

## Half the Story

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A 71-year-old woman presents to the emergency department complaining of severe chest pain, which began two hours earlier. Figure 1 shows her initial ECG.

1. *What is the principal diagnosis?*
2. *What other abnormality is shown?*

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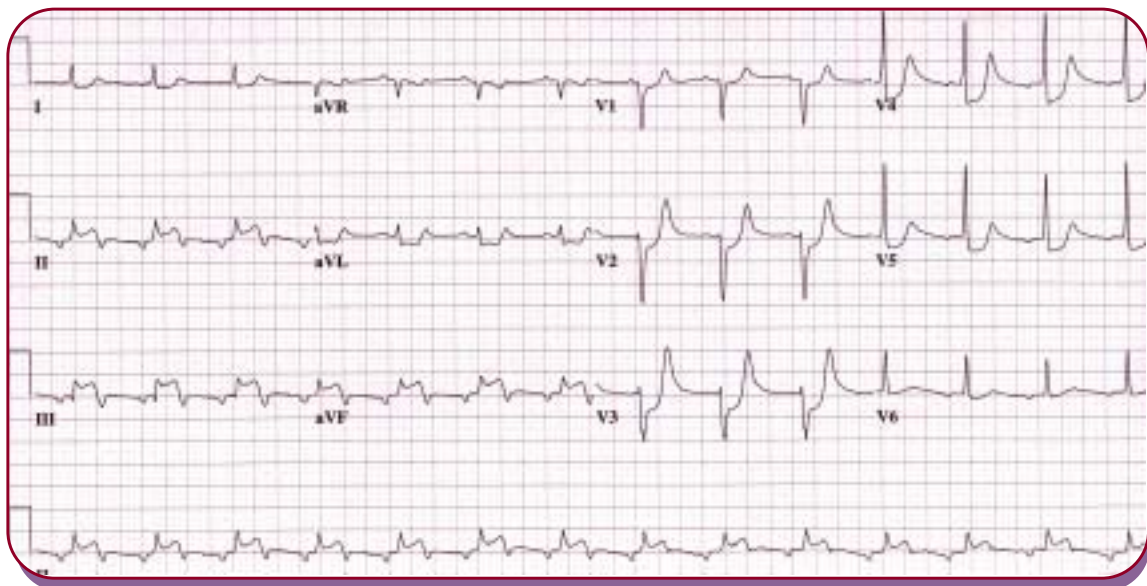


Figure 1. ECG upon presentation.

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## This Month's ECG Diagnosis

**1.** The initial ECG shows a heart rate of 80 beats/minute, normal QRS duration, and ST segment elevation in leads II, III, and aV<sub>F</sub>, indicating acute inferior transmural or epicardial injury.


Leads V<sub>2</sub> through V<sub>5</sub> show marked ST segment depression and prominent, peaked T waves. These latter changes suggest either acute anterior subendocardial injury or, more likely, acute posterior wall epicardial injury.

A repeat ECG, with posterior leads, confirmed the presence of posterior wall injury. Reperfusion therapy was administered promptly.

**2.** In addition to the diagnosis of acute infero-posterior myocardial infarction (MI), an arrhythmia is also present. The P waves are inverted in leads II, III, and aV<sub>F</sub>, and not upright, as one would expect if the rhythm was sinus in origin.

Upon further scrutiny, it becomes apparent there are two P waves for every QRS complex (the second P wave being partially buried in the tail end of the T wave). The rhythm is atrial tachycardia with 2:1 conduction; the absence of 1:1 conduction not only suggests atrioventricular (AV) conduction is impaired, but also practically excludes a re-entry circuit involving the AV node as the etiology. The most likely mechanism is an ectopic atrial tachycardia, perhaps the result of associated atrial ischemia or infarction.

Figure 2 shows an ECG obtained one hour later. There has been an encouraging reduction in the magnitude of ST segment elevation and, in keeping with the persistent impairment of AV node conduction, the rhythm is now sinus with 2:1 AV conduction.

Such conduction disturbances are common in the setting of acute inferior MI, due primarily to the activation of vagal receptors and sometimes to associated AV node ischemia, and can be expected to resolve spontaneously within two to three days. 

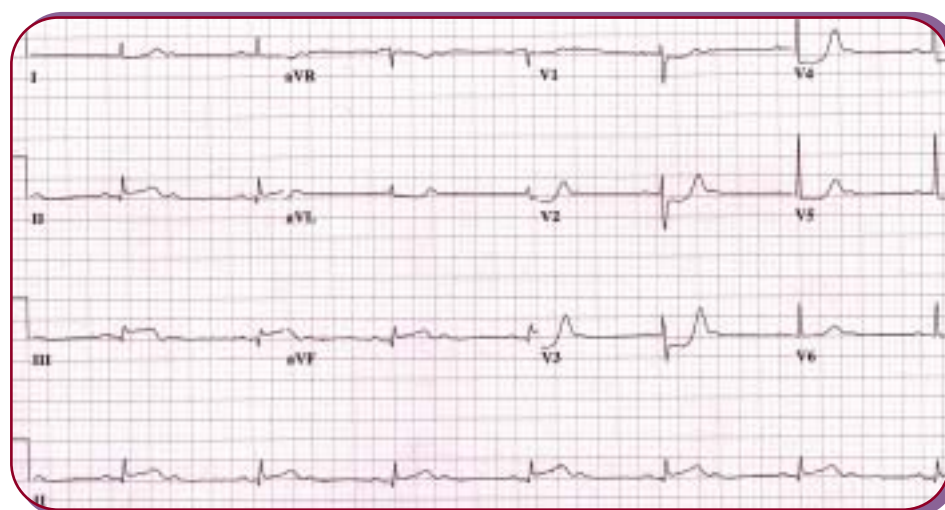


Figure 2. ECG obtained one hour after presentation.