Journal of Cardiology Cases (2011) 4, e67—e70

available at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/jccase

Case Report

Renal thromboembolism in tako-tsubo cardiomyopathy in spite of anticoagulation

Bonello Spiteri Danica (MD)*, Sammut Mark Adrian (MD), Xuereb Robert George (MD)

Department of Cardiology, Mater Dei Hospital, Msida MSD2090, Malta

Received 23 December 2010; received in revised form 3 May 2011; accepted 24 May 2011

KEYWORDS
Tako-tsubo cardiomyopathy;
Kidney;
Thromboembolism;
Anticoagulation

Summary An elderly lady was admitted with chest pain and an electrocardiogram depicting ST segment elevation, indicative of a myocardial infarction. She was treated with intra-venous tissue plasminogen activator. On coronary angiography there was a dilated and akinetic left ventricular apex but no significant coronary artery disease. She was diagnosed with tako-tsubo cardiomyopathy. An echocardiogram performed two days later demonstrated a thrombus in the left ventricular apex. Despite immediate anticoagulation with intravenous unfractionated heparin, she sustained a renal thromboembolic phenomenon.

Introduction

Tako-tsubo cardiomyopathy (TTC) is an uncommon condition first described in Japan. Presentation mimics a myocardial infarction, with electrocardiogram (ECG) changes and an associated troponin and creatinine kinase (CK) rise. There is dilatation of the apex of the left ventricle, which may predispose to thrombus formation, with the risk of embolisation, resulting in end organ damage [1,2]. Anticoagulation plays an important role in thrombus lysis [3], however guidelines on anticoagulation are lacking.

Case report

A 67-year-old lady presented with a 5-h history of left upper limb numbness and central compressive chest pain associated with nausea and sweating. She had a blood pressure of 125/80 mmHg, heart rate of 75 beats/min, and a central temperature of 37 °C. She had passed through a very emotionally traumatic period following the death of her son who had Down's syndrome. He died secondary to metastatic testicular carcinoma one year previously. Her initial ECG showed a normal sinus rhythm and no ischaemic changes. A subsequent ECG, performed 2 h later, revealed ST segment elevation in leads I, aVL, V5, V6. A diagnosis of ST segment elevation myocardial infarction (STEMI) was made. She was thrombolysed with intra-venous tissue plasminogen activator and treated with intravenous unfractionated heparin for 48 h. CK level rose to 510 U/L, with CK-MB ratio of 7%. A complete blood count was normal, except for a mild thrombocytosis (platelet 450 $\times$ 10^9/L). Coronary

* Corresponding author. Tel.: +356 2545 0000; fax: +356 2545 4154.
E-mail address: danica_spiteri@hotmail.com (B.S. Danica).

1878-5409/$ — see front matter.
doi:10.1016/j.jccase.2011.05.006

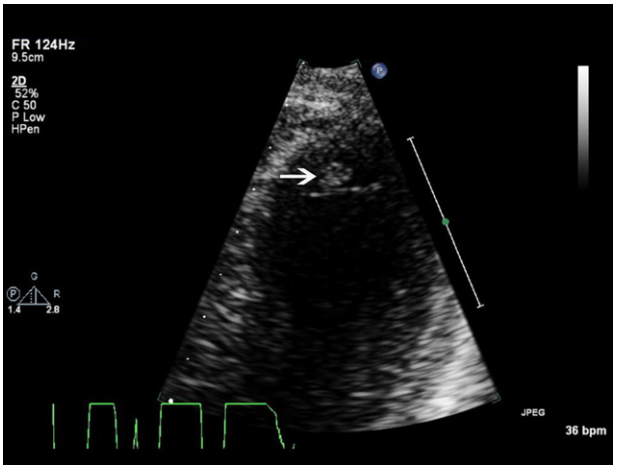

Figure 1 Echocardiogram with thrombus in the apex of the left ventricle.

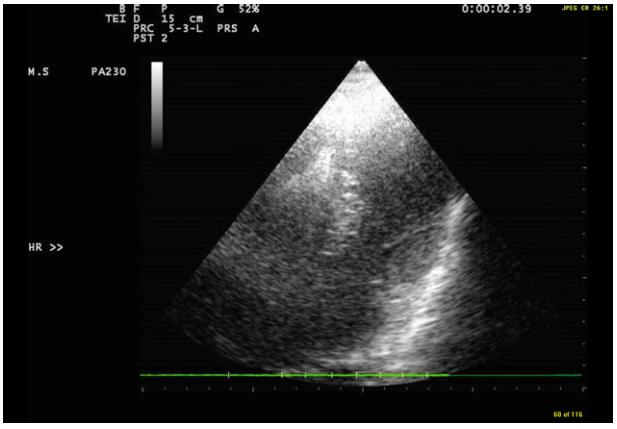

Figure 2 Echocardiogram depicting a dilated left ventricle in systole.

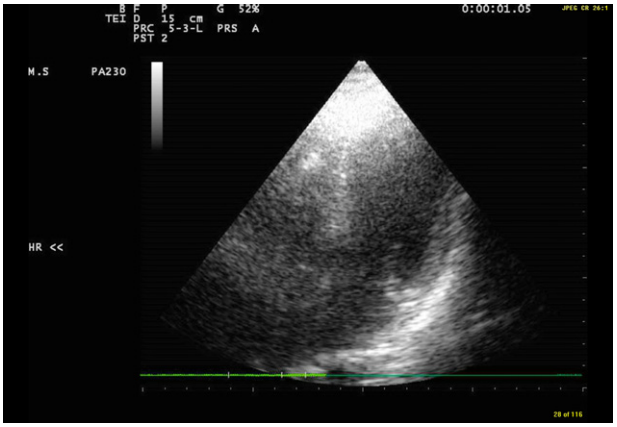

Figure 3 Echocardiogram depicting a dilated left ventricle in diastole.

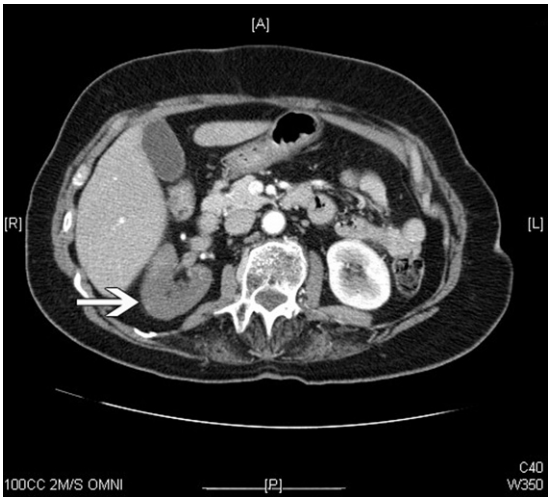

Figure 4 Computed tomography of the abdomen showing no contrast uptake by the lower pole of the right kidney.

angiography was performed 2 days after admission. The left ventriculogram showed a dilated and akinetic apex with bulging out of the left ventricular apex and moderate mitral regurgitation. There were non-significant lesions involving the mid-left anterior descending coronary artery and the diagonal arteries. Heparin was stopped prior to the coronary angiogram, and not re-started following the exclusion of STEMI. An echocardiogram performed on the same day showed a dilated akinetic apex with normal contractility of the basal segment. Significant mitral regurgitation was noted with a flaccid cusp of the posterior segment of the mitral valve. The echocardiogram (Figs. 1—3) was repeated 3 days later and an apical mural thrombus was detected. Anticoagulation was initiated immediately with intravenous unfractionated heparin and also with oral warfarin.

The following day the patient, in spite of anticoagulation (activated partial thromboplastin time ratio of 2.1, international normalized ratio 1.1), sustained severe abdominal pain in the right iliac fossa and right loin associated with feeling faint. A computed tomography scan of the abdomen revealed an embolus in the right renal artery, with an avascular area of the right lower one-third of the kidney (Fig. 4). A right sided pyelogram was carried out via a right femoral artery puncture. Dual right renal arterial supply was noted. The upper and middle poles of the nephrogram were seen, whilst the lower pole (main renal artery) was not vascularized (Fig. 5). Intra-arterial lysis of the avascular lower pole of the right kidney was unsuccessfully attempted, since the thrombus was not visualized. It is most probable that the thrombus had already fragmented and moved into the distal renal arterioles. Serum creatinine level rose to a maximum value of 110 Umol/L. On repeat echocardiography 24 h after the renal infarction the apical mural thrombus was absent, suggesting that the thrombus had embolized out of the left ventricle.

A follow up echocardiogram performed 24 days after the initial event, showed marked improvement in the apical akinesia. No thrombus was visualized in the left ventricle. The flaccid posterior leaflet had resolved, however there was transient systolic anterior leaflet motion of the mitral valve with septal contact and left ventricular outlet obstruction, resulting in mild mitral regurgitation. This resolved completely on echocardiogram performed 3 months after the event. She was advised to continue warfarin for up to one year. Her serum creatinine level returned to normal range at 84 Umol/L. A 2-methoxy isobutyl isonitrile scan performed