CASE REPORT

Tako Tsubo Cardiomyopathy in a Patient with Antiphosholipid Syndrome Secondary to Systemic Lupus Erythemathosus (SLE)

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SUMMARY

Tako Tsubo cardiomyopathy is rare, stress related and indistinguishable from acute myocardial infarction clinically. Proper diagnosis is essential to avoid unnecessary thrombolysis and life long management of coronary artery disease.

KEY WORDS:
Tako Tsubo cardiomyopathy, SLE, Myocardial infarct, Cerebral infarct

INTRODUCTION

The purpose of this case report is to highlight a rare form of cardiomyopathy which has different therapeutic strategy and prognostic implication compared to acute myocardial infarction. Correct diagnosis is essential so that patient may not receive unnecessary thrombolysis and long term management of coronary artery disease.

CASE REPORT

A 62 year old post menopausal lady was admitted with generalized seizure. She had a similar episode two and a half months earlier. CT brain at that time showed left parieto occipital infarct. She was diagnosed with SLE two years earlier based on the history of fleeting joint pains and swelling,raised ESR of>120 mm/hr, ANF 2560, anti double stranded DNA 320(<10), Hb 8.2g/dl(normochromic, normocytic), Rheumatoid factor was negative. On examination, she was initially drowsy(post ictal) but after a few hours was alert, orientated with minimal right sided weakness. CT brain findings were similar to her initial epileptic episode. ECG done about ten hours after admission showed Q waves and ST elevation in V1-3. Echocardiogram (Figure 1) showed apical, mid and distal septal akinesia. Overall EF was 35-40%. ECG and Echocardiogram done two and a half years earlier were normal. Chest x-ray showed pulmonary oedema. Troponin T was 1.0ng/ml(0.1-2) and creatinine kinase was 866U/L. Clinical diagnosis of established anteroseptal myocardial infarction with heart failure was made. She was not thrombolysed as the infarct could have been more than 12 hours old. The following day's ECG showed typical T inversion in the affected leads. She was treated with aspirin, clopidogrel, low molecular weight heparin, frusemide, ACE inhibitor, phenytoin and prednisolone. Coronary angiography four days later surprisingly showed near normal coronary arteries. Echocardiogram two weeks later (Figure 2) showed normalization of the regional wall anomaly. Patient made a full recovery and remained on aspirin, ACE inhibitor, phenytoin and prednisolone. Lupus anticoagulant was positive and VDRL negative. This patient has Tako Tsubo cardiomyopathy and antiphopholipid syndrome secondary to SLE. As the cerebral arteries have not been studied it cannot be certain whether thromboses or a Tako tsubo like phenomenon accounted for the cerebral infarct. She was not warfarinised although she has secondary antiphospholipid syndrome as there is problem with compliance and uncertainty of cerebral thromboses.

DISCUSSION

In 1990, Hikaru Sato[1] and colleagues described a syndrome mimicking clinical acute myocardial infarction (AMI) in the presence of normal coronary arteries. Left ventriculogram revealed a left ventricle (LV) which had acquired the shape (round bottom due to apical akinesia, narrow neck due to basal contraction) that resembled a type of bottle used in Japan for trapping octopus. Tako means octopus. Tsubo means bottle. Colleagues from Mayo clinic[2] suggested the following diagnostic criteria: new ECG changes- either ST elevation or depression, absence of obstructive coronary disease, transient akinesia/dykinesia of LV on echocardiogram or ventriculogram, absence of cardiomyopathy, head trauma, intracranial bleed or phaeochromocytoma.

As recent as 2000, it has only been described in Japan. Incidence is rare, probably 1-2% of patients who present with AMI[3]. Wall motion anomaly usually involves but is not restricted to the apex hence the alternative term – apical ballooning syndrome. Right ventricle can also be involved up to a third. It predominantly but not exclusively affects post menopausal women with a 7:1 female:male ratio. Emotional stress appears to be a triggering or associated factor in the majority of patients[4]. Tako Tsubo has occurred after earthquakes, penalty shoot outs, fierce arguments, court appearances and armed robberies. Broken heart syndrome has also been used to describe this cardiomyopathy.

Patients generally have a good prognosis as LV dysfunction is transient and resolves in days to a few weeks. Recurrence rate

This article was accepted: 25 February 2008
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is less than 10%[5]. However, patients may develop heart failure and rarely cardiogenic shock, left ventricular outflow tract (LVOT) obstruction. Mortality is unlikely to be more than 1-2%. It's difficult to be certain of the exact cause but there are some postulated mechanisms which include excess cathecholamines inducing myocardial stunning, multivessel epicardial spasm, coronary microvascular spasm, transient obstruction of the LVOT and myocarditis.

Treatment is basically supportive. As the syndrome is initially (before angiography is done) indistinguishable from acute coronary syndrome (ACS), patients will usually receive antiplatelet therapy. Pulmonary oedema should be treated accordingly. Cardiogenic shock due to pump failure can be treated with inotropes and intraaortic balloon pump. Inotropes are contraindicated if there's LVOT obstruction.

Clinical features are difficult to distinguish from AMI or ACS. If Tako Tsubo cardiomyopathy is mistaken for AMI, patients may receive unnecessary thrombolysis and life long management of coronary disease. This patient also has SLE. It is unknown whether this is coincidental or there's underlying association with Tako Tsubo. It's plausible that vasculitis(secondary to SLE) may induce severe coronary spasm leading to a Tako Tsubo like syndrome. Seizures could have perhaps also played a role as the stress factor triggering the syndrome.

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