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Embolic complication of Tako-Tsubo cardiomyopathy.

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Embolic complication of Tako-Tsubo cardiomyopathy

Sir,

We present a case of an 81-year-old female presenting with one week worsening dyspnea on exertion

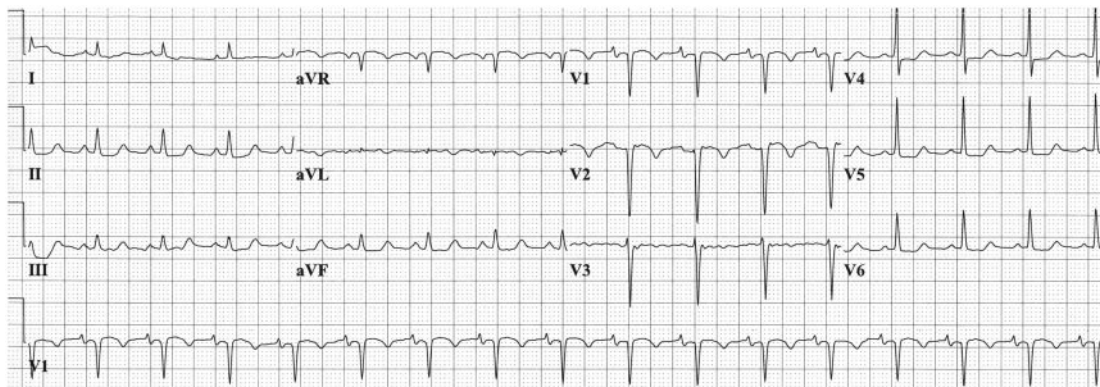


Figure 1. Electrocardiogram showing ST depression in the inferolateral leads and ST elevation in aVR and V1-2.

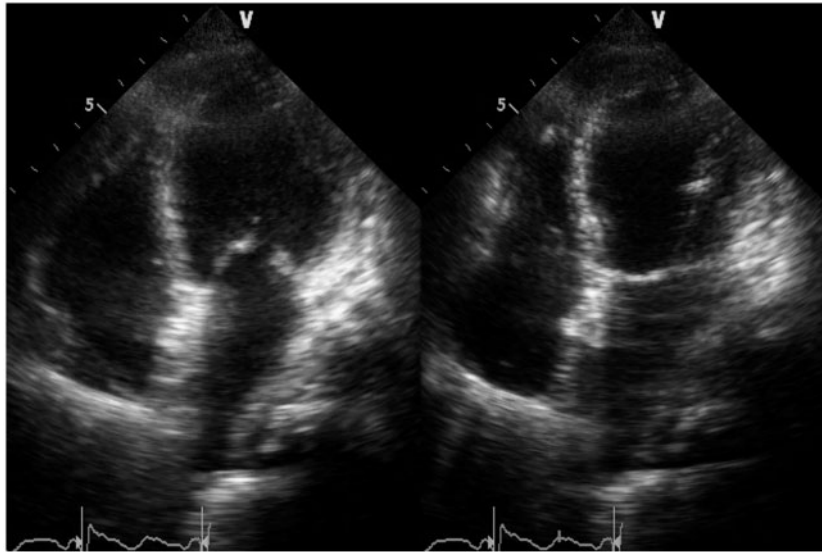


Figure 2. Echocardiogram (apical four chamber view) demonstrating a dilated left ventricle with severely reduced ejection fraction but preserved basal contractile function. A: end diastole and B: end systole.



Figure 3. Catheterization demonstrating occlusion of the distal right popliteal and anterior tibial arteries.

and a cold, discolored, numb foot for 2 days. The patient denied chest pain but reported a history consistent with melanotic stools. Her hemoglobin was

found to be 3.5 in the emergency department. Electrocardiogram showed 0.5–1.0 mm ST depression in leads II, III, aVF, V4-6 and ST elevation in aVR and V1-2 (Figure 1). Echocardiogram demonstrated a dilated left ventricle with a severely reduced ejection fraction but preserved basal contractile function (Figure 2A: end diastole and 2B: end systole). Catheterization demonstrated no epicardial coronary artery disease, but occlusion of the distal right popliteal and anterior tibial arteries (Figure 3). The patient was treated with heparin, aspirin, beta-blocker and angiotensin converting enzyme inhibitor. The patient eventually required amputation of her right lower extremity. Gastroenterological workup revealed a colon adenocarcinoma which was successfully resected. Follow-up echocardiogram 4 weeks later demonstrated normal left ventricular ejection fraction (Figures 4A: end diastole and 4B: end systole). The final diagnosis was tako-tsubo cardiomyopathy, precipitated by severe anemia, leading to distal thrombus embolization. Hagi *et al.*¹ reported in this journal the incidence of left ventricular thrombus in the setting of tako-tsubo cardiomyopathy to be 8%. However, they reported no episodes of peripheral embolization due to the left ventricular thrombus. This case illustrates the potential complications associated with thrombus formation found to occur in a minority of tako-tsubo cardiomyopathy cases.

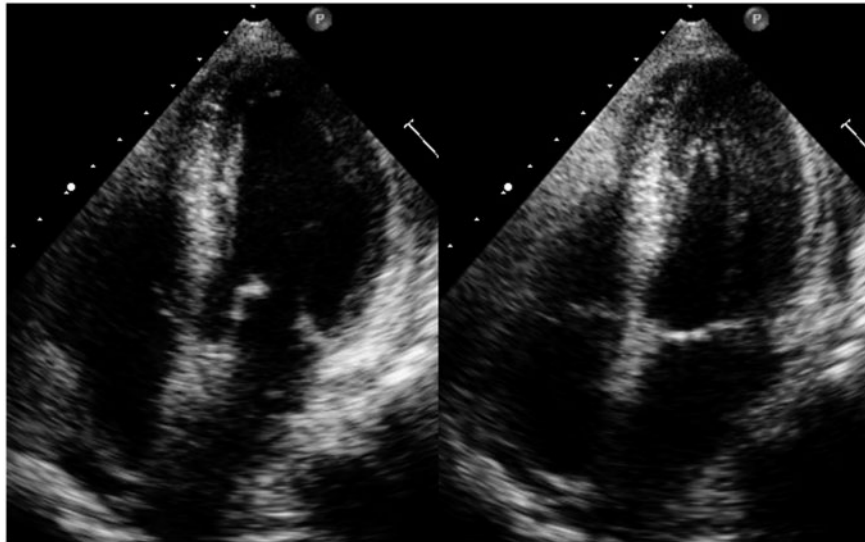


Figure 4. Echocardiogram (apical four chamber view) 4 weeks later demonstrating normal left ventricular ejection fraction. A: end diastole and B: end systole.

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Profound hypokalaemia mimicking acute myocardial infarction

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