



# The Clinical Picture

## Worsening shortness of breath

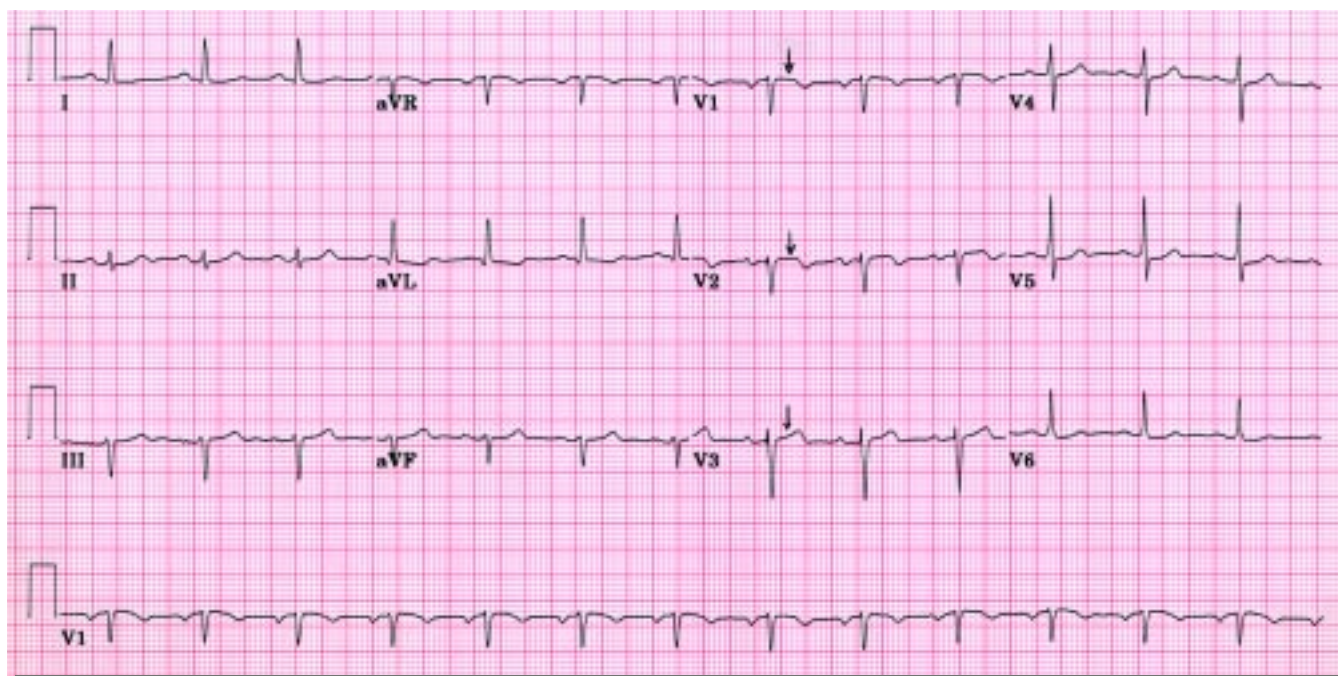


FIGURE 1. Electrocardiogram on presentation.

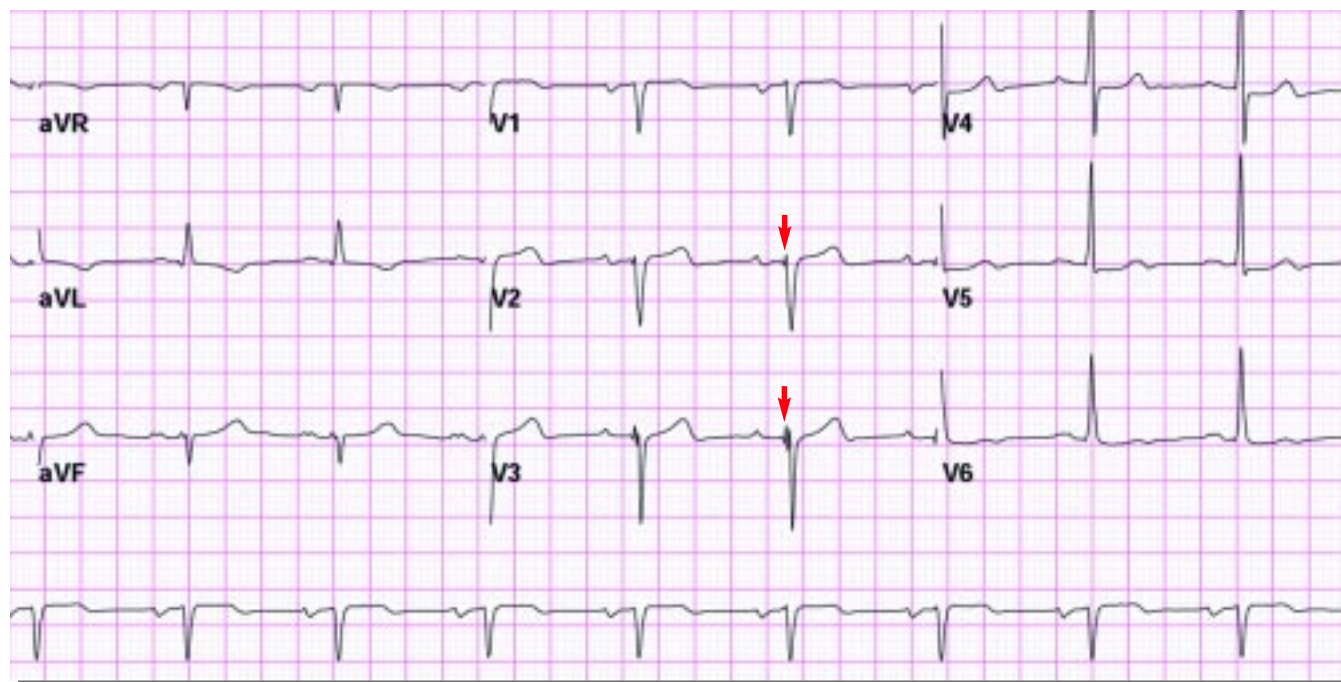
**A** 58-YEAR-OLD MAN with a long history of poorly controlled type 2 diabetes mellitus presents to a hospital emergency room with shortness of breath that began acutely 2 hours previously and has rapidly worsened. He reports no associated chest or neck pain. He states that his health has otherwise been excellent, and that he exercises regularly including a long-distance walking program.

Physical examination: blood pressure 132/80 mm Hg, pulse 68 beats per minute and regular, bibasilar wet rales, jugular venous pressure normal at 45 degrees, no carotid bruits, point of maximal impulse normal, heart sounds diminished but otherwise normal.

The electrocardiogram on presentation (FIGURE 1) shows ST segment elevation in leads V<sub>1</sub>, V<sub>2</sub>, and V<sub>3</sub> (arrows). The chest radiograph shows pulmonary edema, on the basis of which he is admitted to the hospital. Subsequent cardiac enzyme assays are positive, confirming acute myocardial injury.

Heparin, tirofiban, aspirin, furosemide, and metoprolol are started. Thrombolytic therapy is not administered. The patient is deemed medically stable and is transferred to our hospital. Cardiac catheterization is planned for the next day.

An electrocardiogram is obtained 12 hours after admission to our hospital (FIGURE 2).



**FIGURE 2.** Electrocardiogram 12 hours after admission.

**Q:** What is the most important new finding on this electrocardiogram?

- ☐ Ectopic atrial rhythm
- ☐ Left axis deviation
- ☐ Persistent ST segment elevation
- ☐ “Splinter” q (qr) waves

**A:** The new electrocardiogram shows normal sinus rhythm with a normal P wave axis, a regular PP interval, and a rate of 70 per minute. The axis of the QRS complex in the frontal plane is deviated slightly to the left but has not changed. There are nonspecific ST and T wave changes in the precordial and lateral limb leads, but the initial ST segment elevation has nearly resolved. The most important new finding is the development of prominent qr waves, also known as “splinter” q waves, in leads  $V_2$  and  $V_3$  (FIGURE 2, arrows).

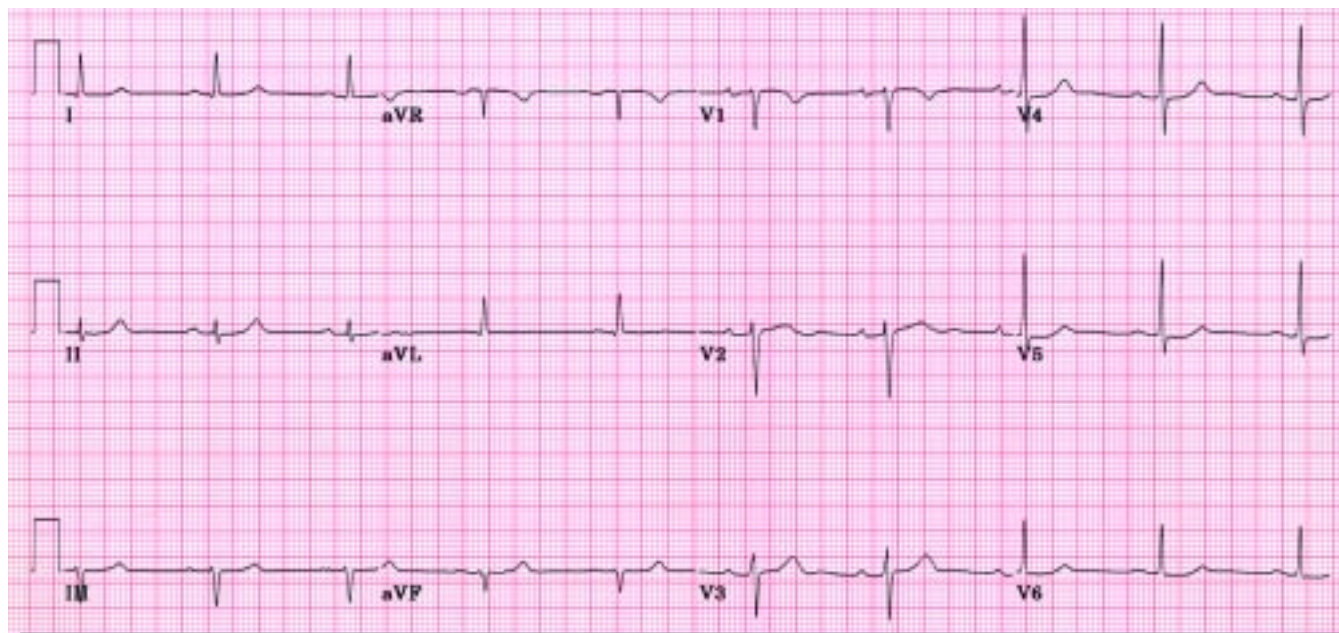
**Q:** The interval development of splinter q waves in leads  $V_2$  and  $V_3$  supports which diagnosis?

- ☐ Myocardial ischemia
- ☐ Left anterior hemiblock
- ☐ Myocardial infarction
- ☐ Pulmonary embolism

**A:** Splinter q waves are an often overlooked and highly specific sign of myocardial necrosis. They are typically located in leads  $V_2$  and  $V_3$  and reflect the distribution of the left anterior descending coronary artery. How they develop is unclear, but most likely a necrosis vector modifies the “septal” r waves normally present in the right precordial leads.

Although not a common presentation of myocardial infarction, splinter q waves often indicate a flow-limiting stenosis of the left





**FIGURE 3.** Electrocardiogram 1 month after admission.

anterior descending coronary artery and prior myocardial infarction, both of which merit further clinical investigation.


As for the other answers, left anterior hemiblock may manifest q waves in the right precordial leads, especially in tall, thin patients and in those with emphysema; however, these q waves are generally associated with left axis deviation greater than  $-30^\circ$  and disappear when the precordial leads are placed in a lower interspace.

Acute pulmonary embolism often manifests with a pattern of right bundle branch block, right-axis deviation of the QRS complex, ST segment depression, and T wave inversion, none of which were found in this patient. The classic frontal-plane QRS changes—ie, a new S wave in lead I, and q wave development with T wave inversion in lead III—occur only in about 15% of patients. In rare cases, acute pulmonary embolism may mimic an anteroseptal myocardial infarction with new q waves in leads  $V_1$  and  $V_2$ .

Myocardial ischemia often coexists with myocardial infarction. However, it does not

explain by itself the development of splinter q waves.

### Case continued

The patient undergoes diagnostic left heart catheterization, which demonstrates a subtotal occlusion of the proximal left anterior descending coronary artery. Percutaneous transluminal coronary angioplasty with stent placement is successfully performed. An electrocardiogram taken 1 month later (**FIGURE 3**) shows resolution of the splinter q waves and no evidence of myocardial infarction. 

### SUGGESTED READING

**Parker AB, Waller BF, Gering LE.** Usefulness of the 12-lead electrocardiogram in detection of myocardial infarction: Electrocardiographic-anatomic correlations, part 1. *Clin Cardiol* 1996; 19:55–61.

**Rimmerman C, Jain A.** Interactive electrocardiogram CD-ROM with workbook. Philadelphia: Lippincott Williams and Wilkins, 2001.

**Schamroth L.** The 12-lead electrocardiogram. Oxford: Blackwell Scientific, 1989.

**ADDRESS:** Curtis M. Rimmerman, MD, Department of Cardiovascular Medicine, F15, The Cleveland Clinic Foundation, 9500 Euclid Avenue, Cleveland, OH 44195; e-mail [rimmerc@ccf.org](mailto:rimmerc@ccf.org)