TAKOTSUBO CARDIOMYOPATHY AFTER INGESTION OF RODENTICIDE AS SUICIDAL ATTEMPT: A CASE REPORT

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ABSTRACT
Takotsubo cardiomyopathy or left ventricular apical ballooning syndrome is an acute cardiac syndrome precipitated by emotional stress. Patients present with chest pain, ecg changes and elevated cardiac enzymes consistent with acute myocardial infarction but have normal coronary arteries on coronary angiography. Here, we report a 29 year female patient who developed the disease after ingestion of rodenticide as a suicidal attempt.

Key Words: Apical Ballooning and Takotsubo cardiomyopathy

INTRODUCTION
Takotsubo cardiomyopathy is an acute cardiac condition that involves left ventricular apical ballooning and mimics acute myocardial infarction. It is described by various names such as transient left ventricular apical ballooning syndrome, stress-induced cardiomyopathy, and broken heart syndrome. Takotsubo is a japanese word meaning octopus pot, which is used to trap octopus, and has a similar visual appearance of the heart on left ventriculography. It was first described by Sato and colleagues in 1990 in Japan (Sato et al., 1990). Takotsubo Cardiomyopathy patients can present with findings consistent of acute coronary syndrome, like symptoms of chest pain, ST-segment elevation on electrocardiogram and raised cardiac markers (Kurisu et al., 2002). Owing to these clinical characteristics this syndrome is frequently misdiagnosed as an acute coronary syndrome, leading to dilemma of treatment strategy. However; coronary angiography reveals no significant coronary artery disease and the left ventricular apex which is found to be ballooned usually resolving in weeks. Takotsubo Cardiomyopathy syndrome appears to be triggered by emotional or physical stress. High stressful lifestyle in present times has lead to increased incidence of this syndrome. There are multiple case reports in literature of Takotsubo Cardiomyopathy associated with conditions of extreme emotional and physical stress like dancing (Kaballo et al 2011) and suicidal attempt with hanging (Chako et al., 2011). Here, we report a patient who developed the disease after ingestion of rodenticide as a suicidal attempt.

CASE REPORT
A 29 yr old female with alleged history of rodenticide containing zinc phosphide ingestion (Fig. 1) presented with complaints of chest pain and breathlessness on exertion. The initial electrocardiogram taken by referring doctor showed global ST elevation suggestive of acute coronary syndrome hence patient was referred to our centre for further management.

Figure 1: Photograph taken of poison taken by patient
On admission the patient was conscious, oriented with pulse rate of 90 / min, blood pressure of 90/60 mmHg, respiratory rate 28/min, Spo2 96%. On examination S1, S2 normal, gr III/VI systolic murmur at apex. Bilateral basal crepitations heard in lung fields, abdomen was soft with no organonegaly & there was no focal neurological deficit. ECG -ST elevation in all the leads (Fig. 2).

Figure 2: ECG taken on Day 1, 3, 5 and 15 respectively
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Chest X-ray-No Cardiomegaly, Normal Pulmonary Vasculature 2 D Echo-Hypokinesia of anterior wall, anterolateral wall & anterior part of septum, Impaired resting LV systolic function with LVEF of 35%, LA/LV dilated GR II MR. Aortic valve normal, Trivial TR with PPG of 29mmHg, Mild PAH (Fig. 3).

Figure 3: Echo on Day 1: Note the Apical Ballooning and Gr II MR; Echo on Day 5; Echo on Day 15: Note Normal sized LV and Apical Ballooning has reverted to normal
Figure 4: Coronary Angiogram suggestive of normal coronaries Left ventricular angiogram revealing apical akinesia

Cardiac Enzymes: CPK-MB and Trop I were raised 59 and 8.82 respectively. We did not thrombolys the patient as ECG taken at local doctor two days back was same with no progression of ST-T changes. With the background of clinical examination and findings the possibility of myocarditis & coronary artery disease was considered and the patient was treated for the same. Patient underwent coronary angiography which revealed normal epicardial coronaries (Fig. 4). During the course in the hospital serial electrocardiograms were taken which showed gradual resolution of ST-T changes. CPK-MB and Trop I decreased to 29 and 2.72 respectively. Repeat 2 D echo on day 5 showed improved LV systolic function with dyskinesia of apical septum and apex, other regions contracting well, LVEF 50%; 2 D echo on day 15 was normal.

DISCUSSION
There is as no consensus on the diagnostic criteria for takotsubo cardiomyopathy. Researchers at the Mayo Clinic proposed diagnostic criteria in 2004, which have been modified (Prasad et al., 2008); (1) Transient hypokinesis,
akinesis or dyskinesis in the left ventricular mid segments with or without apical involvement; regional wall motion abnormalities that extend beyond a single epicardial vascular distribution and frequently, but not always, a stressful trigger; (2) the absence of obstructive coronary disease or angiographic evidence of acute plaque rupture; (3) new ECG abnormalities (ST-segment elevation and/or T-wave inversion) or modest elevation in cardiac troponin; and (4) the absence of pheochromocytoma and myocarditis.

Patients usually present with chest pain typical of acute coronary syndrome. In most cases, presentation is preceded by an emotionally or physically stressful condition (for example, significant arguments, death of a loved one, natural disasters, legal problems, accidents, surgical procedures, a stay in a critical care unit, and use of illicit drugs). Stress is absent in one third of patients.

A hypothesis by Lyon and colleagues (Lyon et al., 2008) is that takotsubo cardiomyopathy is the result of the direct effect of high levels of catecholamines on the ventricular myocardium. High levels of epinephrine are negatively inotropic and switch beta-2 adrenoceptor coupling in ventricular cardiomyocytes from the Gs protein to the Gi protein signalling pathway. The regional nature of the stunning is explained by the presence of more beta-adrenoceptors in the apical myocardium. This effect is reversible after the epinephrine levels return to normal, and this explains why left ventricular function and apical wall motion return to normal within days to weeks of the acute insult. Although zinc phosphide poisoning is known to cause global myocardial depression however in our case only apical involvement occurred hence the diagnosis of takotsubo cardiomyopathy was made.

CONCLUSION
Takotsubo cardiomyopathy is an uncommon, though a potentially serious condition. Although it is commonly precipitated by stressful life events, these may not be definitive etiological factors. Many patients are thrombolysed unnecessarily because this condition may mimic ST elevation myocardial infarction. Further studies regarding etiological factors, precipitating events and management strategies in this area are required.

REFERENCES


