This article can be cited as: Jafari N, Shahabi Rabori V. Stress-induced cardiomyopathy in young military personnel during combat: a case report. Cardiovasc Biomed J. 2025; 5(2): 5-12.

Case Report

Stress-induced cardiomyopathy in young military personnel during combat: a case report

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Article Information:
Received: 27 Aug 2025; Revised: 12 Nov 2025; Accepted: 22 Dec 2025 DOI: 10.18502/cbj.v5i2.21132

Abstract

Objectives: Takotsubo cardiomyopathy (TTC), referred to as stress-induced cardiomyopathy, is characterized as a transient syndrome of myocardial dysfunction mimicking acute coronary syndrome that occurs without significant coronary artery disease (1). While predominantly occurring in postmenopausal women, there is a growing number of young men experiencing extreme stress who are also being diagnosed with TTC.

Case Summary: We present a 21-year-old male soldier who presented with acute cardiac arrest with electrocardiographic signs of extensive anterior ST-elevation myocardial infarction while in active military service in a war zone. Despite an initial presentation suggestive of massive myocardial infarction with severely elevated cardiac biomarkers and cardiogenic shock necessitating prolonged cardiopulmonary resuscitation, coronary angiography revealed normal coronary arteries. Remarkably, full cardiac and neurological recovery occurred within days, confirming the diagnosis of stress- induced cardiomyopathy.

Conclusions: This case underscores the clinical importance of considering TTC for young patients who exhibit signs of acute coronary syndrome, particularly in high-stress settings such as military combat zones.

Keywords: Takotsubo cardiomyopathy, stress cardiomyopathy, young adults, military personnel, combat stress, STEMI

Introduction

Takotsubo cardiomyopathy (TTC), initially described by Japanese physicians in 1990, is an acute cardiac syndrome characterized by reversible regional left ventricular dysfunction in the absence of obstructive coronary artery disease (1). While TTC predominantly affects postmenopausal women in over 80% of cases, recent epidemiological data indicate concerning trends among young males, who demonstrate more than twice the mortality rate compared to their female counterparts (2, 3). Young patients with TTC present unique clinical challenges, being more prone to atypical features with fewer classic cardiovascular comorbidities, yet paradoxically facing higher rates of in-hospital complications and mortality (4). Military personnel represent a particularly vulnerable group due to exposure to extreme psychological stressors, sleep deprivation, physical exertion, and combat-related trauma (5, 6). The complex neuroendocrine pathophysiology involving sudden surges in

sympathetic activation and catecholamine release makes military settings particularly high-risk environments for TTC onset, especially during combat deployments where multiple stressors interact simultaneously (7, 8). The clinical presentation of TTC can mimic acute myocardial infarction with similar electrocardiographic changes, troponin elevation, and acute left ventricular dysfunction, making immediate differentiation crucial for appropriate management decisions. Diagnostic criteria include new ECG abnormalities, mild-to-moderate elevation of cardiac troponins, and most importantly, the absence of obstructive coronary disease on angiography (9). Unless high clinical suspicion is maintained, particularly in atypical presentations, the diagnosis can be overlooked, and patients may be treated inappropriately according to acute coronary syndrome protocols (10). Case reports of TTC in military personnel during active combat remain extremely limited in the literature. We report a case of severe stress-induced cardiomyopathy in a young soldier during active combat, emphasizing the diagnostic challenges and clinical importance of considering TTC in military personnel presenting with acute coronary syndrome, particularly in high-stress combat environments.

Case Presentation

A 21-year-old male soldier developed acute cardiovascular collapse during active military service in a combat zone. The patient had been engaged in continuous military operations for 12 days under extreme psychological stress, with prolonged sleep deprivation and extended duty shifts. His past medical history was unremarkable except for remote leg surgery, with no documented cardiovascular risk factors or family history of cardiac disease. At approximately 07:00 hours during military service, the patient experienced an acute onset of dyspnea followed by loss of consciousness. Following his transfer to a local medical facility, he suffered cardiac arrest. Cardiopulmonary resuscitation (CPR) was performed for 90 minutes until spontaneous circulation was restored. Following intubation, he was transferred to a tertiary cardiac Centre with a preliminary diagnosis of extensive anterior ST-elevation myocardial infarction based on electrocardiographic findings.Initial vital signs at the first medical Centre following successful resuscitation demonstrated:

• Blood pressure: 106/70 mmHg

• Evidence of hemodynamic instability requiring intensive support

Upon arrival at the tertiary Centre at 11:00 hours, vital signs were:

• Blood pressure: 100/70 mmHg

• Heart rate: 82 beats per minute

• Oxygen saturation: 98% on mechanical ventilation

The patient remained unconscious and unresponsive to painful stimuli. He received loading doses of aspirin and antiplatelet agents before transfer to the cardiac catheterization laboratory.

Diagnostic Investigations

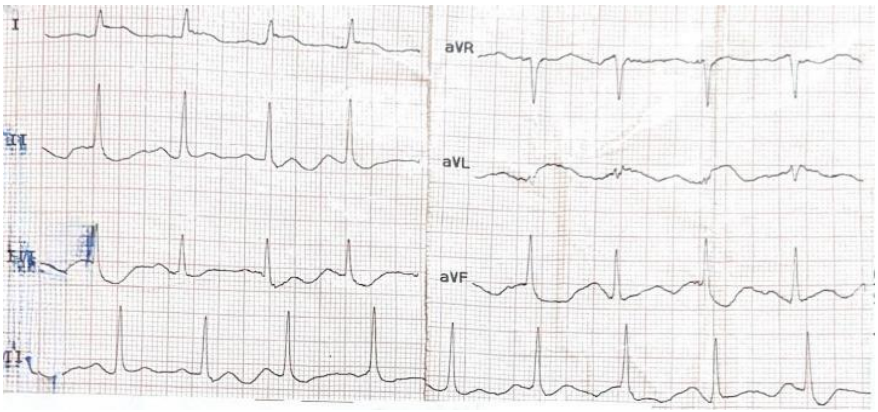
Initial electrocardiogram demonstrated Fig 1:

• ST-elevation in precordial leads V2-V6

• ST-elevation in limb leads I and aVL

• Reciprocal ST-depressions in inferior leads II, III, and aVF

• Pattern consistent with extensive anterior wall ST-elevation myocardial infarction



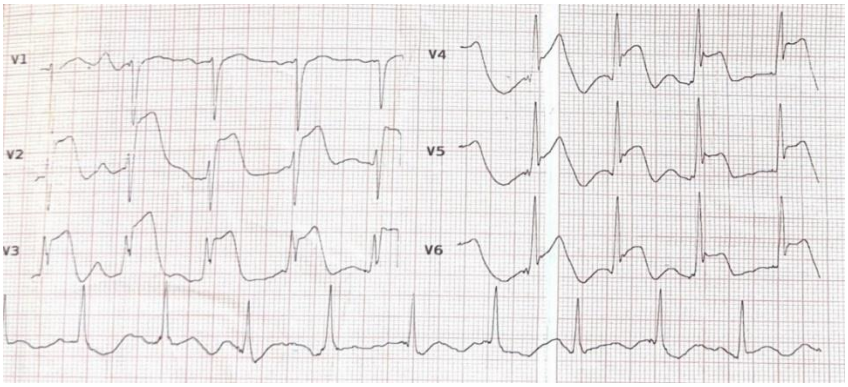


Figure 1. Initial electrocardiogram showing pattern consistent with extensive anterior wall ST-Elevation of myocardial infarction

Laboratory Results

Table 1. Key Laboratory Results

Parameter	Day 1 Result	Day 2 Result	Reference Range	Clinical Significance
Cardiac Biomarkers				
Troponin I	7.6 mg/mL	-	0-1.0 mg/mL	Severely elevated
CK-MB	61 U/L	-	0-25 U/L	Elevated
CPK (Total)	-	5,020 U/L	30-200 U/L	Severely elevated
Key Metabolic Markers				
Glucose (initial)	379 mg/dL	-	70-110 mg/dL	Severely elevated
Potassium	3.5 mEq/L	-	3.5-5.0 mEq/L	Low normal
Magnesium	1.0 mg/dL	-	1.7-2.2 mg/dL	Low

Coronary Angiography

Primary coronary angiography performed on admission day revealed completely normal epicardial coronary arteries with no evidence of stenosis, occlusion, dissection, or other structural abnormalities. This finding prompted reassessment of the initial diagnosis Fig 2.

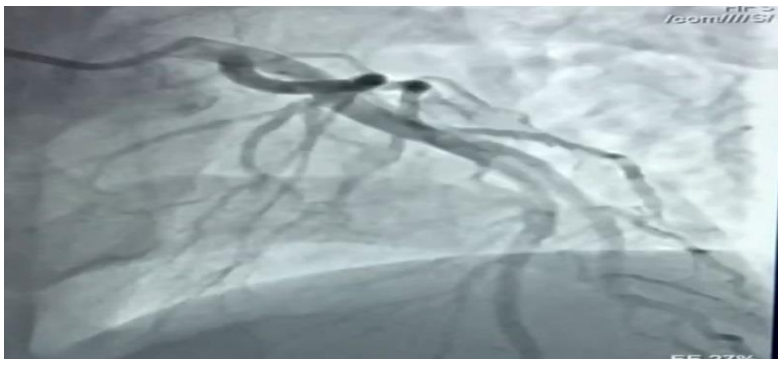


Figure 2. Coronary angiogram showing completely normal epicardial coronary arteries

Echocardiographic Assessment

Initial echocardiogram

Normal left ventricular dimensions with reduced systolic function (estimated ejection fraction 35%). Regional wall motion abnormalities were observed, predominantly affecting apical and mid-ventricular segments. There was no evidence of pericardial effusion or significant valvular abnormalities Fig 3.

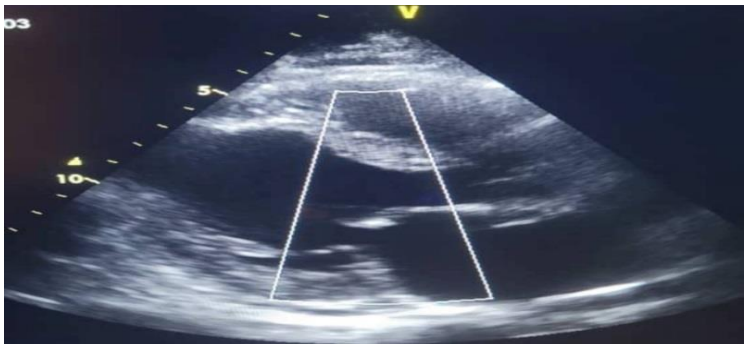


Figure 3. First echocardiogram showed normal left ventricular dimensions with reduced Systolic function. Regional wall motion abnormalities were seen, mostly in apical and Mid-ventricular segments

Follow-up echocardiogram (Day 3)

Complete normalization of left ventricular systolic function (ejection fraction approximately 50%) with resolution of all regional wall motion abnormalities. Left ventricular dimensions remained normal Fig 4.

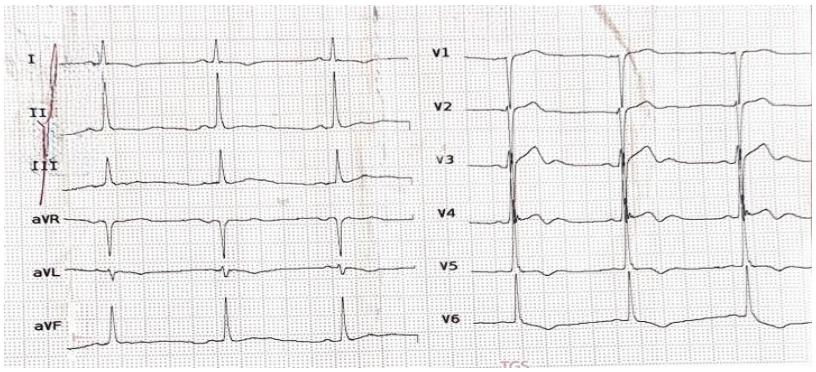


Figure 4. ECG on third day of admission showed dynamic evolution of the previously observed ST-segment changes, with gradual normalization of the anterior lead abnormalities

Neurological Evaluation

Brain computed tomography performed without contrast enhancement was normal with no evidence of acute intracranial pathology. A neurology examination revealed mid-sized reactive pupils with no focal deficits initially. However, the patient subsequently developed seizure activity requiring anticonvulsant therapy with phenytoin and diazepam. A neurology consultation was obtained for ongoing management.

Clinical Course and Management

Following the unexpected finding of normal coronary arteries, the diagnosis was altered to stress-induced cardiomyopathy. Accordingly, the management transitioned from acute coronary

syndrome protocols to comprehensive supportive care. During the initial forty-eight hours, the patient remained mechanically ventilated and unconscious in the coronary care unit, requiring intensive hemodynamic monitoring and support. Given the confirmed normal coronary anatomy on angiography, dual antiplatelet therapy was stopped, since the primary indication for antiplatelet treatment, prevention of coronary thrombosis, was no longer applicable in the absence of coronary artery disease. Concurrently, significant metabolic abnormalities were addressed, including the management of severe hyperglycemia and correction of electrolyte imbalances, particularly hypokalemia and hypomagnesaemia. By the third day of admission, remarkable neurological improvement became evident as the patient demonstrated spontaneous eye opening and progressively improved responsiveness to verbal and tactile stimuli. Electrocardiographic monitoring revealed dynamic evolution of the previously observed ST-segment changes, with gradual normalization of the anterior lead abnormalities Fig. 4. Most significantly, repeat echocardiography performed on this day demonstrated complete recovery of left ventricular systolic function, with resolution of all previously noted regional wall motion abnormalities. The fourth day marked a crucial turning point as the patient achieved full consciousness and complete orientation to person, place, and time. His hemodynamic parameters remained stable throughout this period, which allowed for safe transfer from the intensive coronary care unit environment to the general medical ward for continued monitoring and rehabilitation. During the fifth day of hospitalization, the patient experienced complete clinical recovery with a notable absence of chest pain, dyspnea, or other cardiac symptoms. He reported feeling subjectively well and demonstrated excellent functional capacity during bedside assessments, indicating readiness for discharge planning and outpatient follow-up arrangements. On the sixth day following admission, the patient was successfully discharged to his home environment in excellent clinical condition, demonstrating complete normalization of both cardiac and neurological function, which exemplified the characteristic reversible nature of stress-induced cardiomyopathy when appropriately recognized and managed.

Discharge Management and Follow-up

• The patient's discharge medication regimen was carefully designed to provide cardiovascular protection and address ongoing metabolic concerns. The medications included:

• Pantoprazole 40 mg daily for gastric protection,

• Empagliflozin 10 mg daily for glucose management, targeting persistent mild hyperglycemia,

• Aspirin 80 mg daily for cardiovascular protection,

• Bisoprolol 2.5 mg daily for beta-adrenergic blockade,

• Atorvastatin 40 mg daily for comprehensive lipid management. Initially, beta-blocker therapy was used due to its potential to counteract excessive catecholamine stimulation, which is a contributing factor to TTC pathophysiology. Recent evidence suggests that beta-blocker therapy may be associated with lower follow-up mortality and recurrence rates in TTC patients, with more benefits than long-term treatment (11). This is particularly relevant for military personnel who may face repeated high-stress situations. Statin therapy serves multiple purposes beyond traditional lipid management, including anti-inflammatory properties that may mitigate catecholamine-induced inflammatory cascades and endothelial stabilizing effects relevant to TTC pathophysiology (12). However, evidence for preventing TTC recurrence remains limited, emphasizing the need for individualized therapy based on patient-specific risk factors. Comprehensive outpatient cardiology follow-up was scheduled for two weeks post-discharge to assess continued recovery and optimize medical treatment based on clinical response and any residual symptoms as the patient resumes regular activities.

Discussion

This case illustrates several important clinical and educational points regarding TTC in young males, particularly in military settings. Our patient's presentation with cardiac arrest and severely elevated biomarkers (troponin I 7.6 mg/mL, total CK 5,020 U/L) demonstrates that TTC can produce clinical and biochemical findings indistinguishable from massive myocardial infarction. This case presents several distinctive features that differentiate it from typical TTC presentations reported in the literature. Unlike the classical demographic of postmenopausal

women, our patient represents the minority male population affected by TTC, with the additional rarity of being a young adult under extreme combat stress. The patient's survival is particularly significant given that the International Takotsubo Registry showed male patients have up to three-fold higher risk of in-hospital mortality (13). The severity of presentation with cardiac arrest requiring 90 minutes of CPR is exceptionally uncommon in TTC literature, where most cases present with chest pain without hemodynamic collapse (14). Physical triggers, acute neurologic complications, initial troponin levels exceeding 10 times the upper reference limit, and admission left ventricular ejection fraction below 45% are all predictors of poor in-hospital outcomes—all of which were present in our case (15). The elevated cardiac biomarkers represent values rarely reported in TTC literature, where troponin elevation is typically modest. Most published TTC cases report troponin values well below our patient's levels, making this presentation among the most severe biochemically documented cases in young males. The incidence of TTC was dramatically exacerbated by social isolation, financial stress, and psychological worry during the COVID-19 epidemic, as evidenced by recent research (16). However, the intensity of combat stress surpasses typical civilian stressors by orders of magnitude. The military context adds extraordinary uniqueness, as published cases of TTC in active military personnel during combat operations remain virtually absent from the literature. The intensity of combat stress surpasses typical civilian stressors by orders of magnitude. Most military-related TTC cases reported involve veterans with post-traumatic stress disorder rather than active-duty personnel during combat deployment. Our patient experienced a constellation of military-specific risk factors: extreme psychological stress from direct combat exposure, sleep deprivation during 12 days of continuous operations, physical exhaustion, acute life-threatening situations, and separation from family support systems. This combination created an optimal environment for a massive catecholamine surge and subsequent severe cardiac dysfunction. The initial approach with primary percutaneous coronary intervention was appropriate given the presenting scenario. However, the discovery of normal coronary arteries necessitated a rapid transition to supportive care. The decision to discontinue dual antiplatelet therapy was made due to the absence of coronary lesions and current evidence suggesting DAPT should be discontinued at discharge when TTC is confirmed. The development of neurological complications, including seizures and prolonged unconsciousness, likely resulted from the combination of cardiac arrest, cardiogenic shock, and hypoxic-ischemic injury. The parallel recovery of cardiac and neurological function demonstrates the importance of aggressive supportive care during the acute phase. The complete recovery pattern, while characteristic of TTC, occurred remarkably rapidly despite the severity of initial presentation. Cardiac function returned to the normal state within three days, representing faster recovery than many reported cases, rather than improvement in weeks to months. This rapid recovery exemplifies the dramatic reversibility possible in stress-induced cardiomyopathy, even after life-threatening presentations. However, recent evidence suggests that despite apparent complete recovery, patients may exhibit persistent long-term structural, functional, and metabolic abnormalities more than a year after the acute episode (12). For military personnel, this has important implications regarding fitness for duty assessments and ongoing cardiovascular monitoring, particularly given their continued exposure to high-stress environments. This case emphasizes the critical importance of maintaining high clinical suspicion for TTC in young military personnel presenting with acute coronary syndrome, especially in combat settings. The combination of extreme stress, common coronary angiography, and a characteristic recovery pattern confirms the diagnosis. Military healthcare providers should consider TTC in personnel exposed to combat stress who develop ACS-like symptoms, as early recognition is essential for appropriate management and optimal outcomes.

Conclusion

We presented a case of severe stress-induced cardiomyopathy, also known as Takotsubo cardiomyopathy (TTC), in a young soldier who experienced combat stress. This case demonstrated that TTC can appear as a life-threatening cardiac arrest, resembling a massive anterior ST-Elevation Myocardial Infarction (STEMI). The diagnosis was confirmed through a

combination of extreme military stress, usual coronary angiography, and the patient's full cardiac recovery. Early diagnosis is crucial for the appropriate management and optimization of outcomes. Military healthcare providers should take TTC into account in personnel exposed to combat stress and who have developed ACS-like symptoms. The proper prognosis demonstrated by complete cardiac and neurological recovery emphasizes the reversible nature of TTC when properly recognized and managed, even following severe presentations necessitating prolonged resuscitation.

Ethical Statement

This case report was conducted in accordance with ethical principles for medical case reports. Verbal informed consent for publication was obtained from the patient and documented in his medical file.
Ethics approval ID: IR.UMSU.REC.1404.434.

Acknowledgements

The authors would like to express their sincere appreciation to Dr.A. Ghanivash, cardiologist at Piranshahr Hospital, along with all the medical team in the Accident & Emergency department there, for their lifesaving efforts during the critical first ninety minutes of cardiopulmonary resuscitation and for facilitating the successful transfer of this young soldier to our facility. We extend our gratitude to the medical staff and administration of Seyed-Al-Shohada Hospital, Urmia, Iran, for their assistance throughout this case. We would especially appreciate the cardiology department, cardiac catheterization laboratory team, and coronary care unit nursing staff. We thank the patient for providing informed consent for publication and acknowledge the cooperation between the two medical teams that was essential to achieve the best possible patient outcomes in this challenging clinical scenario.

Authors Contribution

Dr Negar Jafari: The treating physician, case conceptualization, gathering data, clinical data interpretation, manuscript review and approval
Dr Venus Shahabi: Literature review, manuscript writing and editing, submission and revision management

Conflicts of Interest

The authors declare no conflicting interests related to this case report.

Funding

N/A

References

1. Ghadri JR, Wittstein IS, Prasad A, et al. International Expert Consensus Document on Takotsubo Syndrome (Part I): Clinical Characteristics, Diagnostic Criteria, and Pathophysiology. *Eur Heart J*. 2018; 39(22):2032-2046.
2. Movahed MR, Javanmardi E, Hashemzadeh M. High Mortality and Complications in Patients Admitted With Takotsubo Cardiomyopathy With More Than Double Mortality in Men Without Improvement in Outcome Over the Years. *J Am Heart Assoc*. 2025; 14(10):e037219.
3. Singh T, Khan H, Gamble DT, et al. Takotsubo Syndrome: Pathophysiology, Emerging Concepts, and Clinical Implications. *Circulation*. 2022; 145(13):1002-1019.
4. Daralammouri Y, Hamayel H, Abugaber D, et al. Takotsubo Cardiomyopathy: Patients' Characteristics, Mortality, and Clinical Significance of Left Ventricular Outflow Tract Gradient, Retrospective Study. *Cardiol Res Pract*. 2024; 2024:5549795.
5. Fernandez CA, Narveson JR, Walters RW, et al. Takotsubo Cardiomyopathy and Trauma: The Role of Injuries as Physical Stressors. *Cureus*. 2022; 14(7):e27411.
6. Bakshi I, Rawat HS. Exploring the role of psychological stress in Takotsubo cardiomyopathy, a.k.a broken-heart syndrome. *Int J Multidiscip Res*. 2024; 6(1):e14410.
7. Chu B, Marwaha K, Sanvictores T, et al. Physiology, Stress Reaction. In: StatPearls [Internet]. *Treasure Island (FL): StatPearls Publishing* 2025.
8. Singh T, Khan H, Gamble DT, et al. Takotsubo syndrome: pathophysiology, emerging concepts, and clinical implications. *Circulation*. 2022; 145(13):1002-1019.
9. Medina de Chazal H, Del Buono M, Keyser-Marcus L, et al. Stress Cardiomyopathy Diagnosis and Treatment: JACC State-of-the-Art Review. *J Am Coll Cardiol*. 2018; 72(16):1955-1971.
10. Scafa-Udriste A, Horodinschi RN, Babos M, et al. Diagnostic challenges between takotsubo cardiomyopathy and acute myocardial infarction-where is the emergency?: a literature review. *Int J Emerg Med*. 2024; 17(1):22.
11. Rodriguez Mejia RA, Singh A, Bahekar A, et al. Efficacy of beta-blocker therapy in Takotsubo cardiomyopathy: A systematic review and meta-analysis. *Int J Cardiol*. 2025; 437:133483.
12. Scally C, Rudd A, Mezincescu A, et al. Persistent Long-

Term Structural, Functional, and Metabolic Changes After Stress-Induced (Takotsubo) Cardiomyopathy. *Circulation*. 2018; 137(10):1039-1048.
13. Templin C, Ghadri JR, Diekmann J, et al. Clinical Features and Outcomes of Takotsubo (Stress) Cardiomyopathy. *N Engl J Med*. 2015; 373(10):929-938.
14. Glaveckaitė S, Šerpytis P, Pečiūraitė D, et al. Clinical features and three-year outcomes of Takotsubo (stress) cardiomyopathy: Observational data from one center. *Hellenic J Cardiol*. 2016; 57(6):428-434.
15. Santoro F, Núñez Gil IJ, Stiermaier T, et al. Assessment of the German and Italian Stress Cardiomyopathy Score for Risk Stratification for In-hospital Complications in Patients With Takotsubo Syndrome. *JAMA Cardiol*. 2019; 4(9):892-899.
16. Almani MU, Khan R, Qudrat-Ullah M, et al. Gender disparities and predictors of in-hospital mortality with Takotsubo cardiomyopathy. *Int J Cardiol*. 2024; 405:131959.

