We present the ECGs of a 77 year old male patient. He presented three hours after the onset of central chest pain and was diagnosed with acute myocardial infarction. There was no relevant past medical history or drug therapy. The initial ECG (panel A) demonstrated marked QRS prolongation and T wave inversion. Post-thrombolysis ECG (panel B) and echocardiography confirmed the presence of inferior wall infarction. Peak troponin T was 9.3 ng/ml (normal <0.03 ng/ml). Serum electrolytes were normal. Cardiac catheterisation revealed significant single vessel right coronary artery stenosis (panels C and D: pre- and post-percutaneous coronary intervention).

Severe ST segment elevation can occur with a markedly prolonged R wave that produces a “tombstone” configuration. Two distinct mechanisms have been shown to account for ST segment elevation. These involve (a) markedly delayed transmural conduction and (b) loss of the epicardial action potential dome. During ischaemia, extracellular metabolites and potassium accumulate depolarising the myocardium and inactivating sodium channels leading to slowing of conduction. Transmural conduction delay results in a markedly prolonged R wave with tombstone ST elevation and T wave inversion. The transient outward potassium current \( I_{to} \) is primarily responsible for the early “notch” or phase-1 repolarisation of the ventricular action potential. Furthermore, \( I_{to} \) can significantly modulate the plateau and early phase 3 repolarisation phases. Animal studies have demonstrated a heterogeneous distribution of candidate \( I_{to} \) channel subtypes from ventricular epicardium to endocardium that may explain this differential response to ischaemia.

We propose the ECG changes observed with this acute infarction were due to ischaemia-induced reduction in \( I_{to} \).

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