Sir,

A 70-year-old female, recently diagnosed with metastatic rectal carcinoma requiring a de-functioning colostomy, presented to her local emergency department with chest pain in keeping with myocardial ischaemia. An initial electrocardiogram (ECG) demonstrated lateral ST-segment elevation and she was urgently transferred to the regional percutaneous coronary intervention (PCI) centre. On arrival her pain had settled. Emergency coronary angiography revealed single vessel disease in the form of a heavily calcified non-flow limiting stenosis in the mid-portion of the right coronary artery. Echocardiography demonstrated a lateral wall motion abnormality with no evidence of malignant invasion of the pericardium or a pericardial effusion. Troponin I was raised at 1.9 μg/l confirming a myocardial infarction and routine biochemistry revealed a plasma potassium concentration of 2.9 mmol/l, for which she was commenced on an intravenous potassium chloride infusion. The following day she developed recurrent episodes of ventricular tachycardia (VT) and repeat ECG demonstrated gross global ST-segment elevation (Figure 1). She was immediately taken back to the catheterization laboratory, but angiographic appearances were unchanged from the previous day. Biochemistry now revealed profound hypokalaemia (2.1 mmol/l) despite replacement and hypocalcaemia (corrected 1.75 mmol/l). On further questioning she had noticed greatly increased output from her stoma over the previous week. Her rhythm stabilized and the ECG changes resolved over 24 h following aggressive intra-venous replacement of electrolytes up to normal levels.

This lady initially presented with ischaemic chest pain, lateral ECG changes and a correlating wall motion abnormality on echocardiography. Coronary artery disease was confirmed at angiography, but there was no evidence of an acute flow limiting lesion. Whether this was due to coronary vasospasm secondary to electrolyte disturbance or a self-resolving plaque rupture is unclear. Her deterioration the following day with the development of

Figure 4. Echocardiogram (apical four chamber view) 4 weeks later demonstrating normal left ventricular ejection fraction. A: end diastole and B: end systole.

References

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global ST-segment elevation and VT was without chest pain. The ECG changes and rhythm disturbance persisted throughout repeat angiography which did not demonstrate any changes from the day before; in particular there was no evidence of dissection or coronary spasm, and echocardiography did not reveal any pericardial pathology. These findings suggest that electrolyte disturbance secondary to profuse diarrhoea was the cause for the global ST-segment elevation and VT, a hypothesis which is further strengthened by their resolution following electrolyte correction.

Figure 1. ECG 24 h after admission with serum potassium 2.1 mmol/l.

global ST-segment elevation mimicking acute myocardial infarction is well recognized in the setting of hyperkalaemia. However, there is a paucity of reported cases with hypokalaemia and it has never been described in the aftermath of a myocardial infarction. As in our case, hypocalcaemia coexisted in two of the three previously reported cases and it has also previously been postulated that the changes were a result of ‘relative hyperkalaemia’ during potassium replacement. The exact role and interplay of these factors as well as the possible contribution of an acutely injured myocardium in the pathogenesis of this rare manifestation of hypokalaemia can only currently be speculated. This case adds to the literature on the presentation and possible mechanisms of a rare phenomenon and reinforces the importance of urgently ascertaining plasma electrolytes in patients developing VT even if an ischaemic cause is strongly suspected.

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