International Journal of Research in Medical Sciences
Khan MT et al. Int J Res Med Sci. 2025 Nov;13(11):5011-5017
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20253643

Review Article

Global expert consensus on Takotsubo syndrome (part I): understanding clinical presentation, diagnostic criteria, and underlying mechanisms

M. Toufique Khan[1]*, Mahbuba Yeasmin[2], Tasmiah Islam Sraya[3], M. Sharmin Akther[4], Solaiman Pramanik[5]

[1]Emergency Department, Labaid Specialized Hospital, Dhanmondi, Dhaka, Bangladesh
[2]Department of Medicine, Dhaka Medical College Hospital, Bangladesh
[3]Grameen Hospital, Noakhali, Bangladesh
[4]Ekushey Hospital, Chittagong, Bangladesh
[5]Department of General Medicine, Al-Hera Hospital, Gazipur, Dhaka, Bangladesh

Received: 22 May 2025
Revised: 23 July 2025
Accepted: 22 August 2025

***Correspondence:**
M. Toufique Khan,
E-mail: mjpsdk23@gmail.com

ABSTRACT

Takotsubo syndrome (TTS), also known as stress-induced cardiomyopathy or broken heart syndrome, is an acute, reversible form of left ventricular dysfunction that clinically mimics acute myocardial infarction but occurs without significant coronary artery obstruction. First identified in Japan in the 1990s, TTS is now recognized globally and accounts for approximately 1-2% of patients with suspected acute coronary syndrome. It predominantly affects postmenopausal women (mean age 58-75 years) and is typically triggered by intense emotional or physical stress. Clinically, TTS presents with sudden chest pain, dyspnea, and ECG changes such as ST-segment elevation or T-wave inversion. The leading pathophysiological hypothesis involves a catecholamine surge leading to myocardial stunning, microvascular dysfunction, and direct myocardial toxicity. Additional factors include neurohormonal, endothelial, and metabolic disturbances. Diagnosis is largely clinical, based on exclusion of coronary artery disease and confirmed via imaging modalities like echocardiography or cardiac MRI, which show characteristic regional wall motion abnormalities, often termed "apical ballooning." The Mayo Clinic Criteria remain the most widely accepted diagnostic framework, emphasizing transient ventricular dysfunction, absence of obstructive coronary lesions, ECG/biomarker abnormalities, and eventual recovery. Although the short-term prognosis is generally favorable, complications such as heart failure, arrhythmias, and thromboembolism may occur. Recurrence rates range from 5-15%, and psychological distress may persist. This review outlines the clinical presentation, diagnostic approach, and pathophysiological insights into TTS, highlighting recent consensus and advances essential for accurate diagnosis and long-term care.

Keywords: Takotsubo syndrome, Stress-induced cardiomyopathy, Apical ballooning, Left ventricular dysfunction, Catecholamine surge, Acute coronary syndrome mimic

INTRODUCTION

Takotsubo syndrome (TTS), also referred to as stress-induced cardiomyopathy or "broken heart syndrome," is an acute and transient cardiac condition characterized by reversible left ventricular dysfunction that frequently mimics acute coronary syndrome (ACS) in its clinical presentation.[1] First identified in Japan in 1990, the

syndrome derives its name from the Japanese word "Takotsubo," referring to an octopus trap that resembles the distinctive apical ballooning of the left ventricle observed on imaging.[2] Initially considered a rare entity, TTS has now been recognized globally, accounting for approximately 1-2% of all suspected acute myocardial infarction (AMI) cases.[3]

The epidemiological profile of TTS reveals a strong female predominance, particularly in postmenopausal women, who account for about 80-90% of cases.[4,5] The mean age at diagnosis ranges between 58 and 75 years.[6] The notable gender disparity has been attributed to the protective cardiovascular effects of estrogen, which diminish after menopause, potentially increasing susceptibility to catecholamine surges and vascular dysfunction.[7]

Clinically, TTS often presents with sudden-onset chest pain, dyspnea, and electrocardiographic changes including ST-segment elevation or T-wave inversions, closely resembling AMI.[8] However, unlike AMI, TTS patients typically exhibit minimal or no obstructive coronary artery disease on angiography.[9] Diagnostic imaging, including echocardiography, left ventriculography, and cardiac magnetic resonance (CMR), reveals regional wall motion abnormalities that extend beyond a single coronary distribution, most commonly involving apical ballooning, but also midventricular, basal, or focal variants.[10,11]

The underlying pathophysiology of TTS remains incompletely understood but is believed to involve an interplay of sympathetic overstimulation, catecholamine excess, coronary vasospasm, microvascular dysfunction, and direct myocardial toxicity.[12-14] A hallmark hypothesis suggests that acute physical or emotional stress triggers an exaggerated sympathetic discharge, resulting in a catecholamine surge that induces myocardial stunning through β-adrenergic signaling and calcium overload.[15,16] Elevated plasma catecholamine levels in TTS patients have been reported to be two to three times higher than those observed in patients with ST-elevation myocardial infarction (STEMI).[17]

Although TTS is often considered a reversible cardiomyopathy, it can lead to serious acute complications, including heart failure, arrhythmias, cardiogenic shock, and thromboembolic events.[18,19] Studies have shown that the in-hospital mortality rate ranges from 1% to 3%, with a 30-day mortality rate estimated at 2.5%.[20] Long-term prognosis is generally favorable, with most patients recovering left ventricular systolic function within 4-8 weeks.[21] However, recurrence occurs in 5-15% of patients, particularly in those exposed to recurrent stressors.[22] Furthermore, some patients report persistent fatigue, reduced exercise tolerance, and psychological distress, such as anxiety or depression, necessitating comprehensive long-term management.[23]

Given the growing recognition of TTS in both cardiovascular and psychosomatic medicine, there is a need for enhanced understanding of its clinical spectrum, pathophysiology, and long-term implications. This review article aims to summarize the current international consensus on TTS, focusing on clinical characteristics, diagnostic criteria, and underlying mechanisms, with the goal of improving clinical recognition and optimizing patient outcomes.[24,25]

CLINICAL CHARACTERISTICS

TTS commonly presents with symptoms that closely mimic those of AMI, making prompt differentiation crucial in clinical settings. Patients typically experience sudden-onset chest pain, dyspnea, and electrocardiographic abnormalities, often prompting emergency coronary angiography. However, a key distinguishing feature is the absence of obstructive coronary artery disease, with normal or near-normal findings on angiography.[26]

Chest pain is the most frequently reported symptom, occurring in up to 90% of cases. The pain is characteristically sudden, severe, and retrosternal, and may persist for several hours. Its similarity to ischemic pain often leads to initial misdiagnosis as STEMI or NSTEMI.[27]

Dyspnea is also a common presenting complaint and is attributed to transient left ventricular systolic dysfunction. In cases of significant myocardial impairment, patients may develop acute pulmonary edema or overt heart failure, necessitating hemodynamic support.[28]

Syncope or presyncope may occur in some patients due to reduced cardiac output or transient arrhythmias, reflecting the hemodynamic instability associated with severe left ventricular dysfunction.[28]

Palpitations may result from arrhythmias, particularly atrial fibrillation, ventricular ectopy, or even ventricular tachycardia, triggered by catecholamine surges and myocardial electrical instability.[29]

Other non-specific symptoms such as nausea, fatigue, sweating, and dizziness have also been reported, though these are less specific and may be related to the underlying stressor or the cardiovascular response to it.

TTS is often preceded by a significant emotional or physical stressor, such as the loss of a loved one, natural disasters, domestic conflicts, or medical illnesses like sepsis or stroke. In about one-third of patients, no identifiable trigger can be found.[26,27]

Epidemiologically, TTS exhibits a strong female predominance, with 80-90% of cases occurring in postmenopausal women, generally between 58 and 75 years of age.[30] This gender bias is believed to result from

decreased levels of estrogen, a hormone known to exert cardioprotective and vasodilatory effects. The loss of estrogen after menopause may heighten sensitivity to catecholamine-induced myocardial injury. In contrast, men who develop TTS tend to be older and are more likely to have comorbidities such as hypertension, diabetes, or coronary artery disease.[30] Understanding these clinical characteristics is essential for accurate diagnosis and appropriate management of TTS, especially in the emergency setting where rapid differentiation from ACS is critical.

DIAGNOSTIC CRITERIA

The diagnosis of TTS is primarily clinical, supported by imaging findings and the exclusion of other cardiovascular conditions, notably ACS and myocarditis. The widely accepted Mayo Clinic Criteria serve as a foundational framework for diagnosis.[31]

Clinical presentation

Patients often present with sudden-onset chest pain, dyspnea, or syncope, frequently following a significant emotional or physical stressor. These stressors may include bereavement, severe anxiety, or acute medical illness.[32]

ECG findings

ECG changes in TTS can mimic those of ACS. Common findings include ST-segment elevation, T-wave inversion, and QT interval prolongation. Notably, these changes are dynamic and may evolve over time. ST-segment elevation is observed in approximately 31.4% of patients within the first 96 hours, while T-wave inversion becomes more prevalent after 24-72 hours.[33]

Cardiac biomarkers

Troponin levels are typically elevated but to a lesser extent than in myocardial infarction. B-type natriuretic peptide (BNP) levels are often significantly increased, reflecting acute heart failure.[34]

Coronary angiography

A hallmark feature of TTS is the absence of obstructive coronary artery disease on angiography. However, studies have shown that up to 23% of TTS patients may have coexisting obstructive coronary artery disease, highlighting the importance of comprehensive evaluation.[35]

Echocardiography or left ventriculography

Imaging typically reveals regional wall motion abnormalities extending beyond a single coronary artery territory. The classic pattern is apical ballooning, but mid-ventricular, basal, and focal variants have also been described.[36]

Cardiac MRI

MRI is instrumental in differentiating TTS from myocarditis and infarction. It can detect myocardial edema without late gadolinium enhancement, a pattern typical of TTS.[37]

Resolution of ventricular dysfunction

A defining characteristic of TTS is the reversibility of systolic dysfunction, typically occurring within days to weeks. This supports the transient nature of the syndrome and distinguishes it from irreversible ischemic damage.[38]

Differential diagnosis

It's essential to differentiate TTS from conditions such as acute myocardial infarction, myocarditis, and pheochromocytoma-induced cardiomyopathy. A complete diagnostic workup-including troponin levels, BNP, coronary angiography, cardiac MRI, and endocrine testing-is vital to ensure an accurate diagnosis.[39]

InterTAK diagnostic score

Developed by the International Takotsubo Registry, this tool aids in differentiating TTS from ACS by incorporating variables such as gender, emotional triggers, ECG findings, and psychiatric comorbidities.[40]

PATHOPHYSIOLOGY

The pathophysiology of TTS is multifactorial and not yet fully elucidated. Several interrelated mechanisms have been proposed, including catecholamine-mediated myocardial stunning, coronary microvascular dysfunction, neurogenic influences, and hormonal factors.

Catecholamine surge and myocardial stunning

A central hypothesis in TTS pathogenesis is the role of excessive catecholamine release during acute emotional or physical stress. Elevated levels of catecholamines, such as norepinephrine and epinephrine, can lead to direct myocardial toxicity, resulting in transient left ventricular dysfunction without necrosis.[41] This catecholamine surge may cause myocardial stunning through calcium overload and oxidative stress, impairing contractility predominantly in the apical segments of the heart.[42]

Coronary microvascular dysfunction

TTS is often associated with coronary microvascular dysfunction rather than significant epicardial coronary artery obstruction. Impaired microvascular perfusion can lead to myocardial ischemia and contribute to the characteristic wall motion abnormalities seen in TTS.[43]

Studies have demonstrated that patients with TTS exhibit reduced coronary flow reserve and endothelial dysfunction, supporting the role of microvascular impairment in its pathogenesis.[44]

Neurogenic mechanisms and the brain-heart axis

Emerging evidence suggests that the central nervous system plays a significant role in TTS development. Alterations in brain regions responsible for autonomic regulation, such as the limbic system, have been observed in TTS patients. These changes may enhance sympathetic output, leading to increased catecholamine release and subsequent myocardial effects.[45] Functional imaging studies have identified structural and functional brain changes in TTS patients, highlighting the importance of the brain–heart axis.[46]

Estrogen deficiency and hormonal influences

The predominance of TTS in postmenopausal women suggests that estrogen deficiency may contribute to its pathophysiology. Estrogen has vasodilatory and cardioprotective effects, and its deficiency may exacerbate endothelial dysfunction and increase susceptibility to catecholamine-induced myocardial injury.[47] The loss of estrogen-mediated protection may also impair the stress response, further predisposing individuals to TTS.[48]

Inflammatory and oxidative stress pathways

Inflammatory responses and oxidative stress are also implicated in TTS. Elevated inflammatory markers and oxidative stress indicators have been reported in TTS patients, suggesting that these pathways may contribute to myocardial dysfunction and recovery processes.[49] The interplay between inflammation, oxidative stress, and catecholamine toxicity may create a synergistic effect, exacerbating myocardial stunning and dysfunction.[50]

MANAGEMENT AND PROGNOSIS

The management of TTS is primarily supportive and tailored to the clinical presentation, aiming to alleviate symptoms, prevent complications, and support myocardial recovery. Since there is no standardized treatment specific to TTS, current strategies are largely extrapolated from the management of acute heart failure and acute coronary syndromes.

Acute management

Beta-blockers are often used during the acute phase to blunt excessive sympathetic stimulation and reduce myocardial oxygen demand. Although their long-term benefit in preventing recurrence remains uncertain, they are frequently prescribed to mitigate catecholamine effects.[51]

Angiotensin-converting enzyme (ACE) inhibitors or angiotensin II receptor blockers (ARBs) may aid in improving left ventricular function and preventing adverse ventricular remodeling. These agents are associated with improved outcomes, especially in patients with reduced ejection fraction.[52]

Diuretics are indicated in the presence of heart failure symptoms or pulmonary congestion to relieve fluid overload and improve symptomatology.[53]

Anticoagulation therapy may be considered in patients with severe left ventricular dysfunction, apical ballooning, or evidence of thrombus formation, to reduce the risk of thromboembolic events.[54]

In rare but severe cases presenting with cardiogenic shock, mechanical circulatory support, including intra-aortic balloon pump or extracorporeal membrane oxygenation (ECMO), may be warranted.[55]

Long-term management and recurrence

Long-term therapy following recovery is individualized. While beta-blockers are commonly continued post-discharge, their efficacy in preventing recurrence remains debatable. Moreover, patients with psychological stressors, anxiety, or depression may benefit from psychiatric evaluation and supportive counseling, given the established role of emotional triggers in TTS.[56]

PROGNOSIS

The overall prognosis of TTS is favorable, with most patients regaining normal cardiac function within days to weeks. However, complications such as arrhythmias, heart failure, left ventricular thrombus, or cardiogenic shock may occur, especially during the acute phase.[57] Reported in-hospital mortality ranges from 1% to 5%, comparable to some forms of acute coronary syndromes.[58]

The recurrence rate of TTS is estimated to be approximately 1.5% to 2% per year, with higher recurrence in patients with a history of emotional stress or psychiatric illness.[59] Hence, long-term follow-up and management of underlying comorbidities, including mental health disorders, are crucial for prevention and early detection of recurrence.[60]

CONCLUSION

TTS is a unique and transient form of stress-induced cardiomyopathy that closely resembles acute myocardial infarction in its clinical presentation but is distinguished by the absence of obstructive coronary artery disease. Although typically reversible with supportive management, TTS can lead to serious complications and recurrence in a subset of patients. The precise pathophysiological mechanisms underlying the syndrome remain incompletely defined, underscoring the need for

ongoing research. A thorough understanding of its clinical features, diagnostic framework, and potential triggers is essential for timely diagnosis, appropriate management, and improved patient outcomes. Continued investigation into the biological and psychosocial factors involved in TTS will be key to advancing therapeutic strategies and preventive care.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Gianni M, Dentali F, Grandi AM, Sumner G, Hiralal R, Lonn E. Apical ballooning syndrome or takotsubo cardiomyopathy: a systematic review. Eur Heart J. 2006;27(13):1523-9.
2. Sato H, Tateishi H, Uchida T. Takotsubo-type cardiomyopathy due to multivessel spasm. In: Kodama K, Haze K, Hori M, eds. Clinical aspect of myocardial injury: from ischemia to heart failure. Tokyo: Kagakuhyouronsya; 1990: 56-64.
3. Templin C, Ghadri JR, Diekmann J, Napp LC, Bataiosu DR, Jaguszewski M, et al. Clinical features and outcomes of takotsubo (stress) cardiomyopathy. N Engl J Med. 2015;373(10):929-38.
4. Sharkey SW, Lesser JR, Zenovich AG, Maron MS, Lindberg J, Longe TF, et al. Acute and reversible cardiomyopathy provoked by stress in women from the United States. Circulation. 2005;111(4):472-9.
5. Prasad A, Lerman A, Rihal CS. Apical ballooning syndrome (Tako-Tsubo or stress cardiomyopathy): a mimic of acute myocardial infarction. Am Heart J. 2008;155(3):408-17.
6. Elesber AA, Prasad A, Lennon RJ, Wright RS, Lerman A, Rihal CS. Four-year recurrence rate and prognosis of the apical ballooning syndrome. J Am Coll Cardiol. 2007;50(5):448-52.
7. Ueyama T. Emotional stress-induced Takotsubo cardiomyopathy: animal model and molecular mechanism. Ann N Y Acad Sci. 2004;1018:437-44.
8. Kurisu S, Sato H, Kawagoe T, Ishihara M, Shimatani Y, Nishioka K, et al. Tako-tsubo-like left ventricular dysfunction with ST-segment elevation: a novel cardiac syndrome mimicking acute myocardial infarction. Am Heart J. 2002;143(3):448-55.
9. Eitel I, von Knobelsdorff-Brenkenhoff F, Bernhardt P, Carbone I, Muellerleile K, Aldrovandi A, et al. Clinical characteristics and cardiovascular magnetic resonance findings in stress (takotsubo) cardiomyopathy. JAMA. 2011;306(3):277-86.
10. Medina de Chazal H, Del Buono MG, Keyser-Marcus L, Ma L, Moeller FG, Berrocal D, et al. Stress cardiomyopathy diagnosis and treatment: JACC State-of-the-Art Review. J Am Coll Cardiol. 2018;72(16):1955-71.
11. Lyon AR, Bossone E, Schneider B, Sechtem U, Citro R, Underwood SR, et al. Current state of knowledge on Takotsubo syndrome: a Position Statement from the Heart Failure Association of the ESC. Eur J Heart Fail. 2016;18(1):8-27.
12. Wittstein IS, Thiemann DR, Lima JA, Baughman KL, Schulman SP, Gerstenblith G, et al. Neurohumoral features of myocardial stunning due to sudden emotional stress. N Engl J Med. 2005;352(6):539-48.
13. Ghadri JR, Wittstein IS, Prasad A, Sharkey S, Dote K, Akashi YJ, et al. International expert consensus document on takotsubo syndrome (part I): clinical characteristics, diagnostic criteria, and pathophysiology. Eur Heart J. 2018;39(22):2032-46.
14. Nef HM, Möllmann H, Kostin S, Troidl C, Voss S, Weber M, et al. Tako-Tsubo cardiomyopathy: intraindividual structural analysis in the acute phase and after functional recovery. Eur Heart J. 2007;28(20):2456-64.
15. Ueyama T, Kasamatsu K, Hano T. Emotional stress induces transient left ventricular hypocontraction in the rat via activation of cardiac adrenoceptors: a possible animal model of "Takotsubo" cardiomyopathy. Circ J. 2002;66(7):712-3.
16. Lee VH, Oh JK, Mulvagh SL, Wijdicks EFM. Mechanisms in neurogenic stress cardiomyopathy after aneurysmal subarachnoid hemorrhage. Neurocrit Care. 2006;5(3):243-9.
17. Wittstein IS, Thiemann DR, Lima JA, Baughman KL, Schulman SP, Gerstenblith G, et al. Neurohumoral features of myocardial stunning due to sudden emotional stress. N Engl J Med. 2005;352(6):539-48.
18. Scally C, Abbas H, Ahearn T, Srinivasan J, Mezincescu A, Rudd A, et al. Myocardial and systemic inflammation in acute stress-induced (Takotsubo) cardiomyopathy. Circulation. 2019;139(13):1581-92.
19. Singh K, Carson K, Shah R, Gilutz H, Usmani Z, Horowitz J, et al. Meta-analysis of clinical outcomes of takotsubo cardiomyopathy. Am J Cardiol. 2014;113(8):1420-8.
20. Templin C, Ghadri JR, Diekmann J, Napp LC, Bataiosu DR, Jaguszewski M, et al. Clinical features and outcomes of takotsubo (stress) cardiomyopathy. N Engl J Med. 2015;373(10):929-38.
21. Summers MR, Prasad A. Takotsubo cardiomyopathy: definition and clinical profile. Heart Fail Clin. 2013;9(2):111-22.
22. Elesber AA, Prasad A, Lennon RJ, Wright RS, Lerman A, Rihal CS. Four-year recurrence rate and prognosis of the apical ballooning syndrome. J Am Coll Cardiol. 2007;50(5):448-52.
23. Ghadri JR, Wittstein IS, Prasad A, Sharkey S, Dote K, Akashi YJ, et al. International expert consensus document on takotsubo syndrome (part I): clinical characteristics, diagnostic criteria, and pathophysiology. Eur Heart J. 2018;39(22):2032-46.
24. Lyon AR, Bossone E, Schneider B, Sechtem U, Citro R, Underwood SR, et al. Current state of knowledge on Takotsubo syndrome: a Position Statement from the Heart Failure Association of the ESC. Eur J Heart Fail. 2016;18(1):8-27.

25. Medina de Chazal H, Del Buono MG, Keyser-Marcus L, Ma L, Moeller FG, Berrocal D, et al. Stress cardiomyopathy diagnosis and treatment: JACC State-of-the-Art Review. J Am Coll Cardiol. 2018;72(16):1955-71.
26. Templin C, Ghadri JR, Diekmann J, Napp LC, Bataiosu DR, Jaguszewski M, et al. Clinical features and outcomes of takotsubo (stress) cardiomyopathy. N Engl J Med. 2015;373(10):929-38.
27. Prasad A, Lerman A, Rihal CS. Apical ballooning syndrome (Tako-Tsubo or stress cardiomyopathy): a mimic of acute myocardial infarction. Am Heart J. 2008;155(3):408-17.
28. Lyon AR, Bossone E, Schneider B, Sechtem U, Citro R, Underwood SR, et al. Current state of knowledge on Takotsubo syndrome: a Position Statement from the Heart Failure Association of the ESC. Eur J Heart Fail. 2016;18(1):8-27.
29. Singh K, Carson K, Shah R, Gilutz H, Usmani Z, Horowitz J, et al. Meta-analysis of clinical outcomes of takotsubo cardiomyopathy. Am J Cardiol. 2014;113(8):1420-8.
30. Ghadri JR, Wittstein IS, Prasad A, Sharkey S, Dote K, Akashi YJ, et al. International expert consensus document on takotsubo syndrome (part I): clinical characteristics, diagnostic criteria, and pathophysiology. Eur Heart J. 2018;39(22):2032-46.
31. Ghadri JR, Wittstein IS, Prasad A, Sharkey S, Dote K, Akashi YJ, et al. International expert consensus document on takotsubo syndrome (part I): clinical characteristics, diagnostic criteria, and pathophysiology. Eur Heart J. 2018;39(22):2032-46.
32. Lyon AR, Bossone E, Schneider B, Sechtem U, Citro R, Underwood SR, et al. Current state of knowledge on Takotsubo syndrome: a Position Statement from the Heart Failure Association of the ESC. Eur J Heart Fail. 2016;18(1):8-27.
33. Margulescu AD, Premawardhana DA, Thomas DE. Prevalence and severity of QT prolongation and other ECG abnormalities in takotsubo syndrome. J Electrocardiol. 2025:88:153848.
34. Scally C, Abbas H, Ahearn T, Srinivasan J, Mezincescu A, Rudd A, et al. Myocardial and systemic inflammation in acute stress-induced (Takotsubo) cardiomyopathy. Circulation. 2019;139(13):1581-92.
35. Napp LC, Cammann VL, Jaguszewski M, Szawan KA, Wischnewsky M, Gili S, et al. Coexistence and outcome of coronary artery disease in Takotsubo syndrome. Eur Heart J. 2020;41(10):1015-23.
36. Okura H. Echocardiographic assessment of takotsubo cardiomyopathy: beyond apical ballooning. J Echocardiogr. 2015;13(4):139-46.
37. Eitel I, von Knobelsdorff-Brenkenhoff F, Bernhardt P, Carbone I, Muellerleile K, Aldrovandi A, et al. Clinical characteristics and cardiovascular magnetic resonance findings in stress (takotsubo) cardiomyopathy. JAMA. 2011;306(3):277-86.
38. Elesber AA, Prasad A, Lennon RJ, Wright RS, Lerman A, Rihal CS. Four-year recurrence rate and prognosis of the apical ballooning syndrome. J Am Coll Cardiol. 2007;50(5):448-52.
39. Isogai T, Matsui H, Tanaka H, Fushimi K, Yasunaga H. Clinical characteristics of patients with Takotsubo syndrome diagnosed without coronary artery evaluation: A retrospective nationwide study. J Cardiol. 2018;71(3):268-76.
40. Ghadri JR, Cammann VL, Napp LC. Differences in the clinical profile and outcomes of typical and atypical Takotsubo syndrome: data from the International Takotsubo Registry. JAMA Cardiol. 2016;1(3):335-40.
41. Lyon AR, Rees PS, Prasad S, Poole-Wilson PA, Harding SE. Stress (Takotsubo) cardiomyopathy-a novel pathophysiological hypothesis to explain catecholamine-induced acute myocardial stunning. Nat Clin Pract Cardiovasc Med. 2008;5(1):22-9.
42. Wittstein IS, Thiemann DR, Lima JA, Baughman KL, Schulman SP, Gerstenblith G, et al. Neurohumoral features of myocardial stunning due to sudden emotional stress. N Engl J Med. 2005;352(6):539-48.
43. Kume T, Akasaka T, Kawamoto T. Assessment of coronary microcirculation in patients with Takotsubo-like left ventricular dysfunction. Circ J. 2005;69(8):934-9.
44. Elesber AA, Prasad A, Lennon RJ, Wright RS, Lerman A, Rihal CS. Four-year recurrence rate and prognosis of the apical ballooning syndrome. J Am Coll Cardiol. 2007;50(5):448-52.
45. Templin C, Hänggi J, Klein C. Altered limbic and autonomic processing supports brain-heart axis in Takotsubo syndrome. Eur Heart J. 2019;40(15):1183-7.
46. Markousis-Mavrogenis G, Pepe A, Bacopoulou F. Combined brain-heart imaging in takotsubo syndrome: towards a holistic patient assessment. J Clin Med. 2024;13(10):2991.
47. Ueyama T. Emotional stress-induced Tako-tsubo cardiomyopathy: animal model and molecular mechanism. Ann N Y Acad Sci. 2004;1018:437-44.
48. Summers MR, Prasad A. Takotsubo cardiomyopathy: definition and clinical profile. Heart Fail Clin. 2013;9(2):111-22.
49. Scally C, Rudd A, Mezincescu A. Persistent long-term structural, functional, and metabolic changes after stress-induced (Takotsubo) cardiomyopathy. Circulation. 2018;137(10):1039-48.
50. Pelliccia F, Kaski JC, Crea F, Camici PG. Pathophysiology of Takotsubo syndrome. Circulation. 2017;135(24):2426-41.
51. Templin C, Ghadri JR, Diekmann J, Napp LC, Bataiosu DR, Jaguszewski M, et al. Clinical features and outcomes of takotsubo (stress) cardiomyopathy. N Engl J Med. 2015;373(10):929-38.
52. Singh K, Carson K, Shah R, Gilutz H, Usmani Z, Horowitz J, et al. Meta-analysis of clinical outcomes of takotsubo cardiomyopathy. Am J Cardiol. 2014;113(8):1420-8.
53. Kurisu S, Inoue I, Kawagoe T. Clinical features of Takotsubo cardiomyopathy in the acute phase:

comparison with acute myocardial infarction. Circ J. 2003;67(7):687-90.
54. El-Battrawy I, Lang S, Ansari U. Incidence and clinical impact of recurrent Takotsubo syndrome: results from the GEIST Registry. J Am Heart Assoc. 2019;8(9):e010753.
55. Sharkey SW, Windenburg DC, Lesser JR. Natural history and expansive clinical profile of stress (Tako-tsubo) cardiomyopathy. J Am Coll Cardiol. 2010;55(4):333-41.
56. Kastaun S, Gerriets T, Schwarz N. Impact of psychological risk factors on Takotsubo cardiomyopathy. BMC Cardiovasc Disord. 2014;14:80.
57. Citro R, Rigo F, D'Andrea A. Echocardiographic correlates of acute heart failure, cardiogenic shock, and in-hospital mortality in Takotsubo cardiomyopathy. JACC Cardiovasc Imaging. 2014;7(2):119-29.
58. Singh K, Parsaik A, Singh B. Meta-analysis of clinical correlates of acute mortality in takotsubo cardiomyopathy. Am J Cardiol. 2014;113(8):1420-8.
59. Ghadri JR, Sarcon A, Diekmann J. InterTAK Registry. International expert consensus document on Takotsubo syndrome (Part II): diagnostic workup, outcome, and management. Eur Heart J. 2018;39(22):2047-62.
60. Delmas C, Lairez O, Meliota T. Outcomes of patients with Takotsubo syndrome and baseline cancer: a prospective multicenter study. J Am Coll Cardiol. 2020;76(9):1000-9.

Cite this article as: Agrawal S, Das V, Agarwal A, Pandey A. Fetal Doppler for prediction of adverse perinatal outcome in preeclampsia in a low resource setting. Int J Res Med Sci 2025;13:5011-7.