Painful Palpitations: Chest Pain Associated With Postextrasystolic ST Depression

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Postextrasystolic T-wave changes have been described as early as 1915. The significance and mechanism of such changes remain controversial. Because postextrasystolic ST changes are rare, much of the ensuing discussion concentrates on the closely related and more commonly reported phenomenon of postextrasystolic T-wave inversion. This report documents the case of a 67-year-old man with a history of coronary artery disease who was admitted to the hospital with chest pain.

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KEY WORDS

Postextrasystolic ST depression • Palpitations • Coronary artery disease • Premature ventricular contraction

A 67-year-old man with history of coronary artery disease (CAD) was admitted to the hospital with chest pain. The patient has known hypertension and type 2 diabetes. His home medications included amlodipine, 10 mg/d, valsartan, 320 mg/d, aspirin, 81 mg/d, fenofibrate, 135 mg/d, and metformin, 500 mg twice daily. He described the chest pain as sharp to aching and retrosternal, and it followed a feeling of palpitations. A serial cardiac enzymes ruled out myocardial infarction; electrocardiogram (ECG) (Figure 1) revealed sinus rhythm with nonspecific inferior T-wave abnormalities, unchanged from a prior ECG performed 2 years earlier. During one episode of palpitations causing chest pain, his telemetry strips were analyzed and revealed ST depression in the sinus beat following a premature ventricular contraction (PVC), which raised concern for ischemia (Figure 2A). He was treated with carvedilol, 6.25 mg twice daily, and given sublingual nitroglycerin as needed for recurrent chest pains; he was also advised to restart his home blood pressure medications. Clopidogrel, 75 mg/d, and atorvastatin, 80 mg/d, were added to his regimen. The patient underwent coronary angiography the

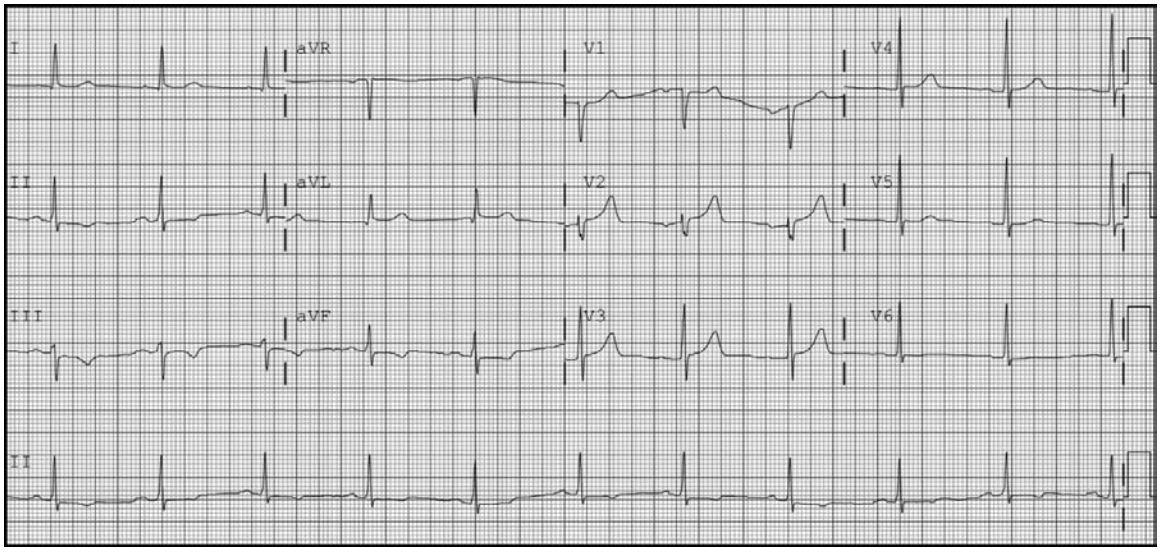


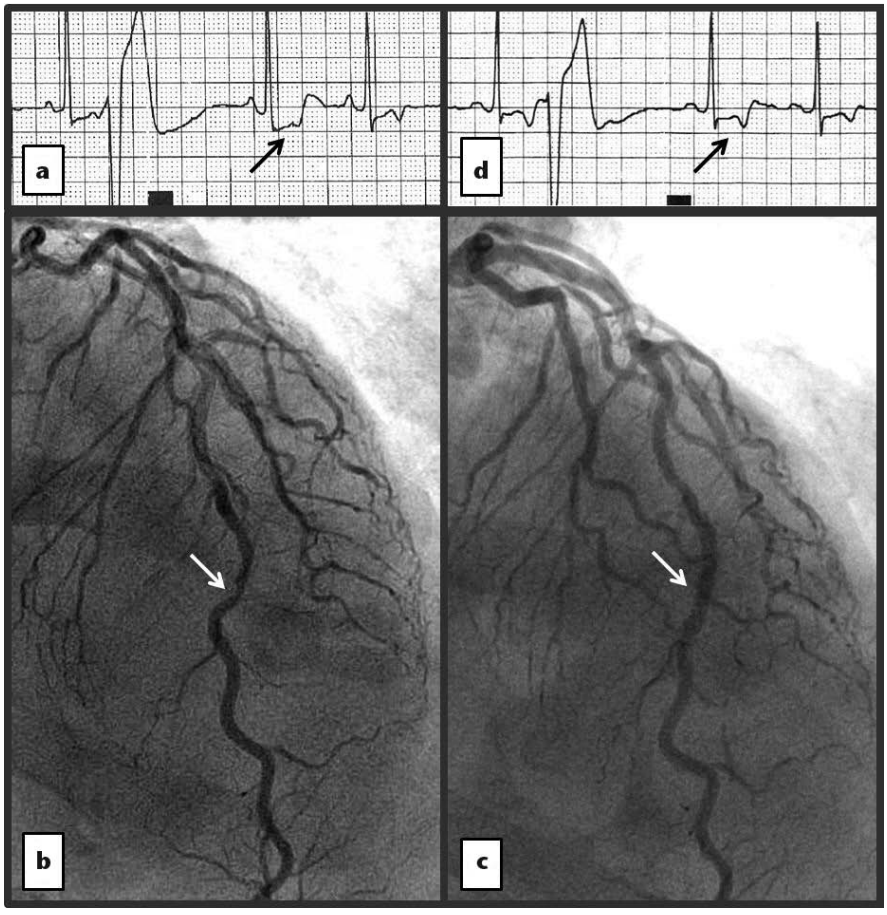
Figure 1. Admission electrocardiogram revealing normal sinus rhythm, with nonspecific inferior T-wave abnormalities, unchanged from a prior electrocardiogram performed 2 years earlier.

following morning, which revealed 70% mid left anterior descending artery lesion (Figure 2B), for which he received a drug-eluting stent (Figure 2C). Subsequent PVCs were not associated with symptoms, and there was significant improvement in the post-PVC ST changes as measured by telemetry (Figure 2D). The patient remained symptom free upon clinical follow-up 4 weeks later.

Discussion

Postextrasystolic ST changes are rare; the study by Murayama and colleagues[1] was the only report found that specifically addressed postextrasystolic ST-T changes. Therefore, much of the ensuing discussion concentrates on the closely related and more commonly reported phenomenon of postextrasystolic T-wave inversion. Postextrasystolic T-wave changes have been described as early as 1915 by White.[2] The significance and mechanism of such changes remain controversial. Levine and colleagues[3] studied the relationship of postextrasystolic T-wave changes to the presence of abnormal stress test results (Master two-step test). They reported that this phenomenon, when pronounced, is typically associated with evidence of myocardial impairment and coronary insufficiency. Among the potential mechanisms they entertained were the variations in

Figure 2. ST depression is seen in the first sinus beat following a premature ventricular contraction (**A**; *black arrow*). Angiogram revealed 70% mid left anterior descending artery stenosis (**B**; *white arrow*). Following deployment of a drug eluting stent (**C**; *white arrow*), a premature ventricular contraction is seen without the profound postextrasystolic ST depression previously observed (**D**; *black arrow*). Both telemetry strips were obtained via midclavicular line lead placement (MCL1).



cycle length causing repolarization abnormalities, versus the mechanical effect of over-distension of the ventricular wall in vulnerable hearts with selective local restriction of blood flow. Hegazy and Alkandary[4] found that persistence of symmetric T-wave inversion (with a cutoff value of 2.2 mV) for 30 minutes after repetitive PVCs was highly predictive of ischemic heart disease in hypertensive subjects. Murayama and colleagues[1] reported that postextrasystolic ST-T changes accentuated ischemic ST-T changes produced by exercise. Cotoi and associates[5] reported two cases of postextrasystolic T-wave inversions as a manifestation of silent myocardial ischemia.

Conversely, Fagin and Guidot[6] compared premature contractions in the presence or absence of postextrasystolic T-wave changes, and reported no relationship between the presence or degree of such change and the existence of clinical heart disease. They also found that the degree of prematurity of the extrasystole and the duration of the compensatory pause did not have any demonstrable effect on the presence or absence of postextrasystolic T-wave changes. Engel and associates[7] showed that patients with normal coronary arteries were just as likely to have T-wave changes following induced PVCs (68%) as patients with angiographically documented CAD (81%), and therefore concluded that such changes cannot be used to diagnose or localize CAD. Fechter[8] analyzed Holter monitors for postextrasystolic T-wave alterations and reported that, on the basis of clinical noninvasive diagnostic procedures, these alterations do not allow a distinction between persons with and without cardiovascular disease. Leachman and colleagues[9] demonstrated that provoked postextrasystolic T-wave alterations appeared neither sensitive nor specific in the identification of patients with cardiac disease. The frequency of postextrasystolic T-wave changes depended on the length of the compensatory pause.

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Experimental studies have also demonstrated the presence of postextrasystolic T-wave changes in normal hearts. Yoon and coworkers[10] induced premature ventricular beats at variable coupling intervals in normal dog hearts and postextrasystolic T-wave changes were observed following various postextrasystolic cycle lengths. They concluded that such changes occur in normal hearts and have no useful diagnostic values. A postextrasystolic compensatory pause may not be a requirement for either the occurrence of postextrasystolic T-wave changes or their relevance. Robitaille and associates[11] reported postextrasystolic T-wave changes associated with interpolated PVCs in 26 patients; only 18 patients (69%) had documented heart disease. Postextrasystolic T-wave changes may be present even in the absence of manifest T-wave abnormalities on the surface ECG. Batchvarov and Camm[12] demonstrated a change in the complexity of the QRS and T waves in the first sinus beat following a PVC in the absence of clearly discernible T-wave changes. A plausible explanation for postextrasystolic T-wave changes they referenced was that when the vector of the post-PVC T wave mirrors the direction of the ectopic QRS complex, the mechanism likely represents a form of short-term cardiac memory.[13]

Our patient developed a nearly 2-mm ST-depression in the first sinus beat following a PVC, the type of change that would render a positive stress test result; it was associated with chest discomfort. Postextrasystolic ST changes improved and the ensuing chest pain resolved following intensive medical treatment and revascularization. In the absence of a clear mechanism, whether such profound postextrasystolic ST-segment depression would qualify as a "poor-man's exercise test" (as has been credited to Levine by Chugh[14]) remains uncertain.

Conclusions

Postextrasystolic T-wave changes lack specificity for cardiac disease.

Postextrasystolic T-wave changes may be present even in the absence of manifest T-wave abnormalities on the surface ECG.

Their mechanism remains unclear. It is likely that the electrocardiographic and clinical extent of the changes, especially if they affect the ST segment and reproduce a patient's chest discomfort, as in our patient, may be more indicative of underlying coronary disease. More observations are needed to qualify this claim, however. Physicians should be vigilant of the potential value of painful palpitations in predicting underlying obstructive coronary disease. ■

The authors report no real or apparent conflicts of interest.

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MAIN POINTS

- The significance and mechanism of postextrasystolic T-wave changes remain controversial.
- When pronounced, postextrasystolic T-wave changes can be associated with evidence of myocardial impairment and coronary insufficiency.
- The persistence of symmetric T-wave inversion (with a cutoff value of 2.2 mV) for 30 minutes after repetitive premature ventricular contractions (PVCs) was highly predictive of ischemic heart disease in hypertensive subjects.
- Conversely, it has been shown that patients with normal coronary arteries were just as likely to have T-wave changes following induced PVCs as patients with angiographically documented coronary artery disease (CAD); therefore, such changes may not be helpful when attempting to diagnose or localize CAD.
- Physicians should be vigilant of the potential value of painful palpitations in predicting underlying obstructive coronary disease.