EVALUATION OF PSEUDO-INFARCTION ECG PATTERN

To the Editor: We read with interest the recent case report by Gaitonde et al regarding pseudo-infarction electrocardiogram (ECG) pattern secondary to cardiac infarction by metastatic lung cancer1 and wish to contribute some comments.

First, the presenting ECG is described as showing a left anterior hemiblock as well as evidence of an inferior myocardial infarction (MI). Although an intraventricular conduction delay is certainly present, left anterior hemiblock is not. The diagnosis of left anterior hemiblock requires the presence QR complexes in leads I and aVL; RS complexes in the inferior leads II, III, and aVF; and left axis deviation to –45° or more.2 The ECG in Figure 1 has none of these features. In fact, the axis is no more leftward than –30°, surprisingly rightward for a post–left pneumonectomy patient with an expected leftward shift of the heart within the thorax. Even prior to imaging, the markedly delayed appearance of the R wave in the precordial leads suggests a rotational shift in a patient without anterior MI.

Second, the initial diagnosis was of a “high lateral” MI was based in part on striking ST-segment elevation in leads I and aVL, with reciprocal ST-segment depressions in the inferior leads. However, all of the precordial leads also demonstrate ST-segment depressions. This suggests a high posterolateral distribution of injury, usually in the area subtended by the proximal left circumflex artery. The catheterization data provide no lesion consistent with the ECG changes; hence, there was no rationale for placement of a left anterior descending (LAD) stent beyond the incidental finding of atherosclerotic disease. Although the postcatheterization ECG is interesting, a more useful image would have been the patient’s baseline (ie, early postpneumonectomy) ECG for comparison with the tracing at presentation.

The patient remained symptomatic and the ECG unchanged despite the LAD intervention, further supporting that the coronary lesion was not related to the ECG changes. The case is a useful reminder that coronary revascularization, particularly with stent placement in nonculprit vessels in patients at risk for metastatic disease to the brain, is best deferred until such metastasis is ruled out, if carried out at all. Procedure-related anticoagulation and antiplatelet therapy with heparin, glycoprotein IIb/IIIa inhibitors, and clopidogrel may dramatically increase the risk of intracerebral hemorrhage from a metastatic lesion.

In a case such as this, a dedicated cardiac magnetic resonance image with coronary and myocardial imaging, if available, may have obviated the need for cardiac catheterization and better defined the location and extent of pericardial and myocardial involvement by the infiltrating tumor mass.

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References