Atypical Case of Takotsubo Cardiomyopathy with Long QT Complicated by Polymorphic Ventricular Tachycardia in a Premenopausal Woman with Alcohol Intoxication

Omar Sheikh*, Deven Gulick, Gregory Kendall, Navid Berenji and Rushit Kanakia

University Hospital, The University of Texas Health Science Center, San Antonio, United States

*Corresponding Author: Omar Sheikh, University Hospital, The University of Texas Health Science Center, San Antonio, United States.

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Abstract

Takotsubo cardiomyopathy is still not a fully understood disease and continues to elude prompt diagnosis. We present an atypical case of an acutely intoxicated premenopausal woman who presented to the emergency department with elevated troponin levels and electrocardiogram changes after suffering nine seconds of polymorphic ventricular tachycardia and was found to have no substantial coronary artery disease on cardiac catheterization.

Keywords: Takotsubo Cardiomyopathy; Ventricular Tachycardia; Cardiac Catheterization

Background

Takotsubo cardiomyopathy, as described recently by Akashi., et al is a syndrome characterized by transient regional systolic dysfunction of the left ventricle mimicking myocardial infarction, in the absence of obstructive coronary artery disease or plaque rupture [1]. The incidence among individuals exposed to physical or emotional stress is unknown. At this time, it is unclear why postmenopausal women are affected disproportionately as indicated by Lemor., et al or why left ventricular mid-cavity and apex are the sites of the heart that are predominantly affected [2]. We present a unique case of stress induced cardiomyopathy following the onset of polymorphic ventricular tachycardia in a 41-year-old woman in the setting of alcohol intoxication.

Case Presentation

41-year-old female, with a past medical history of hypertension, rheumatoid arthritis, bicuspid aortic valve, third degree heart block status post dual chamber PPM (5/2018), polysubstance use disorder including EtOH use disorder, initially admitted for acute alcohol intoxication (ethanol serum level of 272); urine drug screen was also positive for benzodiazepine, amphetamine, cannabinoid, and opiates. She endured a nine second run of polymorphic ventricular tachycardia with associated syncope in the context of a prolonged QT interval (665 QTc), mild hypokalemia (3.3 mmol/L - normal 3.5 - 5.1), and hypomagnesemia (1.6 - Cardiac outcome optimization greater than 2.0 mg/dL) with R on T phenomenon (Figure 1). She then complained of “fluttering” in her throat. EKG revealed diffuse T wave inversions and elevated troponin levels of 1.9 ng/mL which trended down to 1.6 ng/mL six hour later (Figure 2). Echocardiogram showed apical akinesia consistent with left anterior descending infarct or Takotsubo with depressed left ventricular function, ejection fraction of 35 or 40% (Baseline EF 60 or 65%) (Figure 3). Left cardiac catheterization revealed no evidence of luminal narrowing consistent with a diagnosis of Takotsubo cardiomyopathy. EKG upon discharge revealed that the EKG findings began to normalize, and she no longer had the profound T wave inversions as seen previously. The management for the patient’s final diagnosis of Takotsubo cardiomyopathy, initially treated as an N-STEMI, was per ACS protocol. Patient was loaded with aspirin 325mg and received 81mg daily thereafter. She received Ticagrelor loaded at 180mg and then received 90mg twice daily. She was started on a Heparin drip and a beta blocker. Statins were held initially for elevated LFTs.

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Figure 1: Diffuse symmetric T wave inversions concerning for cardiac ischemia.

Figure 2: 2-Chamber view demonstrating apical ballooning of the left ventricle.

**Outcome and follow-up:** Cardiac catheterization revealed no large vessel disease; and although initially depressed at 35 or 40%, ejection fraction recovered to normal levels (60 or 65%) upon follow-up one week after discharge. Electrocardiogram findings also began to regress to a more normal state; the findings were initially concerning for acute myocardial infarction but the electrocardiogram upon discharge revealed resolution of the large T wave inversions.

**Discussion**

The pathogenesis for Takotsubo cardiomyopathy is not well defined. Most widely accepted mechanisms include catecholamine excess, coronary artery spasm, and microvascular dysfunction as indicated by Beltrame, *et al* [3]. Incidence related to ventricular tachycardia and EtOH use is unknown. Most cases are related to withdrawal rather than acute alcohol intoxication as in our case. However, prolonged QT is well reported in takotsubo, but actual torsades de pointes is rare; we attribute our patient’s Torsades to be a result of the takotsubo given the mild nature of her electrolyte disturbances. In addition, acute alcohol intoxication isn’t associated with the development of Torsades but rather acute alcohol withdrawal which at the time was not the case for our patient. M Kinori., *et al* demonstrated higher rate of torsades in QT prolonged takotsubo males compared to female counterparts [4]. Our patient is in her reproductive years, yet takotsubo is generally linked to postmenopausal women [2]. Multiple reports suggest estrogen deficiency as a pathological link, as estrogen exerts cardioprotective effects (inhibition of excessive sympathetic tone and RAAS activation). Ueyama., *et al* reported increased serum estradiol can diminish the pathological changes in the heart induced by stress [5]. This case highlights there is a large area for improvement in our understanding and illness script formation of this multi-complex disease. Takotsubo likely goes undiagnosed quite often given that the population characteristics and risk factors can vary widely as seen in our case; more importantly, they drastically contradict the prototypical population that presents with acute coronary syndrome. Premenopausal women who suffer from takotsubo are likely getting overlooked, underdiagnosed, and subsequently undertreated. Takotsubo should be considered highly on the differential in the setting of the emergency department when a young adult female presents with troponinemia and ECG changes consistent with above. After recognition, bedside echocardiogram can be a tremendous aid in confirmation of the diagnosis as it can reveal the typical findings associated with Takotsubo: akinesis of the apical and/or mid-cavity of the left ventricle. In our patient, bedside echo was delayed because cardiology was not consulted in the first few days of the patient presenting to the hospital. This case is a direct proponent for the argument that not all patient populations should receive the same blanket treatment as dictated in acute coronary syndrome protocol. Atypical patient populations must be heavily reviewed when falling within the umbrella diagnosis of acute coronary syndrome, and suspicion must be high for other, rarer diagnoses such as takotsubo cardiomyopathy.

Conclusion

When a young female, acutely intoxicated with alcohol, presents with symptoms, electrocardiogram findings, and troponin elevations concerning for myocardial infarction, the differential diagnosis needs to include Takotsubo cardiomyopathy.

A relatively quick, simple, and cost-effective way to help support the diagnosis of Takotsubo cardiomyopathy is with the assistance of a bedside echocardiogram. The disease process has a strong predilection for the mid-cavity and apex of the left ventricle.

Patients with Takotsubo cardiomyopathy are falling into the group of acute coronary syndrome and consequently being treated using ACS algorithms. This all stems from the fact that clinical suspicion for the disease is incredibly low thus advocating for increasing awareness of the disease.

Bibliography