

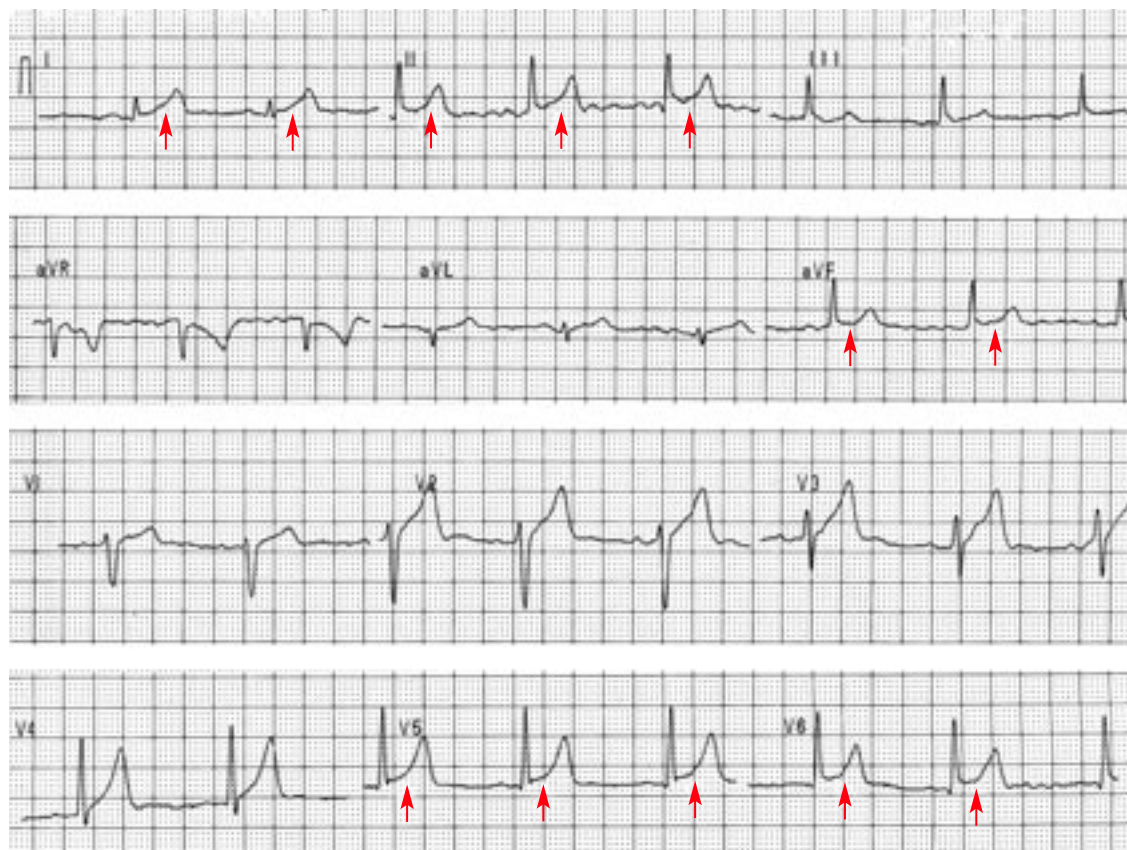


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# The Clinical Picture

## Severe chest pain in a 32-year-old man



**FIGURE 1.** Electrocardiogram (ECG) at time of presentation. ST-segment elevation with upwards concavity, best seen in leads I, II, aVF, V<sub>5</sub>, and V<sub>6</sub>. No reciprocal changes are seen.

**A** 32-YEAR-OLD MAN PRESENTS with a complaint of severe chest pain that lasted for 1 hour and radiated to the right shoulder. He denies that the pain was relieved by changes in position or in inspiration. His serum creatine kinase is 2,950 U/L (normal range 50–200 U/L).

Note that the electrocardiogram (ECG) (FIGURE 1) shows ST-segment elevation in leads

I, II, aVF, V<sub>5</sub>, and V<sub>6</sub>, but no reciprocal changes in the leads facing the negative poles of these vectors. ST-segment elevation with no reciprocal ST-segment changes is characteristic of pericarditis as opposed to acute anterior myocardial infarction.

The lack of reciprocal changes in pericarditis is due to diffuse involvement of the pericardial sac, with all ST-segment vectors



**FIGURE 2.** Follow-up ECG 2 weeks later shows resolution of the ST-segment elevation.

pointing towards the epicardium. In acute myocardial infarction, on the other hand, the ST-segment vectors point only to the area of the infarct, thus causing reciprocal changes in the leads that face the negative pole of that vector and ST-segment elevation in the vectors pointing towards the “injury zone”—ie, the positive pole of the vector.

The author saw the patient 2 weeks after his initial presentation. At that time he admitted that taking a deep breath made the pain worse.

Treadmill testing for 12 minutes (Bruce protocol) produced no chest pain, and a follow-up ECG was normal (**FIGURE 2**). The elevation in serum creatine kinase was presumed to be due to concomitant myocarditis.

From this case it is clear that the ECG can be persuasive for pericarditis even when a classic clinical picture is not present. Follow-up tracings are mandatory to confirm the diagnosis.

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