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Review Article

Others Section

Takotsubo Cardiomyopathy in Myasthenia Gravis-emergency Recognition and Management of a Life-threatening Overlap: A Narrative Review

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ABSTRACT

Takotsubo Cardiomyopathy (TTC), also known as stress cardiomyopathy or broken heart syndrome, is a condition where temporary left ventricular dysfunction occur. This often happens because of emotional or physical stress. Myasthenia Gravis (MG) is an autoimmune disorder that affects the neuromuscular junction, causing fluctuating muscle weakness. Co-occurrence of these two conditions is rare. However, clinicians must distinguish between them, especially in an emergency. The present narrative review examines the unusual association between TTC and MG. This review focuses on rapid recognition in emergencies, the difficulties in diagnosing it, and how to manage this potentially fatal condition. The present review thoroughly examines existing literature, including case reports and systematic reviews. The authors examined articles discussing the co-occurrence of TTC and MG. The authors also examined their biological mechanisms, clinical presentation, diagnostic approaches, and treatment strategies. They thoroughly examined peer-reviewed journals from 2005 to 2025. The present analysis indicates that distinguishing between TTC and MG is difficult when both are present simultaneously. Key challenges include differentiating TTC from Myasthenic Crisis (MC) and determining the optimal management of acute cardiac complications in patients with MG. Providing supportive care and appropriate treatment is essential to helping patients recover from such emergencies. The discussion highlights the need to identify TTC early and to appropriately manage it in patients with MG, who are at risk of such emergencies. Other studies will strengthen clinical guidelines, empowering doctors to manage rare but deadly overlap disease cases more effectively.

Keywords: Autoimmune diseases, Broken heart syndrome, Coronary vasospasm

INTRODUCTION

Takotsubo Cardiomyopathy (TTC), also known as stress cardiomyopathy or broken heart syndrome is an acute and typically reversible left ventricular systolic dysfunction that occurs in response to an emotional or physical stressor [1]. It is clinically similar to Acute Coronary Syndrome (ACS), characterised by chest pain, electrocardiographic alterations and cardiac biomarker elevation. Nonetheless, TTC is not associated with the presence of obstructive coronary artery disease in angiography, which distinguishes this disease from Myocardial Infarction (MI) [2]. TTC is usually self-limiting, but severe complications such as heart failure, arrhythmias, thromboembolism, and death (in some isolated cases) can be related to TTC. The syndrome occurs predominantly in postmenopausal women, but can also affect both sexes across a broad age range and is distributed in a broad age group, especially following major stressors [3,4].

As a pathophysiological mechanism, TTC is closely associated with hypercatecholamine release. It is proposed that acute bursts of stress hormones like adrenaline may cause myocardial stunning by causing coronary vasospasm, microvascular dysfunction or direct catecholamine-mediated myocardial toxicity [5]. This causes temporary ventricular dysfunction, which can manifest as the typical apical ballooning on echocardiography or ventriculography. Recovery of ventricular function occurs in most patients within a few weeks. However, severe conditions can result in long-term morbidity or mortality [6].

The MG is a long-term autoimmune disease of the neuromuscular junction characterised by intermittent skeletal muscle weakness caused by autoantibodies targeting acetylcholine receptors or related proteins. Exacerbations, which can be caused by infections, drugs, or physiological stress, often characterise the disease course [7]. The worst form of MG, known as a MC, occurs when respiratory failure due to respiratory muscle weakness needs intensive care intervention before it can be managed. Although there has been therapeutic success, MC continues to be a significant factor in morbidity and mortality of MG [8].

In addition to their respective clinical courses, there are also significant interrelationships between TTC and MG that make it challenging to manage the patients. MC is a severe physiological stress disorder with the capacity to produce severe catecholamine release and induce TTC in vulnerable patients [9]. On the other hand, drugs employed in the treatment of MG have been found to cause isolated incidences of coronary vasospasm, which may also lead to cardiac complications, especially acetylcholinesterase inhibitors like pyridostigmine [10]. These overlapping physiological and pharmacological mechanisms create a complex clinical situation in which both neurological and cardiac stress reactions overlap.

The TTC and MG co-exist extremely rarely, with approximately 40-50 reported cases as of recent literature reviews [11]. This frequency probably drives their two primary mechanisms: TTC (stress-induced and cardiac-based) and MG (autoimmune and neuromuscular). However, their overlap has clinical significance [12]. Both of these disorders can be characterised by dyspnoea, fatigue, and chest pain, and they can both exhibit electrocardiographic alterations and increased cardiac biomarkers. These similarities increase the risk of misdiagnosis, delayed diagnosis, and poor management [13].

It is thus important to be accurate in differentiation. Due to the similarity of troponin increase and ECG alterations to MI, the lack of obstructive coronary disease and typical ventricular motion abnormalities, a diagnosis of TTC is made. Echocardiography and cardiac Magnetic Resonance Imaging (MRI) are the most important

cardiac imaging modalities for verification. However, in some emergency departments, they may not be available [14,15].

When the two conditions are used simultaneously, it becomes tough to manage. The priority in MC is stabilisation of respiratory dysfunction, which may be accompanied by ventilatory support and immunomodulatory treatment with corticosteroids, Intravenous Immunoglobulin (IVIG), or plasmapheresis [16,17]. Once respiratory stability is achieved, management of TTC focuses on supporting cardiac care, which often involves beta-blockers and Angiotensin-Converting Enzyme (ACE) inhibitors. There should be individualisation of therapeutic decisions, as certain cardiovascular medications can affect neuromuscular transmission and levels of MG. These complexities underscore the need for multidisciplinary care coordination [18,19].

As it is a complex diagnosis, therapeutic dilemma with potentially life-threatening consequences, the intersection of TTC and MG is a clinically significant but under-identified phenomenon [20].

The purpose of the present narrative review is to summarise existing data on the comorbidity between TTC and MG, identify issues in diagnosing TTC in emergency cases, discuss possible pathophysiological correlations, and outline management approaches and therapies. It also aims to identify gaps in the existing literature and to provide priorities for future research.

Through these areas, the present review will contribute to clinical awareness and an evidence-based approach to the timely management of this rare yet potentially fatal overlap syndrome.

LITERATURE REVIEW

Battineni A et al., reported a recurrent episode of TTC during a MC, underscoring the clinical importance of considering TTC as a possible complication during MG exacerbations [21]. Although this case provided valuable clinical insight, it is constrained by its single-patient nature, which limits generalisability, and frequency and risk factors cannot be estimated. Rathish D and Karalliyadda M conducted a systematic review of reported cases of Takotsubo syndrome in patients with MG. They highlighted the difficulties in diagnosing Takotsubo syndrome and the importance of early identification [11]. Even though their work summarised current reports, it mainly relied on previously published case reports, which are subject to publication bias and inconsistent reporting criteria. This minimises the capacity to reach firm clinical conclusions. Hasan SM et al., addressed variants of TTC. They expanded knowledge of its clinical manifestations, including the scenario that applies to MG patients [22]. Nonetheless, the research did not specifically target the MG-TTC overlap, which restricts its relevance to the given subgroup. Chuapakdee O et al., reported that pyridostigmine-induced coronary vasospasm in a patient with MG exists, suggesting a potential pharmacological link between MG treatment and Takotsubo-like syndromes [23]. Although this raises significant safety concerns, such evidence is based on case studies and cannot establish causation or incidence. The diagnosis and treatment of Takotsubo syndrome in emergency departments were reviewed, and the clinically relevant findings were presented by Assad J et al., [24]. However, the review was conducted in a general manner. It lacked MG-specific management approaches, which created uncertainty in cases of overlap. Farjoud Kouhanjani M et al., analysed TTC in autoimmune diseases. They recommended that it is more common in the autoimmune population, including MG [25]. Nonetheless, the diverse nature of autoimmune diseases and the lack of subgroup analysis of MG make direct extrapolation challenging.

All these studies show the increasing interest in the TTC-MG overlap but also indicate gaps in the evidence. The majority of existing data is based on case reports or small series, which limits statistical power and prevents the creation of standardised diagnostic or treatment guidelines. The heterogeneity in diagnostic strategies, inadequate follow-up, and inconsistent reporting also prevent the synthesis of evidence.

Such restrictions have a direct impact on clinical management. Without extensive prospective studies and evidence-based guidelines, clinicians are left to their own judgment and multidisciplinary teamwork when faced with suspected cases of overlap. Such ambiguity can contribute to late diagnosis, inconsistent treatment plans, and inconsistent patient outcomes.

In general, the literature highlights the importance of increasing the size of studies, systematic research, and standardised reporting on the prevalence, mechanisms, and optimal management of this little-known but clinically important overlap, as shown in [Table/Fig-1] [11,21-25].

The present narrative review summarises the existing evidence on the presence of TTC and MG, the present study was conducted as a narrative review. A narrative design was selected to allow a clinically oriented review of diagnostic, pathophysiological, and therapeutic factors. There was no meta-analysis because the literature consists mainly of case reports and small, heterogeneous studies, which cannot be meaningfully synthesised quantitatively.

Literature Search Strategy

Relevant studies on the topic published in 2005 and 2025 were identified through a literature search of PubMed and Google Scholar. The search was conducted iteratively to fully cover the literature on this uncommon clinical overlap fully.

The search terms used were: “TTC” AND “Myasthenia Gravis”; “Takotsubo syndrome” AND “myasthenic crisis”; and “Takotsubo” AND “neuromuscular disorders”. Manual screening of the reference lists of relevant articles was conducted to identify additional sources.

Authors (Year)	Key finding	Research focus	Challenges/Limitations	Future scope	Article type
Battineni A et al., (2017) [21]	Recurrent Takotsubo in Myasthenic Crisis (MC)	Case report on Takotsubo during Myasthenia crisis	Case-specific, limited generalisability	Further studies on the overlap with other autoimmune diseases	Case report
Rathish D and Karalliyadda M (2019) [11]	Takotsubo syndrome in MG is rare but life-threatening	Systematic Review on Takotsubo in MG	Limited data on triggers and predisposing factors in MG	Research on triggers and prevention of recurrence	Systematic review
Hasan SM, (2020) [22]	Variants of Takotsubo complicate diagnosis.	Review of Takotsubo Cardiomyopathy (TTC) variants	Focused on variants, not MG overlap	Studies on MG-specific presentations of Takotsubo	Literature review
Chuapakdee O et al., (2022) [23]	Pyridostigmine-induced coronary spasm in MG	Investigating drug-induced cardiac events in MG	Case-specific, focused on pyridostigmine	Investigating pyridostigmine’s broader cardiovascular effects in MG	Case report and review
Assad J et al., (2022) [24]	Comprehensive Review of Takotsubo Syndrome	Review of Takotsubo syndrome diagnosis and management	Limited focus on MG overlap	Studies on MG-specific Takotsubo management	Review article
Farjoud Kouhanjani M et al., (2024) [25]	Takotsubo syndrome is more common in autoimmune disorders	Review of Takotsubo in autoimmune conditions	Limited data specific to MG	Research on the pathophysiology of Takotsubo in autoimmune diseases	Systematic review

[Table/Fig-1]: Literature review [11,21-25].
This table consolidates results, problems, and potential areas of focus from past studies on Takotsubo syndrome and MG Broken heart syndrome, Coronary vasospasm

Locating Data

The first filter was to screen studies based on titles and abstracts. Articles that covered TTC-MG co-existence, diagnostic, or management issues were reviewed in full.

Inclusion and Exclusion criteria: The inclusion criteria were human studies published in English that involved case reports, case series, reviews, or clinical studies discussing TTC, MG, or their combination. Animal research, non English articles, and articles that did not cover both conditions were eliminated.

Data Extraction

Essential information was retrieved on patient demographics, clinical presentation, diagnostic results, suggested mechanisms, management plans, and outcomes. Emphasis was placed on triggers, pathophysiology, and treatment methods.

Data Synthesis

The rarity and heterogeneity of the TTC-MG overlap led to a qualitative thematic analysis. The results were systematised into themes of clinical presentation, diagnostic issues, pathophysiology, and management, and identified recurrent patterns and recognised variability.

Prevalence and clinical manifestations of Takotsubo Cardiomyopathy (TTC) in Myasthenia Gravis (MG) Patients

The TTC among patients with MG is extremely rare. However, it is gaining increased recognition as a dangerous overlap syndrome. Prevalence of TTC in MG patients has been documented as low but present by studies, with MG exacerbations are frequently the precipitating factors for stress-induced myocardial dysfunction [26]. The condition most frequently occurs in postmenopausal women, who also tend to be the typical demographic experiencing MG as well as Takotsubo syndrome. The clinical picture becomes perplexing when both are present simultaneously, given the similarity of symptoms such as chest discomfort, shortness of breath, and fatigue [27]. Unlike ACS, which usually involves obstructive coronary artery disease, TTC in MG patients is characterised by the absence of coronary obstruction and the presence of transient left ventricular dysfunction that mimics ACS [28].

This potential overlap between MG and TTC may be confounded by medications used by MG patients (e.g., pyridostigmine), enhanced sympathetic nervous activity, and emotional/stressful stimuli that frequently coincide with MG crises [29]. Classic symptoms of both MG and TTC may be present in patients, such as weakness, respiratory distress, arrhythmias, and the classic TTC feature of apical ballooning. TTC development in MG patients has been identified by several risk factors, such as older age, female gender, and a history of an acute MG exacerbation/severe crisis. Early diagnosis is essential, as otherwise cases may proceed towards serious complications such as heart failure and arrhythmias, as depicted in [Table/Fig-2] [30].

These results have some significant implications for emergency management. Clinicians in MG patients with acute respiratory distress or chest symptoms must have a high level of suspicion of TTC, especially in postmenopausal women or in MC. Bedside echocardiography, early electrocardiography, and cardiac biomarker assessment can help differentiate between TTC and ACSs. Early detection enables prioritising respiratory stabilisation and introducing appropriate cardiac monitoring and supportive therapy. This overlap can be minimised through awareness, reducing misdiagnosis and enhancing acute care outcomes as shown in [Table/Fig-3] [11,26-30].

Database	Keyword and operator combination	No. of articles hit	Articles included
PubMed	"Takotsubo Cardiomyopathy (TTC)" AND "Myasthenia Gravis"	49	31
PubMed	"Takotsubo syndrome" AND "myasthenic crisis"	34	19
Google Scholar	"Takotsubo" AND "neuromuscular disorders"	1,480 (top 100 screened)	28
Google Scholar	"Takotsubo syndrome" AND "Myasthenia Gravis"	818 (top 120 screened)	22
Total before deduplication	—	2,381	100
After deduplication (Zotero; 34 duplicates removed)	—	—	66 finalised

[Table/Fig-2]: Search strategy.

Pathophysiology: Mechanisms Behind Takotsubo Syndrome in the Context of Myasthenia Gravis (MG)

The pathophysiology of TTC in patients with MG involves a complex interplay among neuromuscular, autonomic, and cardiovascular systems. The acute catecholamine surges are the leading cause of TTC and are often provoked by extreme physical or emotional pressure. Respiratory muscle weakness and hypoxia cause an extreme activation of the sympathetic nervous system in MG, especially during MC, which results in the oversecretion of stress hormones, like adrenaline and norepinephrine [31]. Such sympathetic hyperactivity provides a plausible mechanistic link between MG and TTC. High concentrations of catecholamine may cause coronary vasospasm, endothelial dysfunction and microvascular damage that cause myocardial stunning and the typical apical ballooning of TTC [32]. In this way, the physiological stress that characterises MG attacks can trigger mechanisms underlying TTC development. Moreover, MG correlates with the immune dysregulation and systemic inflammation. Inflammatory mediators and oxidative stress can be

Author(s)	Nature of report	Key clinical features (TTC in MG context)	Reported triggers/risk factors	Reported complications
Rathish D and Karalliyadda M (2019) [11]	Systematic review	Dyspnoea (81%), chest pain (69%), fatigue; apical ballooning (75%)	Myasthenic crisis (MC); predominantly female (81%); mean age 62±15 yrs	Mortality 25%; mechanical ventilation 81%
Farjoud Kouhanjani M et al., (2024) [25]	Systematic scoping review	Chest pain (35%), dyspnoea (47%); median ejection fraction 40%	Physical stress (74%); predominantly female (84%); mean age 60 yrs	Mortality 8%; median recovery 19 days
Bubuioc AM et al., (2021) [26]	Review / case-based discussion	Dyspnoea, chest pain, and fatigue in MG patients with TTC	Female sex, older age, and MG exacerbation	Cardiac dysfunction
Tahir S et al., (2024) [27]	Case report	Acute Coronary Syndrome (ACS) -like presentation, chest discomfort, and fatigue	Severe MG crisis and emotional or physical stress	Heart failure and arrhythmias
Heckmann JM et al., (2022) [28]	MG epidemiology review	Describes MG phenotypes and epidemiology (not TTC-specific)	Age and sex variations in MG populations	Not TTC-focused
Baik AH et al., (2021) [29]	Case report	Respiratory distress, chest pain, and ECG changes	MG crisis, emotional stress, and advanced age	Arrhythmias and heart failure
Prokudina ES et al., (2021) [30]	Observational study	Apical ballooning and ST-segment elevation	Female sex and stress	Cardiogenic shock and pulmonary oedema

[Table/Fig-3]: Prevalence and clinical manifestations of Takotsubo Cardiomyopathy (TTC) in Myasthenia Gravis (MG) patients [11,26-30].
This table summarises the prevalence, clinical presentation, predisposing factors, and complications of TTC in patients with MG

the cause of dysfunction of the endothelium during exacerbations and myocardial vulnerability, which further predisposes patients to cardiomyopathy caused by stress [33]. Abnormal cardiac sympathetic activity may also result from an autonomic imbalance, as observed in some patients with MG. Pharmacological factors may reinforce this relationship. Acetylcholinesterase inhibitors, such as pyridostigmine, may affect autonomic tone. In an unusual incidence, they have been linked with coronary vasospasm. These effects can increase sympathetic-parasympathetic imbalance and make it more vulnerable to TTC. Combined, all this sympathetic hyperactivation, inflammatory stress, endothelial dysfunction, and autonomic imbalance imply a pathophysiological relationship between MG and TTC. These mechanisms are interrelated and support the reason why TTC can occur in severe MG exacerbations, as shown in [Table/Fig-4] [25,31-34].

Author(s)	Key Mechanism Described	Primary Condition Discussed	Relevance to TTC-MG Overlap
Farjoud Kouhanjani M et al., (2024) [25]	Catecholamine surge related to autoimmune stress and inflammation; microvascular spasm and catecholamine-induced cardiotoxicity	Takotsubo Cardiomyopathy (TTC) in autoimmune disorders (155 cases, including 29 MG)	Identifies MG as the most common autoimmune association (18.8%); MG crisis may precipitate catecholamine excess leading to TTC with marked EF reduction
Abraham M et al., (2022) [31]	Sympathetic dysregulation and catecholamine excess during autonomic stress	Autonomic dysfunction/stress cardiomyopathy	Suggests that autonomic instability during MG crisis may precipitate TTC
Culerrier J et al., (2024) [32]	Emotional and physiological stress trigger catecholamine surges and coronary vasospasm	Takotsubo syndrome	Supports stress-triggered TTC, relevant to MG crisis states
Shams Y (2021) [33]	Catecholamine toxicity causing myocardial stunning and microvascular dysfunction	Takotsubo Cardiomyopathy (TTC)	Provides a mechanistic basis for TTC development during stress
Tahsili-Fahadan P and Geocadin RG (2017) [34]	Brain-heart axis involvement and sympathetic hyperactivation in acute neurological stress	Neurogenic stress cardiomyopathy	Explains how neurological stress (e.g., MG crisis) may trigger TTC

[Table/Fig-4]: Proposed pathophysiological mechanisms linking Myasthenia Gravis (MG) and Takotsubo Cardiomyopathy (TTC) [25,31-34].
The table below compiles the pathophysiological features of Takotsubo syndrome with MG that indicate underlying mechanisms

Diagnostic Pitfalls: Takotsubo Cardiomyopathy (TTC) versus Myasthenic Crisis (MC)

In acute conditions, distinguishing between TTC and MC can be difficult because these conditions share symptoms such as dyspnoea, chest tightness, fatigue, and generalised weakness [35]. MC is often acute respiratory failure, caused by the weakness of the respiratory muscle tissue, and TTC, by acute heart dysfunction. Nonetheless, if both arise from physical or emotional stress, there can be significant overlap in clinical features [36]. Increased cardiac biomarkers, including troponins, can be seen in TTC and, sometimes, in critically ill MG patients, further complicating interpretation. This overlap may lead to mismanagement, so that TTC can be mistaken for isolated MG exacerbation or vice versa [37]. In an emergency, some clinical indicators would help differentiate.

The MC is characterised by predominant weakness of the bulbar or respiratory muscles, with intact cardiac imaging. However, TTC should be suspected at the onset of chest pain, ECG changes, or haemodynamic instability. In patients with MG and acute respiratory or chest symptoms, early ECG and serial troponin testing are advised. TTC can also imitate ACS because of chest pains and elevation of the ST-segments. TTC is rarely associated with obstructive coronary artery disease on angiography, unlike ACS [38]. Bedside echocardiography is an important tool in practical emergency practice, as it can quickly detect the typical apical ballooning sign and left ventricular dysfunction in TTC. This is a non invasive instrument that will be most useful in unstable patients. Cardiac MRI is indicated when the diagnosis is unclear, as it helps differentiate TTC from MI or myocarditis by showing myocardial oedema without infarction. MRI, however, is most limited to haemodynamically stable patients because of time and access constraints [39].

Medication history is also of significance, as medications (like pyridostigmine) can affect autonomic tone and can cause coronary vasospasm. The identification of these factors can help to sharpen clinical judgment.

In general, a multifaceted approach that considers clinical presentation, Electrocardiogram (ECG), cardiac biomarkers, and early echocardiography is the most feasible way to differentiate TTC from MC in an emergency setting, as summarised in [Table/Fig-5].

Therapeutic Interventions: Treatment of Takotsubo Cardiomyopathy (TTC) in MG Patients

The TTC management among MG patients requires a coordinated approach to address cardiac and neuromuscular instability. The priority in cases of MC is respiratory stabilisation, which can be achieved through ventilator support. The therapies required to manage neuromuscular worsening and reduce physiological stressors that could trigger TTC include standard MG crisis therapy, such as corticosteroids, IVIG, and plasmapheresis [40].

The cardiac therapy of TTC is mainly supportive. ACE inhibitors and beta-blockers are widely used to improve left ventricular function and reduce sympathetic hyperactivity. Beta-blockers, however, should be used cautiously in MG patients, as they can exacerbate neuromuscular weakness by affecting neuromuscular transmission.

Author(s) et al.,	Overlap symptoms	Elevated cardiac biomarkers	Differentiating from ACS	Role of cardiac imaging
Sánchez-Camacho A et al., (2025) [35]	Chest pain, weakness, fatigue, respiratory distress (similar to Myasthenic Crisis (MC)	Elevated troponin levels in both conditions	Takotsubo mimics ACS but lacks coronary obstruction	Echocardiography and MRI show apical ballooning in Takotsubo
De Perna ML et al., (2025) [36]	Symptoms overlapping with immune-related myocarditis and MG exacerbations	Elevated cardiac biomarkers such as troponins	Takotsubo must be distinguished from ACS and myocarditis	Cardiac MRI is essential for differentiating between Takotsubo and ACS
Singh T et al., (2022) [37]	Similarities in chest pain, dyspnoea, and fatigue with Myasthenic Crisis (MC)	Elevated biomarkers (troponin) in both MG and Takotsubo	Takotsubo syndrome lacks coronary blockage, unlike ACS	Imaging, including echocardiography, is necessary to assess myocardial dysfunction
Lyon AR et al., (2016) [38]	Weakness, fatigue, and chest pain are symptoms that overlap with MG crisis	Elevated troponin in both MG crisis and Takotsubo	Takotsubo differs from ACS because it lacks obstructive coronary disease.	Imaging, including echocardiography and MRI, is essential for diagnosis.
Gopalakrishnan P et al., (2017) [39]	Fatigue, chest pain, and dyspnoea similar to MG crisis	Elevated cardiac biomarkers (troponin) in Takotsubo and MG crisis	Distinguishing Takotsubo from ACS requires coronary angiography	Cardiac biomarkers and imaging techniques like MRI help in diagnosis

[Table/Fig-5]: Diagnostic challenges: differentiating Takotsubo Cardiomyopathy (TTC) from Myasthenic Crisis (MC) and other conditions.
This table summarises the diagnostic challenges in differentiating TTC from MC and other illnesses based on the available literature. Important factors include common symptoms, biomarker elevations, the need to differentiate from ACS, and the contribution of cardiac imaging methods [35-39].
ACS: Acute coronary syndrome; MG: Myasthenia gravis; MRI: Magnetic resonance imaging

Consequently, cardioselective agents or low doses should be proposed, where needed, under strict surveillance [41].

In this instance of overlap, ACE inhibitors are the preferred choice, as they are cardiac-beneficial and unlikely to have a significant effect on neuromuscular function. Diuretics can be prescribed for patients with heart failure to alleviate volume overload, and anticoagulation can be considered when the risk of a ventricular thrombus is present [42].

The MG medications also need close adjustment to be treated. Pyridostigmine has remained on the list of standard treatments for MG. However, overdosing can be seen to affect autonomic tone and has been infrequently linked to coronary vasospasm. Optimising the dose and conducting close clinical observation are consequently significant. Serial echocardiography and close monitoring of cardiac biomarkers should be used to assess ventricular function recovery. Given this complication, this overlap requires a multidisciplinary approach involving cardiologists, neurologists, and intensivists to balance treatment priorities and prevent therapies that exacerbate either of the aforementioned conditions [43].

Outcome and Prognosis of Takotsubo Cardiomyopathy (TTC) Patients with Myasthenia Gravis (MG)

The clinical presentations of TTC patients with MG depend on the interaction between the two disease entities and their treatments. TTC is frequently reversible with a good left ventricular function returning weeks to months after the crisis [44]. The prognosis of MG patients is defined by the severity of the MC and the timeliness of the intervention. Those patients who present early enough that both disease processes could be treated, i.e., respiratory support as well as judicious immunosuppression for MG, do relatively well. Early treatment and diagnosis are crucial for preventing the progression of both disease processes and secondary complications, such as heart or respiratory failure [45]. The prognosis of patients with MG and TTC is usually reliant on the frequency as well as severity, acute illness stage, myocardial damage degree, and the treatment response. While most patients with both diseases recover adequately, some patients experience recurrent TTC attacks upon the onset of subsequent MG crises or upon the onset of harsh emotional as well as bodily stress [46]. The occurrence of heart failure or arrhythmia in such patients is high, regardless of that, and they require continued monitoring of their cardiac system. Special consideration of neuromuscular and cardiac functions by interdisciplinary treatment guarantees optimal results, superior recovery, and higher total survival [47].

Gaps in Literature and Directions for Subsequent Studies

Though greater awareness of the similarities between TTC and MG has emerged, limited insight into the underlying pathophysiological mechanisms linking the two illnesses persists. Most of the literature comprises case reports and small studies, making it challenging to synthesise results and derive clear management principles and diagnostic criteria [48]. Because there are few multicenter studies on TTC, its prevalence is poorly defined, as are its underlying causes and long-term prognosis. In addition, the effects of drugs that are deployed for the treatment of MG on the development of Takotsubo syndrome need more study [49].

More research is needed to unravel the underlying mechanisms that place MG patients at higher risk of TTC development, including catecholamine involvement, autonomic failure, and inflammatory processes typical of MG crises. Randomised trials and extensive cohort analyses will also be needed to support evidence-based guidelines for diagnostic criteria and treatment of the overlap syndrome. More studies of the best management strategies, including pharmacological intervention and immunosuppressive therapy, could improve patient outcomes. Prospective studies of patients' prognosis through follow-up will identify predictors of recurrent disease and fine-tune more individualised care approaches [50].

DISCUSSION

The TTC and MG are separate disease entities. However, their comorbidity is a unique, infrequent, but clinically challenging problem. The two conditions share similar symptoms, such as dyspnoea, fatigue, and chest pain, making diagnosis and treatment complicated. Both disorders can be triggered by emotional or physical stress, leading to MC with the respiratory dysfunction in MG and catecholamine-driven myocardial dysfunction in TTC [51,52].

The distinction is especially challenging when cardiac biomarkers, such as troponins, are elevated, as they are often associated with MI. This overlap may blur the line between stress cardiomyopathy and MG exacerbation, resulting in competing treatment priorities. Although TTC is defined by the lack of obstructive coronary artery disease, MG crises tend to demand respiratory support and immunosuppressive treatment, which necessitates a close clinical assessment [53,54].

The TTC and MG have a pathophysiological relationship that is primarily mediated by stress. Surges of catecholamine can trigger myocardial stunning during MC, leading to the typical apical ballooning of TTC. In MG, autonomic dysfunction could also predispose to coronary vasospasm, microvascular dysfunction, and transient dysfunction of the myocardium [55].

Pharmacological conditions should also be well considered. Pyridostigmine, which is a staple of MG treatment, has been reported in isolated case reports to be associated with coronary vasospasm. Close observation and clinical attention are thus suggested in the management of patients with co-existing TTC and MG [56].

There are also diagnostic difficulties due to the similarity in the clinical and electrocardiographic manifestations. Chest pain, dyspnoea, and weakness, along with a change in the ECG such as ST-segment elevation, can resemble ACS. TTC, however, is characterised by the absence of coronary obstruction on angiography [57].

In that regard, cardiac imaging is crucial. Echocardiography is particularly useful for detecting the typical apical ballooning of the TTC, with cardiac MRI providing further confirmation and ruling out other possible diagnoses, such as MI or myocarditis [58].

Several published case reports demonstrate the practical clinical implications of the TTC-MG overlap and offer important lessons for clinicians. Beydoun SR et al., reported the case of a patient in whom MC as well as TTC was provoked by a combination of psychological and physiological stressors, which revealed that neuromuscular and cardiac decompensation can be induced simultaneously by stressful situations. The present case shows the significance of cardiac assessment in patients with MG who have a significant emotional stress or acute decline [59].

Douglas TM et al., cited a male patient with MC who presented with chest pain and electrocardiographic alterations that were suggestive of MI. Later, coronary angiography showed normal coronary arteries, and echocardiography confirmed TTC. This situation highlights the importance of TTC being able to resemble ACS and hence being misdiagnosed when cardiac imaging is not performed promptly [60].

Equally, Cuevas HH et al., reported on an elderly MG patient who was admitted to the intensive care unit, where he developed respiratory failure necessitating mechanical ventilation. The patient presented with ECG alterations and an increased level of cardiac biomarkers, which increased suspicion of ACS; nevertheless, the images showed TTC. As highlighted in this case, TTC is a suspected diagnosis in patients with MG when unexplained cardiac findings are observed during crisis events [61].

These clinical scenarios collectively point to several important practical implications. To start with, early cardiac assessment should be performed in MG patients who report chest pain,

dyspnoea proportionate to neuromuscular weakness, or ECG alterations. Second, bedside echocardiography may be a valuable and quick diagnostic tool in the emergency department. Third, early identification of TTC can help avoid unnecessary invasive procedures and inform further supportive management. This degree of awareness leads to earlier diagnosis of this overlap, safer treatment choices, and better patient outcomes.

The TTC in patients with MG is to be managed in a multidisciplinary approach. Respiratory stabilisation is a priority and may require mechanical ventilation in the presence of a MC. Plasmapheresis, IVIG, and corticosteroids are also necessary to treat MG exacerbations and decrease physiological stress that can trigger TTC [62].

Once respiratory stability is achieved, cardiac management becomes a priority. TTC therapy is primarily supportive, which consists of beta-blockers and ACE inhibitors. However, they should be used with care, as they can worsen neuromuscular weakness. In volume overload, diuretics can be suggested. It is essential to continuously monitor cardiac and neuromuscular conditions to prevent deterioration of either [63].

Although awareness of the coexistence of TTC and MG has increased, evidence remains sparse, consisting primarily of case reports and small studies. Consequently, they limit generalisability and lack formal clinical guidelines [64]. More studies are required to elucidate the contributions of catecholamine excretion, autonomic malfunction, and inflammation to the predisposition of MG patients to TTC. The association between MG therapies and TTC risk, especially with pyridostigmine, also needs to be better defined to provide safe treatment plans [65].

There are still significant gaps in the standardised diagnostic criteria for TTC in patients with MG. Due to the lack of specific symptoms and biomarkers, structured diagnostic algorithms are needed to account for the overlap between symptoms and ACS. Determination of disease-specific biomarkers may enhance diagnostic precision and inform treatment. It would require extensive cohort studies and randomised trials to develop evidence-based management guidelines that account for cardiac and neuromuscular aspects [66]. Follow-up studies must overcome limitations, narrow diagnostic tests, and refine personalised treatment guidelines.

Limitation(s)

Most of the available evidence is in the form of case reports and small case series, which constrains generalisability and renders them prone to publication bias. The differences in diagnostic criteria and reporting quality also complicate the synthesis of evidence. The conclusions were based on recurring patterns rather than isolated findings to reduce overinterpretation. The infrequency and heterogeneity of TTC-MG overlap limit the ability to draw concrete conclusions. However, they underscore the need for more systematic studies.

CONCLUSION(S)

The TTC overlapping with MG poses a significant diagnostic challenge and demands increased clinical vigilance. Typical clinical profiles of two disease processes are always present, and at least an acute threat of misdiagnosis is present. Stress-related catecholamine surges, autonomic dyscontrol, as well as vasospastic drugs' actions play an unavoidable pathophysiological part in the establishment of both diseases. Therapy requires an interdisciplinary approach with judicious consideration towards respiratory care and cardioprotection. The present continuation studies have clear-cut criteria for diagnosis and for developing treatment protocols.

Key takeaways: The relationship between TTC and MG is considered when two diseases present with overlapping symptoms. Early recognition and individualised management in emergency settings. Subsequent studies must consider pathophysiology, ideal treatment strategy, and patients' outcomes at the latter stage to fine-tune clinical guidelines.

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REFERENCES

[1] Sheppard MN. Takotsubo syndrome–stress-induced heart failure syndrome. Eur Cardiol Rev. 2015;10(2):83-88.
[2] Chaumont M, Blaimont M, Briki R, Unger P, Debbas N. Acute coronary syndrome mimicking Takotsubo cardiomyopathy or Takotsubo cardiomyopathy mimicking acute coronary syndrome? Case Rep Cardiol. 2020;2020:6562316.
[3] Wittstein IS. Why age matters in Takotsubo syndrome. J Am Coll Cardiol. 2020;75(16):1878-81.
[4] Pattisapu VK, Hao H, Liu Y, Nguyen TT, Hoang A, Bairey Merz CN, et al. Sex- and age-based temporal trends in Takotsubo syndrome incidence in the United States. J Am Heart Assoc. 2021;10(20):e019583.
[5] 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):22-29.
[6] Pelliccia F, Kaski JC, Crea F, Camici PG. Pathophysiology of Takotsubo syndrome. Circulation. 2017;135(24):2426-41.
[7] Lazaridis K, Tzartos SJ. Myasthenia gravis: Autoantibody specificities and their role in MG management. Front Neurol. 2020;11:596981.
[8] Roper J, Fleming ME, Long B, Koyfman A. Myasthenia gravis and crisis: Evaluation and management in the emergency department. J Emerg Med. 2017;53(6):843-53.
[9] Barbaryan A, Bailuc SL, Patel K, Raqeem MW, Thakur A, Mirrakhimov AE. Emotional stress as a trigger for reverse Takotsubo cardiomyopathy: A case report and literature review. Am J Case Rep. 2016;17:137-42.
[10] Jahangir A, Niazi MR, Sahra S, Javed A, Krzyzak M. An unfortunate case of Takotsubo cardiomyopathy during plasmapheresis for myasthenic crisis. Cureus. 2022;14(1):e21015.
[11] Rathish D, Karalliyadda M. Takotsubo syndrome in patients with myasthenia gravis: A systematic review of previously reported cases. BMC Neurol. 2019;19(1):281.
[12] Bijulal S, Harikrishnan S, Namboodiri N, Ajitkumar VK, Gupta D, Mathuranath PS. Tako-tsubo cardiomyopathy in a patient with myasthenia gravis crisis: A rare clinical association. BMJ Case Rep. 2009;2009:bcr0620080182.
[13] Primetshofer D, Agladze R, Kratzer H, Reisinger J, Siostrzonek P. Tako-Tsubo syndrome: An important differential diagnosis in patients with acute chest pain. Wien Klin Wochenschr. 2010;122(1-2):37-44.
[14] El-Battrawy I, Behnes M, Hillenbrand D, Haghi D, Hoffmann U, Papavassiliu T, et al. Prevalence, clinical characteristics, and predictors of patients with thromboembolic events in Takotsubo cardiomyopathy. Clin Med Insights Cardiol. 2016;10:1179546816681697.
[15] Eitel I, Lücke C, Grothoff M, Sareban M, Schuler G, Thiele H, Gutberlet M. Inflammation in Takotsubo cardiomyopathy: Insights from cardiovascular magnetic resonance imaging. Eur Radiol. 2010;20(2):422-31.
[16] Stensæth KH, Fossum E, Hoffmann P, Mangschau A, Skretteberg PT, Kløw NE. Takotsubo cardiomyopathy in acute coronary syndrome: Clinical features and contribution of cardiac magnetic resonance during the acute and convalescent phase. Scand Cardiovasc J. 2011;45(2):77-85.
[17] Verschuuren JJ, Palace J, Murai H, Tannemaat MR, Kaminski HJ, Bril V. Advances and ongoing research in the treatment of autoimmune neuromuscular junction disorders. Lancet Neurol. 2022;21(2):189-202.
[18] Brunetti ND, Santoro F, De Gennaro L, Correale M, Gaglione A, Di Biase M, et al. Combined therapy with beta-blockers and ACE-inhibitors/angiotensin receptor blockers and recurrence of Takotsubo (stress) cardiomyopathy: A meta-regression study. Int J Cardiol. 2017;230:281-83.
[19] AlKowatli H, Alkowatli O, Karzoun A, Sawaf B, Habib MB, Bansal N, et al. Recurrent Takotsubo cardiomyopathy with variable patterns and psychiatric comorbidities: A case report and comprehensive literature review. Clin Case Rep. 2025;13(7):e70635.
[20] Escobar JA, Aung M, Amin S, Gulraiz A, Gandhi FR, Malik BH, Gandhi F. Pathogenesis of ventricular arrhythmias and its effect on long-term prognosis in patients with Takotsubo cardiomyopathy. Cureus. 2020;12(10):e11158.
[21] Battineni A, Mullaguri N, Thanki S, Chockalingam A, Govindarajan R. A case report of recurrent takotsubo cardiomyopathy in a patient during myasthenia crisis. Case Rep Crit Care. 2017;2017:5702075.
[22] Hasan SM, Patel JD, Faluk M, Patel J, Singh P. Takotsubo cardiomyopathy and its variants: A case series and literature review. J Community Hosp Intern Med Perspect. 2020;10(3):210-15. Doi: 10.1080/20009666.2020.1767271.
[23] Chuapakdee O, Layangkool T, Theerasuwipakorn N. Pyridostigmine-induced coronary artery spasm in early-onset myasthenia gravis: A case presentation and Review of the literature. BMJ Case Rep. 2022;15(9):e249819. Doi: 10.1136/bcr-2022-249819.
[24] Assad J, Femia G, Pender P, Badie T, Rajaratnam R. Takotsubo syndrome: A review of presentation, diagnosis, and management. Clin Med Insights Cardiol. 2022;16:11795468211065782. Doi: 10.1177/11795468211065782.
[25] Farjoud Kouhanjani M, Hosseini SA, Mousavi SM, Noroozi Z, Sadeghi P, Jokar-Derisi A, et al. Takotsubo cardiomyopathy and autoimmune disorders: A systematic scoping review of published cases. Int J Clin Pract. 2024;2024(1):7259200. Doi: 10.1155/2024/7259200.
[26] Bubuioc AM, Kudebayeva A, Turuspekova S, Lisnic V, Leone MA. The epidemiology of myasthenia gravis. J Med Life. 2021;14(1):7.

[27] Tahir S, Iguh C, Sadik O, Duggirala N, Ezenwa V, Ahmed S. Cardiovascular comorbidities in myasthenia gravis: A systematic review and meta-analysis. Neuromuscul Disord. 2024;43:104441-474.
[28] Heckmann JM, Europa TA, Soni AJ, Nel M. The epidemiology and phenotypes of ocular manifestations in childhood and juvenile myasthenia gravis: A review. Front Neurol. 2022;13:834212.
[29] Baik AH, Tsai KK, Oh DY, Aras MA. Mechanisms and clinical manifestations of cardiovascular toxicities associated with immune checkpoint inhibitors. Clin Sci. 2021;135(5):703-24.
[30] Prokudina ES, Kurbatov BK, Zavadovsky KV, Vrublevsky AV, Naryzhnaya NV, Lishmanov YB, et al. Takotsubo syndrome: Clinical manifestations, aetiology and pathogenesis. Curr Cardiol Rev. 2021;17(2):188-203.
[31] Abraham M, Bhattacharjee S, Ram A, Maramattom BV, Padmanabhan S, Soman A. Understanding and managing autonomic disorders in the neurocritical care unit: A comprehensive review. Neurol India. 2022;70(2):485-90.
[32] Culerrier J, Terrier B, Groh M, Lopez-Sublet M, De Sainte Marie B, Falgarone G, et al. Takotsubo syndrome occurring in systemic diseases: A French multicenter retrospective case-control study and literature review. Autoimmun Rev. 2024;23(2):103483.
[33] Shams Y. Coronary microvascular dysfunction in Takotsubo syndrome: Cause or consequence. Am J Cardiovasc Dis. 2021;11(2):184-89.
[34] Tahsili-Fahadan P, Geocadin RG. Heart–brain axis: Effects of neurologic injury on cardiovascular function. Circ Res. 2017;120(3):559-72.
[35] Sánchez-Camacho A, Torres-Zurita A, Gallego-López L, Hernández-Pacheco R, Silva-Romeiro S, Álamo de la Gala MD, et al. Management of immune-related myocarditis, myositis and myasthenia gravis (MMM) overlap syndrome: A single institution case series and literature review. Front Immunol. 2025;16:1597259. Doi: 10.3389/fimmu. 2025.1597259.
[36] De Perna ML, Rigamonti E, Zannoni R, Espeli V, Moschovitis G. Immune checkpoint inhibitors and cardiovascular adverse events. ESC Heart Fail. 2025;12:2404-16. Doi: 10.1002/ehf2.15281.
[37] Singh T, Khan H, Gamble DT, Scally C, Newby DE, Dawson D. Takotsubo syndrome: Pathophysiology, emerging concepts, and clinical implications. Circulation. 2022;145(13):1002-19. Doi: 10.1161/CIRCULATIONAHA.121.055854.
[38] 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 Taskforce on Takotsubo Syndrome of the Heart Failure Association of the European Society of Cardiology. Eur J Heart Fail. 2016;18(1):8-27. Doi: 10.1002/ejhf.424.
[39] Gopalakrishnan P, Zaidi R, Sardar MR. Takotsubo cardiomyopathy: Pathophysiology and role of cardiac biomarkers in differential diagnosis. World J Cardiol. 2017;9(9):723-30. Doi: 10.4330/wjc.v9.i9.723.
[40] Rosyich MW. Recurrent myasthenic crisis triggered by infection: Successful management with plasmapheresis. Vasc Endovascular Rev. 2025;8(2 Suppl):198-200.
[41] Sheikh S, Alvi U, Soliven B, Rezania K. Drugs that induce or cause deterioration of myasthenia gravis: An update. J Clin Med. 2021;10(7):1537.
[42] Abanador-Kamper N, Kamper L, Wolfertz J, Pomjanski W, Wolf-Pütz A, Seyfarth M. Evaluation of therapy management and outcome in Takotsubo syndrome. BMC Cardiovasc Disord. 2017;17:225.
[43] Yanagihashi M, Okamoto R, Morioka H, Sawada M, Matsumoto S, Ikeda T, et al. Coronary spastic angina after the administration of intravenous immunoglobulin in myasthenia gravis: A case report. BMC Neurol. 2020;20(1):319.
[44] Akashi YJ, Musha H, Kida K, Itoh K, Inoue K, Kawasaki K, et al. Reversible ventricular dysfunction: Takotsubo cardiomyopathy. Eur J Heart Fail. 2005;7(7):1171-76.
[45] Hsu CW, Chang CC, Lin CS. Intraoperative cardiogenic shock induced by refractory coronary artery spasm in a patient with myasthenia gravis: A case report. World J Clin Cases. 2023;11(36):8589-95.
[46] Uribarri A, Núñez-Gil IJ, Conty DA, Vedia O, Almendro-Delia M, Duran Cambra A, et al. Short- and long-term prognosis of patients with Takotsubo syndrome based on different triggers: Importance of the physical nature. J Am Heart Assoc. 2019;8(24):e01370.
[47] Jesel L, Berthon C, Messas N, Lim HS, Girardey M, Marzak H, et al. Atrial arrhythmias in Takotsubo cardiomyopathy: Incidence, predictive factors, and prognosis. Europace. 2019;21(2):298-305.
[48] Nishinarita R, Kawamura Y, Yasuda T, Horikoshi Y, Ito D, Sugihara T, et al. A case of Takotsubo cardiomyopathy leading to the diagnosis of myasthenia gravis. J Cardiol Cases. 2012;6(5):e141-e144.
[49] Finsterer J, Stöllberger C. Stress from myasthenic crisis triggers Takotsubo (broken heart) syndrome. Int J Cardiol. 2016;203:616-17.
[50] Ghadri JR, Ruschitzka F, Lüscher TF, Templin C. Takotsubo cardiomyopathy: Still much more to learn. Heart. 2014;100(22):1804-12.
[51] Law C, Flaherty CV, Bandyopadhyay S. A review of psychiatric comorbidity in myasthenia gravis. Cureus. 2020;12(7):e9204.
[52] Wittstein IS. Stress cardiomyopathy: A syndrome of catecholamine-mediated myocardial stunning? Cell Mol Neurobiol. 2012;32(5):847-57.
[53] Fröhlich GM, Schoch B, Schmid F, Keller P, Sudano I, Lüscher TF, et al. Takotsubo cardiomyopathy has a unique cardiac biomarker profile: NT-proBNP/myoglobin and NT-proBNP/troponin T ratios for the differential diagnosis of acute coronary syndromes and stress-induced cardiomyopathy. Int J Cardiol. 2012;154(3):328-32.
[54] Wiendl H, Abicht A, Chan A, Della Marina A, Hagenacker T, Hekmat K, et al. Guideline for the management of myasthenic syndromes. Ther Adv Neurol Disord. 2023;16:17562864231213240.
[55] Akashi YJ, Barbaro G, Sakurai T, Nakazawa K, Miyake F. Cardiac autonomic imbalance in patients with reversible ventricular dysfunction, Takotsubo cardiomyopathy. QJM. 2007;100(6):335-43.
[56] Usenko OY, Sydiuk AV, Sydiuk OY, Antonenko VV, Savenko GY, Teslia OT, et al. Perioperative management of a patient with myasthenia gravis: A case report, treatment of myasthenic crisis and literature review. Emergency Medicine. 2025;21(2):189-98.
[57] Kosuge M, Ebina T, Hibi K, Morita S, Okuda J, Iwahashi N, et al. Simple and accurate electrocardiographic criteria to differentiate Takotsubo cardiomyopathy from anterior acute myocardial infarction. J Am Coll Cardiol. 2010;55(22):2514-16.
[58] Citro R, Okura H, Ghadri JR, Izumi C, Meimoun P, Izumo M, et al. Multimodality imaging in Takotsubo syndrome: A joint consensus document of the EACVI and JSE. Eur Heart J Cardiovasc Imaging. 2020;21(11):1184-207.
[59] Beydoun SR, Wang J, Levine RL, Farvid A. Emotional stress as a trigger of myasthenic crisis and concomitant Takotsubo cardiomyopathy: A case report. J Med Case Rep. 2010;4:393.
[60] Douglas TM, Wengrofsky P, Haseeb S, Kupferstein E, Kariyanna PT, Schwartz J, et al. Takotsubo cardiomyopathy mimicking myocardial infarction in a man with myasthenic crisis: A case report and literature review. Am J Med Case Rep. 2018;6(9):184-88.
[61] Cuevas HH, López RD, Trejo BP, Ramón RR, Isunza HE, Brito MR. Takotsubo syndrome associated with myasthenic crisis: A case report. Rev Mex Cardiol. 2016;27(3):123-28.
[62] Ipe TS, Davis AR, Raval JS. Therapeutic plasma exchange in myasthenia gravis: A systematic literature review and meta-analysis of comparative evidence. Front Neurol. 2021;12:662856.
[63] Santoro F, Sharkey S, Citro R, Miura T, Arcari L, Urbano-Moral JA, et al. Beta-blockers and renin–angiotensin system inhibitors for Takotsubo syndrome recurrence: A network meta-analysis. Heart. 2024;110(7):476-81.
[64] Kariyanna PT, Sutarjono B, Jayarangaiah A, Okwechime R, Wengrofsky P, McFarlane IM. Defining features of patients who develop Takotsubo cardiomyopathy during myasthenic crisis: A systematic review of case studies. Am J Med Case Rep. 2019;7(9):203-09.
[65] Hu C, Bao S, Lin J, Li S, Wang L, Liu W, et al. A case of myasthenia gravis with Takotsubo cardiomyopathy presenting with respiratory and cardiac arrest. Clin Auton Res. 2025;35:01-04.
[66] Y-Hassan S, Törnvall P. Epidemiology, pathogenesis, and management of takotsubo syndrome. Clin Auton Res. 2018;28(1):53-65.

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