Brief Communications

Takotsubo cardiomyopathy mimicking postoperative myocardial infarction in a young healthy patient

INTRODUCTION

Takotsubo cardiomyopathy (TCM) has been described in various ways in literatures, such as transient apical ballooning syndrome, apical ballooning cardiomyopathy, stress-induced cardiomyopathy, broken heart syndrome and simply stress cardiomyopathy.[1] It is a type of non-coronary ischaemic cardiomyopathy in which there is a sudden reversible weakening of the myocardium. This weakening can be triggered by emotional stress, such as the death of a loved one, a break-up, constant anxiety or physical pain.[2] This condition is a well-recognised cause of acute heart failure, lethal ventricular arrhythmias and even ventricular rupture.[3] During the course of evaluation, a high cardiac biomarker with or without ST changes in echocardiography mimicking a myocardial event, rise in cardiac biomarkers including troponin and CK-MB. The hallmark of bulging out of the apex of the heart with preserved function of the base that earned the syndrome its name is 'tako-tsubo' or octopus trap of Japan. The condition is diagnosed with ventriculography or echocardiography with no evidence of coronary stenosis in angiography.[4]

CASE REPORT

A 38 year male, with no other co-morbidity scheduled for interlocking nail replacement and bone grafting for of non-union fracture of mid shaft tibia.

Except for mild elevation of blood pressure (160/85 mmHg) which was labelled as white coat hypertension, there was no other significant finding in pre-anaesthetic check up. The anaesthesia technique involved combined spinal epidural (CSE) anaesthesia with 3 ml of 0.5% heavy bupivacaine intrathecally, intraoperative course was uneventful and duration of surgery lasted 2 hours with systolic blood pressure ranging from 130 to 150 mmHg.

Since the patient had epidural catheter as a hospital protocol, we kept this patient in HDU for post-operative analgesia. Epidural analgesia was delayed till the recovery of motor power, but as soon as motor blockade wore off, patient complained of severe pain at surgical site which settled after epidural bolus but there was significant rise in blood pressure (200/110 mmHg) during the painful crisis with straining pattern of ST segment in infero-lateral leads [Figure 1]. The acute rise in blood pressure was treated with intravenous boluses of hydralazine 10 mg. After initial two 10 mg boluses, blood pressure fell down rapidly to 80/60 mmHg and the patient had transient bradycardia up to 50 beats/min, hypotensive episode was treated with fluids bolus and intravenous ephedrine 6 mg. The ST segment progressively evolved into a shagging pattern during the hypotensive episodes [Figure 2].

Associated with the wide swing in the haemodynamics, the patient started to have chest pain, headache and sweating. Serial ECG did not show any acute ischemic changes except some non-specific ST changes in inferolateral leads in the form of ST segment depression with shallow T wave, this ECG pattern lasted for couple of days [Figure 3].

The initial cardiac biomarkers (CK MB and Troponin) were normal but second (after 6 hours of 1st sample) and third (after 12 hours of 1st sample) readings of cardiac markers were surprisingly elevated to very high levels.

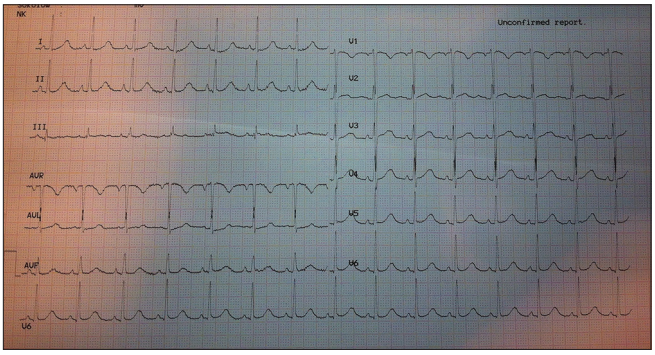

Figure 1: ECG image at the time of hypertension

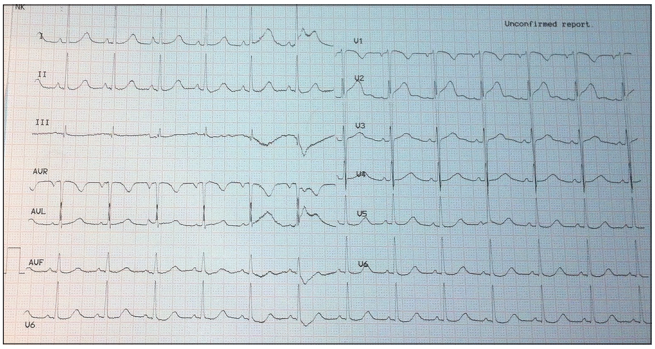

Figure 2: ECG image during a hypotensive episode

The serial cardiac biomarkers Troponin and CK MB respectively are provided below:
Ist (at the time of hypotension): 0.020 μg/l and 0.9 ng/ml
2nd (6 hours of first sample): 8.201 μg/l and 78 ng/ml
3rd (12th hours of first sample): 31.875 μg/l and 240 ng/ml.

Most probable initial diagnosis of acute coronary syndrome (ACS) was kept in mind and case was treated with all supportive management including anticoagulation, analgesics and haemodynamics monitoring. Echocardiography picture revealed akinesia with ballooning of left ventricular apex and normal base. He was kept in HDU for 48 hours and serial Echocardiographies were done within 48 hours and the apical ballooning started getting better; other possibilities of myocarditis and pheocromocytoma were ruled out on the basis of negative history, absence of generalized ST changes with multiple regional wall motion abnormality in myocarditis and normal levels of 24 hours urinary catecholamines and Vanillylmandelic acid in blood.

Our case had a remarkable improvement within third day and angiographic study revealed completely clean coronaries without any signs of obstruction [Figure 4] favouring the diagnosis of TCM. The patient was followed up for a week without any further events and discharged home without any sequelae.

DISCUSSION

The diagnosis of TCM is based on strong clinical suspicion with laboratory evidence of raised cardiac biomarkers, normal coronaries on angiography, akinesia and ballooning of left ventricular apex.[4-6] TCM is often encountered in post-menopausal women after physical or emotional stress. Excessive catecholamine stimulation, coronary artery vasospasm, microcirculatory dysfunction, and transient obstruction of the left ventricular outflow tract have been proposed as possible causes of this disorder.[2,4-7]

TCM has been reported in perioperative periods[7-10] It is commonly encountered in post-menopausal women with hypertension and is precipitated by acute physical or emotional stress.[2,4-9] Identification of susceptible patients, reduction in perioperative stress and postoperative pain is an integral part of anaesthesia management which prevents the occurrence of such major cardiovascular complication.

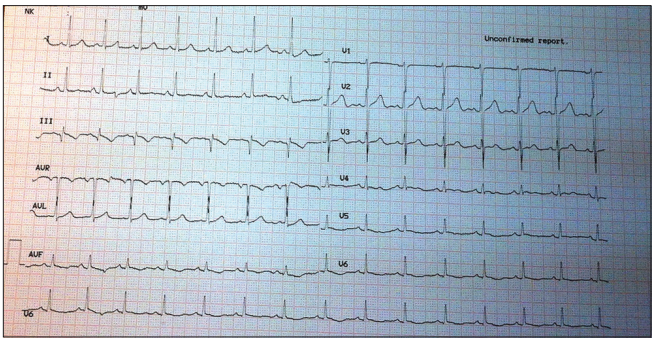

Figure 3: ECG pattern during chest pain

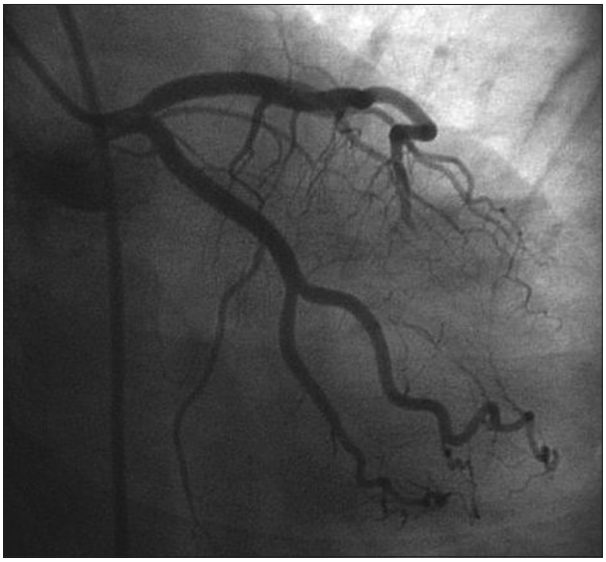

Figure 4: Coronary angiography image

Our case is unusual in that the patient was a young male without any underlying pathology and had an uneventful surgery 6 months ago. Perioperative TCM can be a preventable condition as the acute physical or emotional stress are iatrogenic related to medical or surgical procedures which can be properly managed with adequate counselling or pharmacotherapy. Use of intraoperative transesophageal echocardiography has been helpful in diagnosis of intraoperative stress cardiomyopathy.[10] Although TCM is underreported (2-5%) of all acute coronary syndromes, this transient pathological entity may be much higher than reported. Any suspected cardiac event in susceptible patients should be followed up with a coronary angiography not only for patient safety but for accurate understanding of this disease entity which may or may not be rare.[4,6]

CONCLUSION

Appropriate perioperative anxiolysis, better postoperative pain management and haemodynamic management might prevent this potential serious

cardiac condition. Strong clinical suspicion, early use of echocardiography and angiography are recommended to detect perioperative Takotsubo cardiomyopathy. Further study is definitely warranted to identify the proper patho-physiology and susceptible patients to prevent this complication in future.

Piyush N Mallick, Surjya Prasad Upadhaya, Anupam K Das[1], Raj Kumar A Singh

Department of Anaesthesiology and Critical Care, Al Jahra Hospital, Ministry of Health, [1]Anaesthesiologist, Chest Hospital, Ministry of Health, Kuwait

Address for correspondence:
Dr. Piyush N Mallick,
Department of Anaesthesiology and Critical Care,
Al Jahra Hospital, Ministry of Health, Kuwait.
E-mail: piyush_mallick@hotmail.com

REFERENCES

1. Virani SS, Khan AN, Mendoza CE, Ferreira AC, de Marchena E. Takotsubo Cardiomyopathy, or Broken-Heart Syndrome. Tex Heart Inst J 2007;34:76-9.
2. Nef HM, Möllmann H, Akashi YJ, Hamm CW. Mechanisms of stress (Takotsubo) cardiomyopathy. Nat Rev Cardiol 2010;7:187-93.
3. Jaguszewski M, Fijalkowski M, Nowak R, Czapiewski P, Ghadri JR, Templin C, *et al*. Ventricular rupture in Takotsubo cardiomyopathy. Eur Heart J 2012;38:1027.
4. Zeb M, Sambu N, Scott P, Curzen N. Takotsubo cardiomyopathy: A diagnostic challenge. Postgrad Med J 2011;87:51-9.
5. Neil CJ, Nguyen TH, Sverdlov AL, Chirkov YY, Chong CR, Stansborough J, *et al*. Can we make sense of takotsubo cardiomyopathy? An update on pathogenesis, diagnosis and natural history. Expert Rev Cardiovasc Ther 2012; 10:215-21.
6. Hessel EA 2nd, London MJ. Takotsubo (Stress) Cardiomyopathy and the Anesthesiologist: Enough Case Reports. Let's Try to Answer Some Specific Questions!. Anesth Analg 2010;110:674-9.
7. Little john FC, Syed O, Ornstein E, Connolly ES, Heyer EJ. Takotsubo cardiomyopathy associated with anesthesia: Three case reports. Cases J 2008;1:227.
8. Brucoli M, Arcuri F, Giarda M, Benech A. Transient cardiac failure due to Takotsubo cardiomyopathy after surgical reduction of nasal fracture. J Craniofac Surg 2011; 22:1907-10.
9. Sukenaga N, Noma H, Komasawa N, Sugi T, Nakamoto S, Tokuoka T, *et al*. Case of Takotsubo cardiomyopathy after induction of general anesthesia. Masui 2011; 60:957-60.
10. Gologorsky E, Gologorsky A. Intraoperative stress cardiomyopathy. J Am Soc Echocardiogr 2010;23:340.e3-4.

Access this article online	
Quick response code	Website: www.ijaweb.org
	DOI: 10.4103/0019-5049.111856