Communications

Pseudoinfarction Without Underlying Cardiopulmonary Pathology

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The electrocardiogram has long been used as an accurate and objective method for evaluating patients whose symptoms are suggestive of acute myocardial infarction. Although various pseudoinfarction patterns associated with underlying structural cardiopulmonary disease occasionally confuse the issue, the finding of Q waves in association with ST segment elevation without any obvious cause of pseudoinfarction is generally accepted as being a highly specific indicator of acute myocardial infarction.[1] The patient who is described presented with electrocardiographic changes suggestive of acute myocardial infarction despite the absence of acute or chronic cardiopulmonary pathology.

Case Report

A 41-year-old man developed chest pain and dizziness following physical exertion. These symptoms were associated with a cardiac dysrhythmia thought to be ventricular tachycardia by the responding paramedic team, and cardioversion was performed with resumption of sinus rhythm. An electrocardiogram (ECG) done in the emergency department revealed Q waves and ST segment elevation in the inferior leads (Figure 1), and the patient was admitted to the hospital with the presumptive diagnosis of acute myocardial infarction.

The patient had a history of intermittent chest discomfort during the preceding five years. Usually exacerbated by exercise or emotional upset, the pain occasionally occurred at rest and had a variable response to nitroglycerin.

Cardiac risk factors included smoking and male sex, but there was a negative family history of premature atherosclerosis, and the patient had no history of hypertension, diabetes mellitus, or hypercholesterolemia. The patient was currently under the care of a dermatologist for discoid lupus for which he was being treated with oral corticosteroids. Past medical history was otherwise unremarkable.

Physical examination disclosed a slightly obese white man lying comfortably in bed with an obvious facial skin rash. Blood pressure was 106/72 mmHg, pulse rate was regular at 82 beats/min, respirations were unlabored at 18/min and the temperature was 37° C orally. The chest was normally shaped, and the point of maximal impulse was not displaced. The neck veins were not distended, and no carotid bruits were evident. The heart sounds were normal, and no murmurs, clicks, or rubs were heard. The lungs were clear, the abdomen was benign on palpation, and examination of the extremities revealed normal peripheral pulses and the absence of pedal edema.

Admission laboratory evaluations including complete blood count, automated chemistry screen, urinalysis, arterial blood gases, and chest roentgenogram were all unremarkable.

Consultation the next day with the patient's previous physician disclosed that his ECG had not changed in over a year. No myocardial infarction had ever been documented, and an exercise stress

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test performed eight months earlier was normal through stage five of the Bruce protocol.

The patient became pain-free rapidly and had no further chest discomfort during the course of his hospitalization. Subsequent ECGs demonstrated persistent ST segment elevation in the inferior leads without any evolution. M-mode and two-dimensional echocardiography did not suggest ventricular aneurysm or other abnormality. Serial cardiac enzymes remained normal. On two occasions while in the intensive care unit, the patient manifested evidence of increased sensitivity to vagal tone in response to venipuncture by developing marked sinus bradycardia with an appropriate accelerated idioventricular escape rhythm. Both episodes were self-limited.

After ruling out an acute myocardial infarction, cardiac catheterization was performed on the 11th hospital day in an attempt to clarify the etiology of the patient's symptoms and abnormal ECG. This procedure demonstrated completely normal coronary arteries and excellent left ventricular function. Coronary artery spasm was ruled out by a negative ergonovine maleate stimulation test. During the catheterization the patient developed an episode of supraventricular tachycardia with reproduction of his typical chest pain; this was successfully treated with intravenous propranolol and underdrive cardiac pacing. He was discharged from the hospital shortly thereafter to be followed by his regular physician.

Discussion

Electrocardiographic pseudoinfarction patterns have been well described in the literature, but generally are associated with underlying cardiopulmonary disease.[1-12] Causes most commonly associated with pseudoinfarction include chronic obstructive pulmonary disease, pulmonary embolism, left or right ventricular hypertrophy, congestive or obstructive hypertrophic cardiomyopathy, ventricular conduction abnormalities (eg, left anterior fascicular block or complete left bundle branch block) and the Wolff-Parkinson-White syndrome. In addition, ST segment elevation simulating acute injury current occurs in acute pericarditis and early repolarization variants. Q waves are

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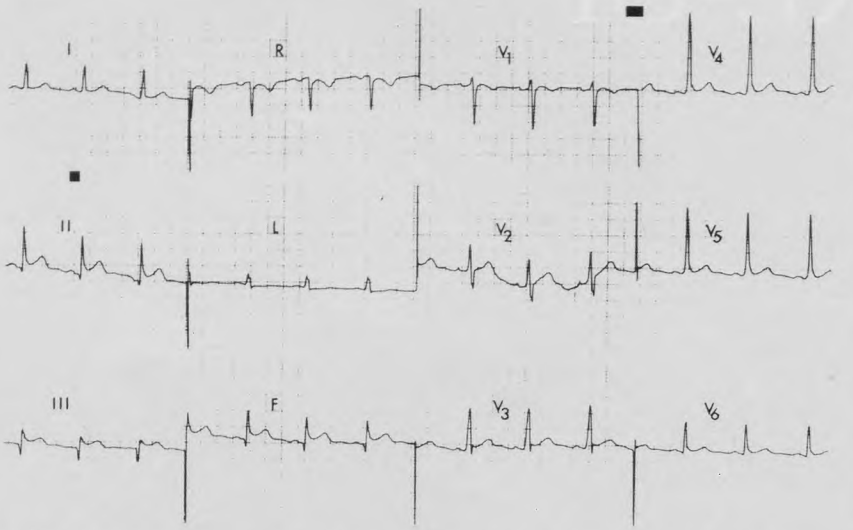

Figure 1. Initial electrocardiogram of the patient demonstrating Q waves and ST segment elevation in the inferior leads

not usually seen with these two entities, however.

Physiologic (ie, positional) Q waves due to normal variations in septal activation and cardiac position may occur in any of the 12 standard leads.[2] These Q waves can usually be differentiated from infarction Q waves by their smaller size and lack of associated ST segment abnormalities. Although one could argue that the Q waves in leads II and aV_F of Figure 1 do not meet the strict criteria for being pathologic (ie, width of Q wave $\geq$ 0.04 sec or depth of Q wave $\geq$ 25 percent of R wave), their association with ST segment elevation in these inferior leads would certainly suggest acute infarction in a patient presenting with the new onset of chest pain.

The case of pseudoinfarction reported here is unusual because it occurred in the absence of underlying structural cardiopulmonary disease. Cardiac enzymes never became elevated and serial ECGs failed to demonstrate evolutionary changes of acute infarction. The history did not suggest chronic obstructive pulmonary disease or pulmonary embolism. Physical examination and echocardiography excluded cardiomyopathy, pericarditis, and pericardial effusion. There was no evidence of left or right ventricular hypertrophy on the echocardiogram, chest roentgenogram, or ECG. Finally, the completely normal cardiac catheterization and negative ergonovine maleate stimulation test effectively ruled out previous infarction, ventricular aneurysm, and coronary spasm.

In retrospect, the electrocardiographic abnormalities of the pseudoinfarction pattern in this case may easily be explained by the coincidental and simultaneous occurrence of positional Q waves and ST segment elevation of early repolarization. Yet this conclusion was not at all obvious from the clinical presentation of chest pain, dizziness, cardiac dysrhythmia, and the admission ECG (Figure 1). This case illustrates that the finding of Q waves in association with ST segment elevation without an obvious cause of pseudoinfarction is not necessarily a specific indicator of acute myocardial infarction.

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Fetal Well-Being Assessed by Maternal Daily Fetal-Movement Counting

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Normal fetal movements are an indicator of fetal well-being, whereas reduced fetal activity may precede fetal death. In 1960 Bernstine[1] noted that decreased activity may reflect disturbance of placental function and indicate impending demise of the fetus. In 1972 Mathews[2] noted cases in which fetal death was preceded by a period of markedly reduced fetal activity. In 1973 Reinold[3] reported that absence of spontaneous fetal movement places the fetus at high risk. Sadovsky and Yaffe[4] observed that a definite decrease in fetal activity occurs before fetal death from chronic diseases such as toxemia. They considered the decrease in fetal movements to be an "alarm signal" of impending fetal death. Normal fetal activity has been associated with a good outcome and provides reassurance that delivery can be deferred to gain further fetal maturity.[5]

Women report 80 to 90 percent of the fetal movements that are picked up by electronic devices.[6-8] Fetal movements are first felt by women at about the 18th week of pregnancy, increasing to a maximum between 29 and 38 weeks and decreasing slightly just before term.[4] Ehrstrom[6] reported a 12-hour median of 86 fetal movements in the 24th week, 132 in the 32nd week, and 107 in the 40th week. There is a daily rhythm with maximum activity in the evening. Activity is relatively uniform in the morning and afternoon, with approximately seven fetal movements per hour.

An abnormal daily fetal movement count has been a better predictor of impending fetal death than placental lactogen levels or urinary estriol determinations.[5,8,9] A record of fetal activity by a compliant patient has been shown to be a reliable alternative to antepartum fetal heart-rate testing for initial screening of fetal well-being.[10]

Several protocols have been developed and used for maternal counting of fetal movements.[4-6,8-19] Of these protocols, patients have readily accepted and complied with the Cardiff "count-to-ten" system first developed by Pearson.[16] In this system, patients note the time when ten movements have been felt for that day and report for evaluation if there have been fewer than ten movements in 12 hours. This protocol was demonstrated to be efficacious in the study by Liston et al.[18]

Case studies[4,11] have demonstrated a decrease in fetal movements prior to fetal death from

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