ QRS axis

Approximately 45° (normal in adult is -30 to +90°)

■ QRS morphology

Normal with no pathological Q waves and normal R wave progression in chest leads.

■ ST segments

Elevation in leads I, II, aVL, aVF, V2-V6 up to 4mm and depression in aVR of 2.5mm

■ T waves

Unremarkable: upright in the all the chest leads except VI (which is normal). In the limb leads, the T wave axis is 60° (which should be within 45° of the QRS axis and which it is).

■ QT interval

7x small blocks (7x40ms)= 280ms which gives a QTc corrected to a standard rate of 60 bpm of 395ms. (Normal in males is ≤ 440ms.)

DIAGNOSIS AND DISCUSSION

This case presentation suggests a number of possible diagnoses: myocardial ischemia / infarction, pulmonary embolism, pericarditis, even non-specific cardiac pain associated with tachycardias and also non-cardiac pain. This ECG is diagnostic.

Could the ECG be in keeping with myocardial ischemia as in an acute coronary syndrome presentation requiring heparin or myocardial infarction of the ST segment elevation type requiring a thrombolytic or PCI in the cath lab? Certainly, there is ST segment elevation. However:

■ The elevation is widespread, occurring in 5 of the 6 chest leads and in 4 of the limb leads, also with ST segment depression in aVR. Thus, there are only 2 leads with iso-electric STs. If due to myocardial ischemia, it would suggest extensive involvement affecting multiple arterial territories. This would be rather unusual in a young man who would most likely have single vessel coronary artery disease.

■ The elevated ST segments, of which V5 has good examples, have a sag in the middle, which is not typical of ischemia, in which it should be coved upward.

■ There is no accompanying loss of R waves and, in particular, also no abnormality (peaking or inversion) of the T waves, which is unusual for extensive ischemia. (Furthermore, T wave observations in acute myocardial infarction are not static and would evolve over a period of hours. Thus, a repeat ECG 6 hours later showing no change or evolution of T waves, whether treated or not, would not be in keeping with myocardial infarction.)

So, myocardial ischemia and infarction are unlikely. A diagnosis of pulmonary embolism always needs to be considered when someone presents with chest pain and dyspnea. The ECGs, if there are any

abnormalities, usually have only relatively minor changes affecting predominantly the RV. Apart from sinus tachycardia, this ECG is not in keeping with pulmonary embolism.

It is not uncommon for patients with rapid supraventricular tachycardias (SVT) such as AV junctional re-entry tachycardias and those associated with WPW, to experience atypical chest pain. This ECG does not show any such pathological SVT.

Acute pericarditis can account for all the ECG observations: sinus tachycardia, widespread ST abnormalities without R wave or significant T wave changes and the "saddle-back" midsegment sagging of elevated ST segments. Some additional typical changes confirm this diagnosis. Pericarditis usually affects the entire pericardium, which covers both ventricles and atria. True pericarditis causes no ECG morphological abnormalities. It is the inflammatory myocarditis just under the pericardium, i.e. a myopericarditis, which gives the ECG changes. The inflammatory process overlying the atria may cause repolarization changes similar to those arising from the ventricles. The repolarization of the atria is usually ignored because it is only rarely abnormal, even if there is true atrial myocardial infarction, which itself is extremely rare. This atrial repolarization, or the equivalent of the ST segment of the P waves, occurs in the PR segment, after the end of the P wave up to the beginning of the QRS. This should be isoelectric with the reference baseline, which is always taken to be the T to P segment. In this patient's ECG, one can observe in lead II that the PR segment is depressed below the TP segment and, almost pathognomonicly, in lead aVR the PR segment is elevated after the inverted P wave (Figure 1).

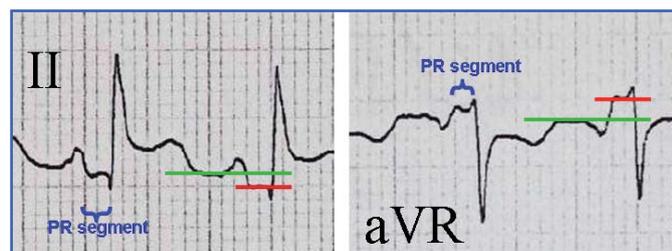


FIGURE 1: PR segment abnormality in Lead II and Lead aVR, the leads most typically affected: The reference baseline is the TP segment (green lines). Note the PR segment deviations (red lines) relative to the baseline, with depression in lead II and elevation in lead aVR.

CONCLUSION

One should examine the ECG for these PR / atrial repolarization changes because, whilst not seen in all cases of acute (myo-)pericarditis, if present, and occurring together with the typical widespread ST segment changes, they are strong confirmation of the diagnosis.

The treatment of uncomplicated acute pericarditis is analgesia (answer "c").