ORIGINAL RESEARCH PAPER
Volume - 12 | Issue - 06 | June - 2023 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

CASE REPORT: A RARE CASE OF TAKOTSUBO CARDIOMYOPATHY

Medicine

Dr. Sohan M	Junior resident, Department of General Medicine, Shridevi Institute of Medical Sciences and Research Hospital, Tumkur-572106, Karnataka, India
Dr. Suresh S	Interventional cardiologist, Shridevi Institute of Medical Sciences and Research Hospital, Tumkur-572106, Karnataka, India
Dr. Virupaksha K. L	Professor and HOD, Department of General Medicine, Shridevi Institute of Medical Sciences and Research Hospital, Tumkur-572106, Karnataka, India
Dr. Naveen K	Professor, Department of General Medicine, Shridevi Institute of Medical Sciences and Research Hospital, Tumkur-572106, Karnataka, India
Dr. Raman M. H	Professor, Department of General Medicine, Shridevi Institute of Medical Sciences and Research Hospital, Tumkur-572106, Karnataka, India

ABSTRACT

Takotsubo cardiomyopathy also known as stress induced cardiomyopathy is a transient regional left ventricular dysfunction that is triggered by emotional or physical stress. We report a case of 47 year old lady from Tumkur city who presented with acute onset chest pain and breathlessness. Her initial ECG, cardiac biomarkers and echocardiography were suggestive of acute coronary syndrome. Her CAG showed no evidence of occlusion or plaque rupture. Patient improved clinically with medical therapy. Her follow up echocardiography showed normal left ventricular function.

KEYWORDS

Takotsubo cardiomyopathy, stress induced cardiomyopathy, acute coronary syndrome.

INTRODUCTION

Takotsubo cardiomyopathy involves transient wall motion abnormalities involving left ventricular apex and midventricle[1]. In Japanese, "tako-tsubo" means fishing pot for trapping octopus[2]. The left ventricle characteristically shows apical ballooning during systole, resembling octopus trap, hence the name. It has a specific pathophysiology that likely reflects neurocardiogenic myocardial stunning[1].

It is predominantly preceded by emotional triggers; but physical triggers are also identified[5]. This condition mostly affect women, especially after menopause[3]. It accounts for approximately 1-2% of all cases of suspected acute myocardial infarction[3]. Most patients with stress cardiomyopathy will recover ventricular function rapidly. Although 20% of patients suffer complications such as heart failure, arrhythmias and death[1].

With this background we report a rare case of Takotsubo cardiomyopathy in a 47 year old lady.

Case Presentation

A 47 year old lady, known hypertensive since 5 years on regular medication with history of minor surgical procedure developed acute onset chest pain and breathlessness immediately after the procedure. She was referred to our hospital for further management. On arrival, she had tachycardia, tachypnoea and elevated JVP. However, her BP and peripheral oxygen saturation were within normal limits. On auscultation, S3 and bilateral basal crackles were heard. Her ECG showed ST depression in lateral and precordial leads and cardiac biomarkers were slightly above normal limit.

A bedside echocardiography was done and it showed severe left ventricular dysfunction with apical hypokinesia with basal segment hyperkinesia. Patient was stabilized using loop diuretics, beta blockers, ACE inhibitors and other supportive measures like non-invasive ventilation. She was taken up for CAG and it revealed normal epicardial vessels with no evidence of any plaque rupture. A left ventricular angiogram was done which showed apical ballooning during systole.

Patient improved clinically after 3 days of medical therapy. Her echocardiography after 3 days showed improvement in LV function. She was discharged after 5 days with ACE inhibitors and beta blockers. Her follow up echocardiography after one month showed normal left ventricular systolic function.

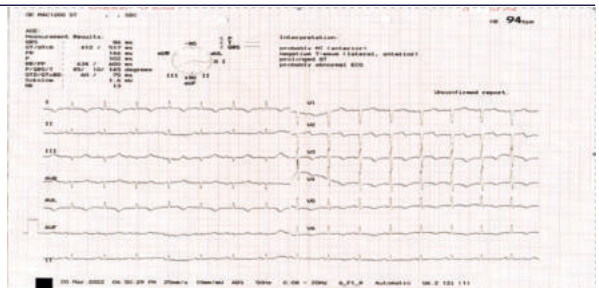

Fig 1: ECG at the time of presentation showing ST segment depression in lateral and precordial leads

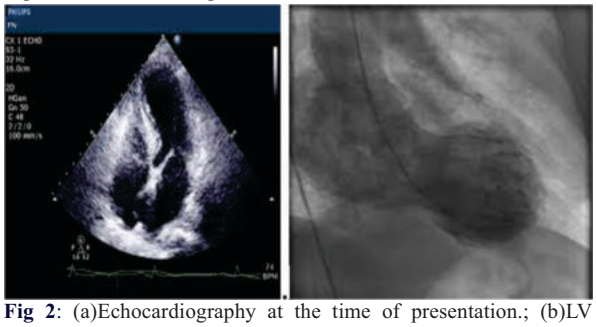

Fig 2: (a)Echocardiography at the time of presentation.; (b)LV Angiogram showing ballooning of left ventricle during systole.

DISCUSSION

Takotsubo cardiomyopathy is an acute reversible condition first recognized in 1990. International Takotsubo Registry shows majority of patients were women, accounting to 89.8% where a vast majority were postmenopausal[1]. The most common clinical presentation involved chest pain, dyspnoea and syncope[1-9]. The risk factors include female sex, post menopause, schizophrenia, anxiety, depression, asthma, diabetes and substance abuse disorders[1]. It is important to distinguish it from acute coronary syndrome since the presentation is similar.

Based on anatomical location of LV hypokinesia there are five different variants of Takotsubo cardiomyopathy, most common being the apical ballooning (apical segment hypokinesia) which constitutes 75-80% of all cases[1,7,10]. This type can result in LV outflow tract obstruction due to SAM[1,3] and/or apical thrombus formation. Other

variants being midventricular (10-20%), basal (5%), biventricular and focal dysfunction which are rare[1].

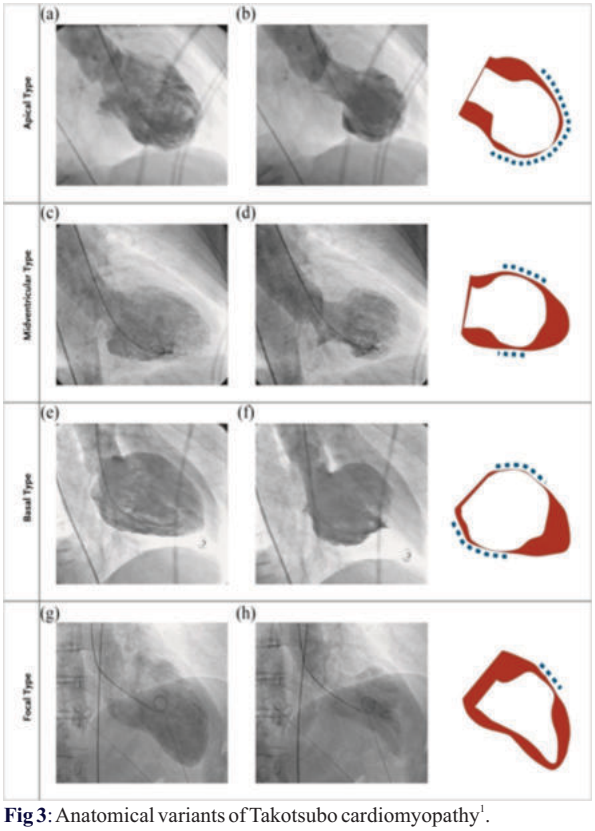

Fig 3: Anatomical variants of Takotsubo cardiomyopathy[1].

The pathophysiology is attributed to excessive catecholamine bioavailability[10]. Any kind of emotional stress-positive/negative or physical stress such as trauma, surgery, intoxications, drug withdrawal will stimulate HPA axis that causes excessive release of epinephrine from adrenal gland and epinephrine release that are stored in the presynaptic terminal of postganglionic neurons. This sudden spillage at the myocardial level induces beta-2-adrenoreceptor coupling from G_s to G_i as a cardiac myocyte protective mechanism which results in decreased cardiac myocyte contractile function- "Direct catecholamine toxicity". Additional to this, there is acute microvascular dysfunction which causes cardiac myocyte ischemia resulting ultimately in myocardial stunning[1,9,10]. Since beta-2-adrenoreceptors are abundantly located in the apex, it is most commonly affected in this condition. A possible role of genetic factors is proposed where L41Q polymorphism of GRK5 is involved resulting in diminished reaction of beta adrenergic receptor to catecholamine stimulation[6].

The most common ECG changes that can be observed is ST segment elevation and/or T wave inversion[6]. Other ECG changes that can be observed include QT prolongation, ventricular tachycardia, ventricular fibrillation, Torsade de pointes[6] and abnormal Q waves in precordial leads that resolve within days to weeks[7]. Electrocardiography shows LV dysfunction due to aberration of regional wall motion exceeding a single vascular distribution[6]. Cardiac biomarkers can be mildly elevated owing to ischemia due to microvascular dysfunction[6]. CAG helps in accurately ruling out ACS by revealing normal coronary arteries or non-significant atherosclerosis[7,10]. Also, LV angiogram shows characteristic apical ballooning representing the apical variant. The role of cardiac magnetic resonance imaging is becoming increasingly popular to establish the diagnosis of Takotsubo cardiomyopathy as this modality allows accurate identification of reversible myocardial damage by visualization of wall motion abnormalities in each area, quantification of ventricular function and assessment of inflammation and fibrosis[7]. In a study done by HZ Amin et al, it is mentioned that myocardial biopsy can show mononuclear lymphocytes, leukocytes, macrophage infiltration with myocardial fibrosis and contraction bands[6]. Other blood work up like BNP and N-terminal pro BNP is also found to be 3-4 folds higher compared to patients with ACS.

The diagnosis of Takotsubo cardiomyopathy made using InterTAK diagnostic criteria developed by Takotsubo task force[1,6,9,10].

Treatment is mainly symptomatic. It include use of ACE inhibitors or beta blocker. Both of these drugs are used in high risk groups which is classified based on LV ejection fraction (<45%). In hemodynamically unstable patient, vasopressors must be used with caution as it can precipitate LV outflow tract obstruction. Levosimendan can be used for its inotropic action ad vasodilator effects. In the presence of LV outflow tract obstruction, beta locker with alpha-adrenoreceptor agonist along with volume expansion can be used[7]. Anticoagulation is recommended if there is LV clot formation[1,3,7].

Most cases resolve completely or nearly complete within days to weeks. Major adverse events such as cardiogenic shock or death occurs in as many as 5% of individuals with male preponderance.

Recurrence risk is estimated to be about 5% in the first year[1]. Another study showed recurrence of symptom in 11% of patients with Takotsubo cardiomyopathy in a 4-year follow up[7]. Long term use of ACE inhibitors have been shown to reduce recurrence[1,8,9,10]. Co-treatment of depression or other psychiatric disorder has been postulated to reduce recurrence[1].

International Takotsubo Diagnostic Criteria (InterTAK Diagnostic Criteria)

1. Patients with transient[a] left ventricular dysfunction (hypokinesia, akinesia, or dyskinesia) presenting as apical ballooning or other left ventricular segmental (midventricular, basal, or focal) wall motion abnormalities. Right ventricular involvement can be present. Besides these regional wall motion patterns, transitions between all types can exist. The regional wall motion abnormality usually extends beyond a single epicardial vascular distribution; however, rare cases can exist where the regional wall motion abnormality is present in the subtended myocardial territory of a single coronary artery (focal TTS).[b]
2. An emotional, physical, or combined trigger can precede the Takotsubo syndrome event, but this is not obligatory.
3. Neurologic disorders (e.g. subarachnoid haemorrhage, stroke/transient ischaemic attack, or seizures) as well as pheochromocytoma may serve as triggers for takotsubo syndrome.
4. New ECG abnormalities are present (ST-segment elevation, ST-segment depression, T-wave inversion, and QTc prolongation); however, rare cases exist without any ECG changes.
5. Levels of cardiac biomarkers (troponin and creatine kinase) are moderately elevated in most cases; significant elevation of brain natriuretic peptide is common.
6. Significant coronary artery disease is not a contradiction in takotsubo syndrome.
7. Patients have no evidence of infectious myocarditis.[b]
8. Postmenopausal women are predominantly affected.

[a] Ventricular contraction abnormalities may remain for a prolonged period of time or documentation of recovery may not be possible. For example, death before evidence of recovery is captured.

[b] Cardiac magnetic resonance imaging is recommended to exclude infectious myocarditis and diagnosis confirmation of Takotsubo syndrome.[1]

Fig 4: InterTAK diagnostic criteria[1]

CONCLUSION

Due to its close resemblance, presentation and clinical course with acute myocardial infarction, Takotsubo cardiomyopathy should be included as one of the differential diagnoses of acute coronary syndrome. The vast majority of cases among post menopausal women explains the role of estrogen in myocardial protection. Increased awareness of takotsubo cardiomyopathy will likely result in it being diagnosed more frequently. Identifying the stressor and addressing it along with medical therapy helps prevent recurrence.

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