The Near Fatal Crossing: A Rare Recurrence of Takotsubo Cardiomyopathy in a 66-year-old woman

A Hafiz, F Jan, S Allaqaband

INTRODUCTION

Although first described in 1991 by Dote and colleagues in Japanese population, Takotsubo Cardiomyopathy is being increasingly recognized as a cause of acute “stress cardiomyopathy”. It is characterized by a transient left ventricular apical ballooning (resembling a takotsuno-the Japanese octopus trap) in the absence of a significant coronary artery disease, even though the presentation may mimic an acute coronary syndrome. It is known to be precipitated by emotional stress predominantly in women. Even though recurrence is very rare and has been infrequently reported, recurrence exactly due to the same precipitating event has not been reported. We describe a patient presenting with recurrence of Takotsubo Cardiomyopathy after witnessing an accident similar to the one that precipitated the first episode.

CASE REPORT

A 66-year-old caucasian woman (who lives at a busy crossing frequented by speeding motorcyclists) with a history of a Takotsubo cardiomyopathy and hypertension, presented to the emergency department with acute onset left sided sharp and constant chest pain accompanied by increasing shortness of breath minutes after witnessing a motorcyclist crash into her yard.

In the emergency department (ED) she was hemodynamically stable. On physical examination she was tachypneic (respiratory rate 28 breaths/min) and lung exam demonstrated bibasilar rales. Her initial laboratory work-up was unremarkable. Chest x-ray showed pulmonary edema and a mildly enlarged cardiac silhouette. Electrocardiogram (ECG) revealed a new onset left bundle branch block (LBBB) (Figure 1A). Echocardiogram showed severe hypokinesis of the mid and apical septum and mid and apical wall with an ejection fraction of 30%. She was managed on an “acute coronary syndrome protocol” and given aspirin, enoxaparin, simvastatin, morphine and diuretics. She was emergently taken to the cardiac catheterization laboratory where a coronary angiogram revealed no significant coronary artery disease (Figure 1 B and C), a hyper-contractile left ventricular base with mid to apical ballooning (Figure 1 D and E and Video). A diagnosis of apical ballooning syndrome was made and patient had an uneventful recovery with supportive treatment within two days. Her troponin I peaked (c-TnI) at 4.8 micrograms/liter (normal 0 to 0.4 mcg/L).
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Figure 1
Figure 1: Recurrence of Takotsubo Cardiomyopathy. (A): ECG at time of admission showing a new onset LBBB. (B, C): Catheterization of the coronary arteries revealing left and right coronary arteries largely free of any stenosis at time of catheterization. The left anterior descending artery (LAD) wrapped around the apex in other views. (D, E): Left ventriculography at end-systolic and end-diastolic phases respectively showing hyperkinesis of the base (white arrows) and hypokinesia with ballooning of apex and mid ventricle (white arrowhead).

Our patient had witnessed a very similar accident six years ago outside her house that precipitated a similar presentation. Her ECG in the ED after that accident is shown in Figure 2A. Cardiac catheterization at that time also had exactly the same features (Figure 2B). A follow up echo four years after the initial episode had been normal (Figure 2C). Hence the second presentation at our center was actually a repeat of Takotsubo Cardiomyopathy due to a similar emotional stressor.

Figure 2
Figure 2: First episode of Takotsubo cardiomyopathy. (A): ECG at time of admission consistent with ischemia. (B): Left ventriculography during first episode of Takotsubo cardiomyopathy six years earlier at end-systolic phase showing similar features as the second presentation. (C): M-mode 2D echocardiography four years after the initial episode revealing normal function of the left ventricle and complete recovery.

VIDEO: Left ventriculography during the second episode of Takotsubo cardiomyopathy: Classic features of Takotsubo cardiomyopathy are evident with hyperkinesis of the base during systolic phase and hypokinesia with ballooning of apex and mid ventricle during systole are evident.

DISCUSSION
Takotsubo Cardiomyopathy most commonly presents as a combination of chest pain, ECG abnormalities and a mild elevation of cardiac markers, usually in females with a median age of 63-71 years. It has a very low incidence of recurrence reported in different studies: 0%, 2.7%, 6% and 8% in different series. But recurrence rate due to same stressor is unknown. Our case suggests that it can be precipitated by a very similar stressor—a previously unreported occurrence. It is noteworthy that patients with a recurrence do not differ in baseline characteristics, clinical presentation and in hospital course compared to patients with an initial typical Takotsubo Cardiomyopathy. However the rare recurrence in some patients may be to a genetic predisposition. Identification of a CD 36 deficiency in a
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A patient with Takotsubo cardiomyopathy recently is an interesting finding suggesting a genetic basis for this syndrome. It is also now known that genetic polymorphisms which modulate the catecholamine sensitivity of the adrenoceptors are associated with an increased risk of cardiac injury.

An interesting recent theory by Ibanez et al., suggesting transient coronary thrombosis followed by rapid spontaneous resolution before angiography as the cause of Takotsubo cardiomyopathy, has considered that occlusion of the proximal or middle portion of a left anterior descending (LAD) artery that “wraps around” the apex supplying a large portion of the inferior left ventricular wall could result in the same regional dysfunction as that of Takotsubo syndrome. Our patient had such an LAD, but it was largely disease free. When considering the role of catecholamines as well, such an anatomy of the LAD may have a role in predisposing patients to Takotsubo syndrome; however its role in predisposing patients to recurrence has not been studied.

Many stressors have been identified so far including both emotional (26%) and physical (36%) and in many cases (44%) none have been identified. Among emotional stressors, unexpected death of a loved one is the most common precipitant. The pathophysiology of this syndrome is still poorly understood and possible explanations for myocardial injury seen in the setting of elevated sympathetic stimulation due to stress include catecholamine mediated myocardial stunning, epicardial coronary arterial spasm and coronary microvascular spasm. Even more intriguing is the greater incidence in women (78.6-95%) who seem to be more vulnerable to sympathetically mediated myocardial stunning.

The prognosis for these patients is favorable and a rapid recovery is a hallmark of this syndrome. However in view of a possible recurrence, it is difficult to know how long to continue treatment.

References
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Author Information

Abdul Moiz Hafiz, MBBS
Department of Medicine, Division of Cardiology., Aurora Sinai Medical Center, University of Wisconsin School of Medicine And Public Health, Milwaukee Clinical Campus

Fuad Jan, MD
Department of Medicine, Division of Cardiology., Aurora Sinai Medical Center, University of Wisconsin School of Medicine And Public Health, Milwaukee Clinical Campus

Suhail Allaqaband, MD, FACC, FSCAI, FCCP
Department of Medicine, Division of Cardiology., Aurora Sinai Medical Center, University of Wisconsin School of Medicine And Public Health, Milwaukee Clinical Campus