Not all ST-segment elevation myocardial infarctions become Q-wave myocardial infarctions

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Figure 1. Electrocardiograms recorded just before midnight (i.e., soon after the patient's arrival in the emergency department) and each of the following 2 mornings.

A 60-year-old man came to the emergency department complaining of a sensation of severe midsternal pressure that persisted for 30 minutes after awakening him. He had first noted chest pressure several weeks earlier, when it would come with exertion and be relieved with rest. Over the previous several days, the discomfort had occurred intermittently at rest. The current episode was by far the longest and most severe, was unaccompanied by other symptoms, and spontaneously disappeared as soon as the patient arrived in the emergency department. The history further revealed good health in the past, 2 to 3 packs of cigarettes per day for 40 years, and no medications. Physical examination findings were unremarkable except

for a blood pressure of 180/90 mm Hg.

An electrocardiogram (Figure 1) recorded soon after the patient's arrival in the emergency department shows sinus bradycardia (rate, 59 beats/min). Broad, negative, terminal P-wave deflections in lead V₁, diphasic ($\pm$) P waves in lead III, and broad notched P waves in leads V₂–V₆ suggest left atrial enlargement. The deep S waves in leads V₁ and V₂ and the tall R waves in V₅ and V₆ are not quite diagnostic of left ventricular enlargement, but in the presence of left atrial enlargement, they suggest that interpretation. The most important electrocardiographic findings are the broad, tall T waves in leads V₂–V₄, together with the slight, but definite, ST-segment elevation in those leads: 2.5 mm in V₂, 2 mm in V₃, and 1 mm in V₄. The following day, the ST-segment elevation had resolved slightly, and terminal T-wave inversion can be seen following an acute-angle turn from the peaks of the T waves in leads V₂ and V₃. As

pointed out by Wellens' group (1), this T-wave change in the anterior precordial leads usually occurs after the chest pain has subsided and indicates severe narrowing of the left anterior descending coronary artery, often proximally. On day 3, ST seg-

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Table. Cardiac enzymes

Day, hour	CK (U/L)	CK-MB (ng/mL)	Troponin I (ng/mL)
Day 1, 22:00	132	2.5	0.10
Day 2, 04:30	214	20.0	3.30
Day 2, 10:00	209	17.5	2.60

Normal values are as follows: CK, <176 U/L; CK-MB, <4.0 ng/mL; and troponin I, < 0.05 ng/mL. CK indicates creatine kinase; CK-MB, MB isoenzyme of CK.

ments are nearly isoelectric; T waves are deeply inverted, so-called Pardee T waves (2), in the anterior precordial leads with lesser changes in leads I, II, aVF, V₅, and V₆. No Q waves are seen, and there has been no decrease in R waves in the anterior precordial leads.

Among patients with acute coronary syndromes, those with prolonged pain and persistent ST-segment elevation are classified as having an ST-segment elevation myocardial infarction, and in the absence of a contraindication, they receive reperfusion therapy, either thrombolysis or angioplasty. Without reperfusion, most such patients will develop Q waves and have sizable infarcts. All other patients with acute coronary syndromes are said to have unstable angina. This is a heterogeneous group that ranges from those with no electrocardiographic or enzymatic change, and thus no evidence of current myocardial damage and a low risk of infarction or death within the next 30 days, to those with electrocardiographic or enzymatic change, each of which has a sizable adverse effect on prognosis (3).

In patients with unstable angina, the earliest electrocardiographic change associated with ischemia often is ST-segment depression or, as in this patient, transient elevation accompanied by upright, often accentuated, T waves in the affected leads. T-wave inversion often follows the ST-segment change. Thus, the electrocardiographic pattern depends on when in the course of ischemia the patient is seen. Striking ST-T changes, such as those that occurred in this patient, usually indicate myocardial damage, i.e., infarction. These T-wave changes will likely resolve over a period of weeks, returning from the Pardee morphology through that of Wellens back to normal—a reversal in slow motion of the initial sequence of changes (4). Other indicators of myocardial damage are duration and intensity of pain and a rise in cardiac enzymes (Table). All criteria indicate that this patient had an infarct and was at high risk for further ischemic events.

Had this patient arrived in the emergency department before his pain subsided, he would have received reperfusion therapy. Once his pain was gone, thrombolysis was no longer mandated, and a combination of aspirin, clopidogrel, heparin, a beta-blocker, and a nitrate was indicated (5, 6). In addition, he should have gone to the cardiac catheterization laboratory. Evidence in favor of an early invasive strategy in such high-risk patients is now compelling (7, 8).

Unfortunately, nights and weekends have been shown to be impediments to optimal management (9, 10), and the patient came in late on a Sunday night. Several hours after the first elec-

Figure 2. Cine coronary arteriographic frame recorded in a right anterior oblique and cranial projection. The culprit lesion (arrow) is a severe narrowing in the left anterior descending coronary artery distal to one small septal branch and one small diagonal branch.

trocardiogram was recorded, the patient had another bout of chest pain and was taken to the cardiac catheterization laboratory, where the culprit lesion was found in the left anterior descending coronary artery (Figure 2). This lesion and lesions in the larger 2 of 3 narrowed left circumflex obtuse marginal branches were stented with good results.

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