Case Report

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Neurogenic Heart Syndrome: A Rare Complication in A Syncopal Attack Patient

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Abstract

This 50 year old lady presented to the emergency with history of one episode of syncopal attack followed by fall on the floor and involuntary movement of body parts. No history of vomiting and ENT bleeding. On presentation she was conscious, oriented to time, place and person and her vital status were heamodynamically stable. This case highlights the occurrence of Takotsubo Cardiomyopathy (TCM) in a middle age female patient perimenopausal women.

Keywords: Cardiomyopathies; Cardiogenic Shock; Blunt Cardiac Injury; Stress Cardiomyopathy.

Introduction

First described by Sato et al in Japan in 1990,Stress Cardiomyopathy (also known as Takotsubo Cardiomyopathy (TCM) or 'Broken Heart' syndrome) is a newly recognized disorder of unknown etiology. It leads to sudden onset nonischemic cardiomyopathy that result in akinesis of the left ventricle, classically the apex.

Stress Cardiomyopathy is usually triggered by sudden emotional or physical stress - characterized by rapid development and subsequent spontaneous resolution of severe left ventricular dysfunction. It can appear identical to myocardial infarction with elevated cardiac biomarkers and electrocardiographic changes.

More recently, the ''inverted-Takotsubo'' cardiomyopathy, has been described as a variantof this syndrome, that causes dysfunction of the basal and mid-ventricularsegments with preserved function of the apical segments.

Though thought to be rare, it is a life-threatening condition with high mortality rates. A prompt diagnosis and treatment, as in our case, is often lifesaving. We describe a case of stress cardiomyopathy in a 50 years old healthy female with syncope.

Clinical Case

We present the case of a 50 years old female with no significant past medical history presented to the Emergency Department with a history one episode of syncopal attack in bathroom at her home around 2:00pm, followed by fall on the floor and involuntary movement of body parts,with no history of vomiting and ENT bleeding.

Treated at local hospital as a case of myocardial infarction and she received loading dose. On arrival the patient was fully conscious and oriented. She was heamodynamically stable with a blood pressure of 140/90, heart rate of 80bpm, respiratory rate of 18 bpm, and oxygen saturation of 100% on room air, GCS-E3V4M6. EKG shows ST elevation in lead v1-v3, Trop T-POSITIVE, CT brain plain shows Subarachnoid haemorrhage. She was shifted to I.C.C.U.She was evaluated by consultant cardiologist and adviced to withhold all antiplatelet medication

and bedside ECHO revealed normal biventricular function with LVEF around 60% and no pericardial effusion or RWMA. She was also seen by a Neurologist consultant and his advised were folloed. She remained asymptomatic over the next 24 hours and was planned for transfer under Neurosurgeon consultant care.

Under Neurosurgeon consultant brain angiography was done and it shows A large lobulated saccular aneurysm visualised at the right middle cerebral artery bifurcation which is posteroinferior laterally directed having a wide fundus and gross wall irregularly.

Two units prbc transfused; antiepileptic and antihypertensive drug started, taget blood pressure 140/80 mmhg; potassium chloride was also started and Her antibiotics were escalated as total leukocyte count was 13000; haemoglobin-8g/dl; platelet count-129000; sodium-142; potassium-3.0; crp-64.4; ck-21; cpkmb-352; pre op serology negative. One day two patient underwent right middle cerebral artery Aneurysm clipping under general anaesthesia.Post Aneurysm patient was shifted to I.T.U. After Aneurysm clipping for around 24 hours patient BP drop to 100/60 mmhg and vasopressor support initiated and antihypertensive medication was withhold, target blood pressure 140/80 mmhg. One days 6 ct brain plain was repeat and it shows right temporal lobe infarct with mass defect on the ventrum. On day 10 vasopressor support was tapered and gradually withdrawn.

At this point repeat echocardiography was done and there was no significant changes from the previos echo,subsequently repeat blood investigation shows,hemoglobin-8.8g/dl,total leucocyte count-15500,sodium-145,potassium-5.0 and a chest X-ray shows left lower zone infiltration and indistinct left costo-phrenic angle. She was then refer to a medicine consultant and advice to escalate higher group of antibiotics and hyperkalemic correction. She gradually improved and on day 14 repeat total leucocyte count 13000, chest x-ray shows significant improvement in comparison to the previous X-ray. Patient was then tolerating oral feeding, she began to sit up and have good seating balance, on making her ambulate she was ataxic and was seen by a Psychiatrist consultant who supervised her rehabilitation, thereafter she was able to ambulate to the bathroom with support. Her verbal response is slightly irrelevant. She was discharged with follow up advice of repeat ct angiography of brain after 3 months to check for completeness of clipping.

Discussion

Takotsubo Cardiomyopathy or "Broken Heart syndrome" has emerged as an important cause of acute onset; severe left ventricular dysfunction in response to a stressful event. The predominant population has definitely been post-menopausal women in nearly all case series described so far. Prasad et all found in their study that about 90% of the subjects were women with a mean age between 58 to 75 years. There have been multiple possible etiologies suggested for TCM most of which have revolved around an acute stressor as a precipitant including death of a family member, automobile accidents and natural disasters such as earthquakes among others. In the case described here the victim had a syncope followed by fall on the floor with no significant external injuries nor any bony fracture– this potentiates the theory that catecholamine overdrive can play a significant role in the pathophysiology of TCM. Trauma has been associated with systemic inflammatory response and catecholamine surges – both of which have a central role in the pathophysiology of cardiogenic shock and can very well be of great significance in the development of TCM. The fact that TCM has been invariably associated with either physical or mental stress lends credence to the role of catacholamines. The role of other inflammatory mediators which are released secondary to stress such as interleukins, tumour necrosis factor, nitric oxide may also be related to the pathophysiology of TCM. The female predisposition to TCM might be linked to gender differences in the myocardial sensitivity to circulating catecholamines.

In most of the case series reported so far it has been seen that TCM patients develop transient ECG changes including anterior lead ST segment elevations and deep evolutionary T wave inversion with QT prolongation over the first 24 hours while troponins were only marginally elevated. However in our case we found that the patient had only minimal ST segment elevation – however the cardiac biomarker were slightly elevated. In this case the recorded LV ejection fraction is 60%. Therefore we feel that it is interesting to note that TCM can also present with even mild elevations of troponins and other cardiac biomarker and with slight st elevation on EKG in a preserve EF. In a prospective study of 3265 patients who presented with troponin-positive acute coronary syndrome, Kurowski et al found 35 cases of transient cardiomyopathy of which 21 (60%) had the classic apical wall motion abnormality while 14 (40%) had

the inverted mid-ventricular pattern. The effect of age on the distribution should be investigated as there may be age related variation in the wall motion abnormalities seen in TCM patients.

Though there are no consensus guidelines on the treatment protocol for patients with TCM. The following modalities have been suggested in literature: (1) fluid resuscitation (if respiratory status allows) and (2) betablocker administration to reduce contractility, slow the heart rate, and increase end-systolic volume. However in our case the patient had presented with normotensive which later on need a vasopressor support due to falling of blood pressure. Therefore in patients with TCM who are presenting with severe hypotension we would advocate the early use of IABP support. In this case the patient had a IABP support on the later days.

Conclusion

This case illustrates several interesting features. Here we have TCM developing in a perimenopausal patient aged 50 years following syncope – Here mild elevated troponins and other cardiac biomarker along with slight st elevation in EKG, with preserved EF. This case demonstrates interesting deviations from the general descriptors of this condition as is documented in literature while leading credence to the role of catecholamine surges and inflammatory mediators in the pathophysiology of TCM.

Key Message

Takotsubo Cardiomyopathy can occure in middle age patients following syncope.

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