



ORIGINAL RESEARCH PAPER

Medicine

MENDING THE BROKEN HEART

KEY WORDS:

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ABSTRACT

Takotsubo Cardiomyopathy, also known as Stress induced Cardiomyopathy or the BROKEN HEART SYNDROME, is a rare condition characterized by transient decrease in ejection fraction and symptoms mimicking Acute Coronary Syndrome, accompanied by hypokinesia of apex of left ventricle, with ballooning of left apex with non obstructive coronary artery disease. Seen primarily in Whites, Asians, females with postmenopausal preponderance, it usually follows intense emotional and mental stress. Patients present with dyspnea, pulmonary edema and at times with cardiogenic shock. Despite clinical symptoms, most patients have almost normal coronaries on cardiac catheterization and this actually establishes the diagnosis. Here we discuss a case of an Asian postmenopausal female who presented with dyspnoea and shock following intense mental stress at home.

Case report

A 64 year old female with a known history of hypothyroidism, primary hypoparathyroidism, sick sinus syndrome and hypertension (on regular medication) was admitted with sudden onset giddiness and uneasiness for one day along with one episode of vomiting. History revealed that she had lost her only child under very tragic circumstances and had been continuously brooding over it for the past one week. She did not have any known allergies. Her family history was insignificant.

The patient was conscious, alert to time, place and person and was afebrile (97.4 F). Her skin was normal without any rashes. There was no evidence of clubbing, or cyanosis. Her peripheral pulses were normal. There was no focal neurological deficit. RBS was 78 mg/dl, pulse 90 /min, BP 60 mmHg systolic, respiratory rate of 18 breaths/min, and oxygen saturation of 93% on room air. Extremities were cold and clammy and she was drenched in sweat.

Her lungs had evidence of basal crepitations. Her heart sounds were faint, there was an S3 gallop. Her abdominal examination revealed a soft, nontender abdomen, and there was no organomegaly.

ECG on admission had tall T waves in V2-V4 (figure 1). The patient was initially treated for hypotension with iv fluids, but she developed shortness of breath after infusing 750 ml of fluid. Patient was shifted to the cardiac cathlab where a 2D ECHO screening suggested normal size cardiac chambers with akinetic apex and hypokinetic mid anterior septum, mid septum, mid anterior wall, EF was around 40%, mild MR, and trivial TR, along with grade 1 diastolic dysfunction. Chest X ray was done which was suggestive of pulmonary edema (Figure 2). B-type natriuretic level was 215 pg/ml (normal range up to 100 pg/ml). and TROP I was positive - 6.14 ng/ml (normal range, 0 to 0.4 ng/ml). CPK MB was raised -37.

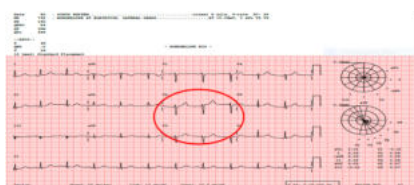


Figure 1 - ECG: Tall T wave in V2-V4, poor R wave progression



Figure 2 - CX ray: suggestive of pulmonary edema (day1)

Figure 3 - Chest X ray: Resolution of pulmonary edema (day3)

Table 1-Tabular laboratory reports

PARAMETERS	10-Oct	13-Oct
PROTHROMBIN TIME(INR)	0.92	
SERUM CALCIUM	8.9	
TSH	3.08	
HIV	NEGATIVE	
HBSAG	NEGATIVE	
HCV	NEGATIVE	
CPK MB	37	
TG	100	
LDL	132	
TOTAL CHOLESTEROL	218	
LFT	NORMAL	
SERUM MAGNESIUM	1.87	
SERUM CREATININE	1.11	1.23
HB	12.7	12.9
TLC	11800	

Based on above findings she was treated for Acute coronary syndrome and left ventricular failure with antianginals, antiplatelets, statins, diuretics, oxygen and low molecular weight heparin. Next day her dyspnoea settled some what and she was taken for Coronary Angiography which was done through the right radial route

CAG showed slow flow in LAD and RCA with minor plaques in LAD which took us by surprise.(figure 5-7).We were actually expecting significant blockages in the LAD .

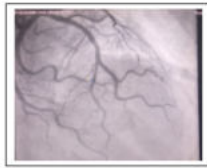


Figure 4: LCX



Figure 5: RCA

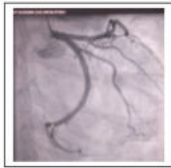


Figure 6: LAD

In the absence of such findings we now knew that we were dealing with a case of TAKOTSUBO CARDIOMYOPATHY following a bout of extreme stress. A 2D ECHO done 3 months prior had revealed a normal heart with an EF of 70%. Most of the cardiac drugs were hence stopped – only the diuretics were continued. On day 3, she was much comfortable and was having a saturation of 94% on room air with a respiratory rate of 14 per minute. Chest X ray showed clear lung fields (Figure 3). She was discharged the next day.[day 4] in a haemodynamically state.

She was comfortable in her further follow up and a repeat 2D ECHO six weeks later showed normal ejection fraction.[65 %]

Discussion

Takotsubo Cardiomyopathy also known as (stress cardiomyopathy, apical ballooning syndrome, broken heart syndrome) is defined as transient systolic dysfunction due to any kind of stress which usually mimics acute coronary syndrome, with wall motion abnormality but without evidence of obstructive coronary artery disease or plaque rupture.

There is no evidence of any blockages in coronary angiogram, but there may be marginal but rapid rise of cardiac biomarkers like TROP I and CPK MB unlike in acute myocardial infarction where we get slow but high values of the same, similarly echocardiogram shows left ventricular dysfunction.

Usually there is no standard treatment, but presenting complaints need to be addressed.

Normal myocardium utilizes most of its energy from fatty acid metabolism, but during ischemia this pathway is suppressed and glucose is utilized instead of fatty acid resulting in impaired cardiac function

Most commonly accepted possible mechanism is considered to be stress induced catecholamine release, eventually resulting in stunning of myocardium confirmed by endomyocardial biopsy which demonstrated reversible contraction band necrosis, mononuclear infiltrates and focal myocytolysis and by myocardial imaging studies

ECG may mimic acute coronary syndrome with STT changes, as was seen in our case. (tall T waves)

According to another opinion possible hypothesis might be neurohormonal stimulation in susceptible humans especially females resulting in acute myocardial dysfunction as reflected by LV wall motion abnormality. Though this is under review regarding possible triggering mechanism. Possible reason can be direct myocardial toxicity, epicardial vessel occlusion, thrombosis or multivessel spasm Review of literature also reports cases of Takotsubo Cardiomyopathy following intake of drugs like cocaine, excessive phenylephrine use and use of methamphetamine. Some studies have also shown to have higher levels of serum catecholamines (norepinephrine, epinephrine, dopamine), than seen in patients of acute myocardial infarction, as apical portion of left ventricle has more innervation, that could

possibly explain reason for involvement of apex of left ventricle in Takotsubo Cardiomyopathy.

According to another school of thought, underlying coronary endothelial dysfunction may also play important role as abnormal tendency towards spasticity in coronaries which can manifest as angina due to diffuse ,transient spastic obliteration of coronaries.

To summarize the exact etiology is not known , but based on above postulations following can be possible reasons

1. Underlying coronary endothelial dysfunction
2. Impaired myocardial fatty acid metabolism
3. Endogenous catecholamine induced myocardial stunning
4. Multivessel coronary artery spasm

Usually seen in post-menopausal women (9) undergoing intense physical and emotional stress. This is a temporary condition and condition reverses after the initial insult. Different stress includes condition like sudden drop in blood pressure, financial loss, intense pain, panic, public speaking, fierce argument, unexpected loss of someone close, receiving bad news etc. Takotsubo Cardiomyopathy has also been reported after near drowning states (8). Study of various patients by investigators found out amongst young patients, there was female preponderance (10). Studies have also shown both groups(Takotsubo and ACS) have similar rates of inpatient complications like shock and death. There is preponderance of Asians and Whites and amongst females more than 90% were postmenopausal.

Prognosis is good and >95% and patient usually recover completely within 4-6 weeks

Now , if we see our case , she was a post menopausal female and her symptoms were triggered by emotional stress. On admission she had ECG changes suggestive of acute coronary syndrome and features of left ventricular failure and slight raised Troponin I values, but next day after she settled and stabilized, she was taken for angiography. She had almost normal coronaries except for mild LAD plaques. Her dyspnoea disappeared and was evident from serial chest x rays which showed resolution of pulmonary odema .When she was followed up after 6 weeks, her ejection fraction was also normal. A DUAL CHAMBER pacemaker was implanted three months later when she developed undue fatigue due to chronotropic incompetence.

Review of literature has shown many cases where the triggering factors varied from reasons like post procedural endoscopy (1), intense emotional trauma, mental stress due to prolonged hospital stay(2) , during myasthenia crisis (3) , in a patient admitted for alcohol withdrawal (5), in case of recently widowed lady (6) and post Zumba (7),.In one particular case, the patient even had a cardiac rupture eventually leading to death (proven on autopsy) (4)

Conclusion:

In the article above , we have tried to highlight how extreme emotional stress could hamper cardiac function suddenly – a phenomenon called STRESS or TAKOTSUBO CARDIOMYOPATHY- every physician or medical officer must be in a position to diagnose and treat such a condition since the returns could be gratifying ,both to him and the patient per se. ECG changes could be misleading- the diagnosis can finally be clinched by a low EF and apical ballooning on 2D ECHO with normal coronaries on a CAG .Its actually a herculean task for a team of physicians to mend the BROKEN HEART.

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