Ventricular Tachycardia in a patient with chest pain and apical hypokinesia

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Citation

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Abstract

We discuss a 48 year old female of takotsubo cardiomyopathy, who presented with chest pain, T wave inversion on electrocardiogram (EKG) and elevated cardiac enzymes following an episode of emotional stress. In emergency room she had an episode of sustained ventricular tachycardia. She was taken to cardiac catheterization laboratory, which revealed normal epicardial coronary arteries. Her left ventriculogram revealed a balloon shaped apex.

CASE HISTORY

48-year-old female with past medical history of gastroesophageal reflux disease (GERD), hypertension, hyperlipidemia and diabetes presented with squeezing chest pain, it was 5/10 in intensity and radiated to left arm. The pain lasted up to half an hour and she reported to emergency room. ECG was done which showed T wave inversions in multiple leads and incomplete right bundle brach block(RBBB). Her PR interval was also on higher side of the normal (Fig1). She also had elevated cardiac enzymes with a troponin of 6.8. She was started on heparin, beta-blockers, aspirin and clopidogrel and nitrates. Cardiology consult was sought and they suggested cardiac catheterization. While in ER she had an episode of spontaneously terminating sustained ventricular tachycardia. She remained hemodynamically stable during this episode of ventricular tachycardia. She was taken for cardiac catheterization which revealed normal epicardial coronary arteries. Left ventriculogram was done which revealed a balloon shaped hypokinetic apex during cardiac systole (Fig 2). On further questioning patient revealed that she has recently lost her job and was going through a rough period of her life. Her echocardiogram also showed dyskinetic apex and mid ventricular portion. Her hospital course remained uneventful and she was discharged home after 7 days on beta blockers, angiotensin converting enzyme inhibitors with a diagnosis of Takotsubo cardiomyopathy. She had no recurrence of any ventricular arrythmias during her stay in the hospital.

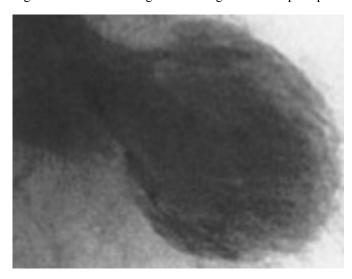
Figure 1

Figure 1: ECG showing diffuse T wave inversions in multiple leads



Figure 2

Figure 2: Left ventriculogram showing balloon shaped apex



DISCUSSION

Takotsubo cardiomyopathy (TC) is known by different names like transient apical ballooning, broken heart syndrome, stress cardiomyopathy, ampulla cardiomyopathy and neurogenic myocardial stunning (1,2,3). This syndrome is usually seen in postmenopausal females during acute emotional or physical stress and is usually reversible(4). Takotsubo is the Japanese name for octopus trap that fishermen still use to catch octopus. In this syndrome, the heart (left ventricle) takes the shape of an octopus trap (takotsubo)(5,6,7). This syndrome is usually confused with acute coronary syndrome as both present with chest pain and elevated biomarkers for cardiac ischemia and electrocardiographic changes. However, coronary angiogram shows nonobstructive arteries in takotsubo cardiomyopathy(1, 8,9,10). Takotsubo cardiomyopathy (TC) is the diagnosis of exclusion, it is imperative to rule out other causes of chest pain and elevated cardiac biomarkers before the diagnosis is $made(_6, _7)$.

Some common complications are arrythmias, mural thrombi, cardioembolic phenomenon, cardiogenic shock, ventricular wall rupture and death(5, 11,12,13). Ventricular arrythmias occur in almost 9% of patients with TC(14,15,16,17,18). The mechanism for these arrythmias is not completely understood but QT interval prolongation and excess catecholamine, ventricular dysfunctions and dyskinesis precipitated by acute emotional or physical stress have been proposed(19). Jaboudan etal(15) reports a case of TC presenting with ventricular tachycardia that warranted treatment with lidocaine. Nault etal(11) describes the mechanism of Torsade de pointes in a patient who developed

prolongation of QT interval following devolpoment of TC. There have been reports of sudden death and takotsubo like cardiomyopathy following earthquakes and lightning strike and the proposed mechanism has been ventricular arrhythmias following acute takotsubo like cardiomyopathy(20). There are even reports of Torsade de pointes occurring in patients after they recover from TC. Denney etal (18, 21) reports a case of left ventricular apical ballooning syndrome with QT interval prolongation in a young man who developed torsade de pointes and experienced aborted sudden cardiac death. Patient had a complete recovery of cardiac function and normalization of QT interval in a few days. Our patient had a normal QT interval and had only one episode of sustained ventricular tachycardia that was self terminated and did not recur.

CONCLUSION

Ventricular arrhythmias are potentially fatal complication of TC. Prolongation of QT interval during acute phase of TC has been proposed a culprit mechanism. Our patient had no ECG evidence of prolonged QT and still had an episode of sustained ventricular Tachcardia. We conclude that all patients of takotsubo cardiomyopathy need to be closely monitored for such potentially fatal arrythmias regardless of prolongation of QT interval especially during acute phase of illness.

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