Pseudo-Myocardial Infarction Versus Pseudo-Pseudo-Myocardial Infarction

To the Editor:

Hung and colleagues present a very important example of a pseudo-infarction pattern presumably related to pancreatitis, one of several clinical situations in which thrombolytic therapy is either not indicated or contraindicated. I am intrigued by the pattern of the bizarre $T$ waves in the limb leads and wonder if part or all of this phenomenon is artifact. The $T$ waves are of unusually abrupt onset and offset. They are of greatest magnitude in leads I, II, and aVR. Lead III, however, appears to be isoelectic to this postulated artifact but not to the $T$ wave that is 1 to 2 mm in height and gently (ie, normally) inscribed. The artifact is of about half the magnitude and is approximately equal in leads aVL and aVF. The precordial leads reflect the artifact, which is increasing the apparent height of the actual $T$ waves, which appear inscribed on top of the artifactual elevation. The initial j-point or ST elevation in the precordial leads appears real, in that the onset is before that of the artifact in the limb leads but difficult to interpret in the presence of the artifact.

Because the artifact is of greatest magnitude in the 3 leads that have a right arm electrode attachment in common, could there have been motion of the electrode or its connections leading to this artifact? For instance, the timing of onset, duration, and contour of the artifact seem compatible with an arterial pulse wave altering skin contact with each heart beat. It would be important to know whether the bizarre $T$ waves in the limb leads and the terminal $T$ wave elevation in the precordial leads all resolved slowly or whether there was an abrupt change that would favor artifact. The follow up electrocardiograms are essential to resolving this issue.

Finally, whenever an unusual, bizarre, or unexplainable electrocardiographic finding is detected, it is most helpful to repeat the electrocardiogram personally, with a machine that is known to be trustworthy, to confirm that the findings are indeed real.

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Pseudo-Infarctions

To the Editor:

The pseudoinfarction ECG presented by Hung and associates was described as revealing “ST-segment elevation of 2 mm and peaked upright $T$ waves in leads $V_1$ through $V_6$, with reciprocal changes in lead II.” Actually, the ST-segment elevations spared the first 0.12 s in all the leads. Furthermore, the so-called ST-segment elevations occurred in all the leads except lead aVR, not lead II, as the authors alleged.

An alternative explanation for these ECG changes in their patient with acute pancreatitis could be acute pericarditis with ST-segment elevations in all the leads except aVR, which “faces” the endocardium. ECG changes similar to acute myocardial infarction have been reported in association with acute pancreatitis; they are probably caused by pancreatic enzymes or vasoactive peptides.

Although ST-segment displacement and $T$ wave changes, with or without abnormal Q waves, are the characteristic ECG findings in myocardial infarction, similar changes may be seen in patients without coronary artery disease (“pseudoinfarctions”).

The latter may occur in the following conditions.

1. In left ventricular hypertrophy, there is often a QS deflection or poor R wave progression in the right precordial leads that suggests anterior myocardial infarction. The secondary ST-segment elevation in these leads may be mistaken as a current of injury.

2. In pulmonary emphysema, the $R$ waves in the right precordial and sometimes midprecordial leads become quite small or are absent, suggesting anterior myocardial infarction. These QRS changes are explained by the vertical displacement of the heart secondary to a low-lying diaphragm and the intervention of hyperinflated lungs.

3. The pseudoinfarction pattern may also be seen in patients with pneumothorax. The voltage of the QRS complex may be reduced. QS deflection may appear in the right precordial leads.

4. In pulmonary embolism, the $Q$ waves in lead III (as part of the $S_Q_3$ pattern), and sometimes in lead aVF, that are accompanied by ST-segment and $T$-wave changes are often interpreted as inferior myocardial infarction. In addition to $T$-wave inversion, with or without an rSr’ pattern in the right precordial leads due to acute right heart strain, QS complexes with ST-segment elevation may occasionally develop in these leads and mimic acute anterior myocardial infarction.

5. In hypertrophic cardiomyopathy, abnormal Q waves are often seen, especially in the left precordial leads and lead I. These Q waves have been attributed to ventricular septal hypertrophy.

6. Myocardial fibrosis is often responsible for the pseudo-infarction pattern in patients with dilated cardiomyopathy, progressive muscular dystrophy, Friedreich’s ataxia, scleroderma, amyloidosis, and primary and metastatic tumors of the heart.

7. QS deflections are often seen in the right precordial leads in patients with complete left bundle branch block in the absence of myocardial infarction.

8. Left anterior hemiblock is occasionally associated with small Q waves in the precordial leads that mimic anterior myocardial infarction.

9. The delta waves in Wolff-Parkinson-White syndrome are frequently interpreted as abnormal $Q$ waves of myocardial infarction.

10. Pheochromocytoma may be associated with striking ECG changes masquerading as ischemic heart disease.

11. Other conditions that may be associated with ECG changes simulating myocardial infarction include intracranial hemorrhage, hypokalemia and, as mentioned above, acute pericarditis.

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Response

We are grateful to Drs Kessler and Cheng for their insightful comments regarding our article. The possibility of artifact in this ECG recording came to our minds, too. However, the follow-up
ECGs, which were performed with another ECG machine, revealed similar findings. The likelihood of interference by other extrinsic factors, such as an arterial pulse (suggested by Dr Kessler), in producing the observed bizarre T waves cannot be completely excluded. The bizarre T waves disappeared several hours later, and the subsequent ECGs, which were not shown, still revealed ST segment elevation with positive T waves in V1 to V6, and inverted T waves in V4 to V6. We think the key finding was the ST segment elevation in the right precordial leads, especially V3, and we consider it a good example of pseudo-myocardial infarction.

The possibility of acute pericarditis, as suggested by Dr Cheng, should be considered as well. However, the ST segments in several leads, including leads I, II, and aVL, show depression instead of elevation, a finding uncommon for pericarditis. The list of causes of pseudoinfarction raised by Dr Cheng is important. Moreover, an intriguing ECG finding in the contemporary era of interventional electrophysiology is the ST-T change after radiofrequency catheter ablation of the accessory atrioventricular pathway; such a change includes an elevated ST segment and tented T waves in the right precordial leads after elimination of the left accessory pathways. The repolarization changes after successful ablation of accessory pathway is presumably due to “cardiac memory.”

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