



β-blocker Therapy In Takotsubo Syndrome: Current Evidence And Future Perspectives

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SUMMARY

Introduction: Takotsubo syndrome (TTS) is an acute, stress-induced form of transient left ventricular dysfunction, most commonly triggered by emotional or physical stress, and may mimic various acute cardiac conditions at initial presentation. Despite increasing recognition, optimal long-term pharmacological management remains uncertain, particularly regarding the role of β-blocker therapy.

Methodology: This narrative review was based on a literature search performed in PubMed and Google Scholar databases during May 2026. Studies published between January 2008 and April 2026 were considered, including original research articles, meta-analyses, registry data and expert consensus documents addressing the role of β-blockers in Takotsubo syndrome.

Topic: β-blocker represent a widely used class of drugs in clinical practice in patients diagnosed with Takotsubo syndrome. The rationale for β-blocker use is based on the hypothesis of catecholamine excess in its pathophysiological basis. However, available evidence regarding their impact on mortality and recurrence is inconsistent. Although β-blockers are indicated in certain clinical situations, such as atrial fibrillation, hypertension, heart failure, left ventricular outflow tract obstruction, and the prevention and treatment of arrhythmias, their routine use in all patients with TTS is not supported by current evidence from studies. Increasing evidence suggests that therapy should be individualized according to the clinical phenotype and associated cardiovascular conditions, rather than the diagnosis of TTS itself.

Conclusion: β-blocker therapy in Takotsubo syndrome should be guided by established cardiovascular indications and individual clinical characteristics of patients. Routine long-term use in all patients is not supported by current evidence, highlighting the need for randomized controlled trials to define their exact role in this population.

Keywords: Takotsubo Syndrome, β-blockers, Stress Cardiomyopathy, Pharmacotherapy, Cardiovascular Diseases

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INTRODUCTION

Takotsubo syndrome (TTS) is an acute, usually reversible form of myocardial dysfunction, most commonly triggered by emotional or physical stress [1]. Clinically, it often presents as an acute coronary syndrome mimic and is most frequently diagnosed during evaluation for suspected acute myocardial infarction. Although once considered a rare condition, the incidence of TTS has increased over time, likely reflecting both improved recognition among clinicians and the growing burden of psychosocial stress in modern society [2]. Although its pathophysiology remains incompletely understood, excessive catecholamine release is considered the central mechanism, leading to myocardial stunning through multiple pathways, including microvascular dysfunction, vasospasm, and inflammatory activation [3–5]. Contrary to earlier beliefs that TTS is a benign condition, contemporary studies have demonstrated that long-term mortality and cardiovascular events may approach those observed after acute coronary syndrome [6,7]. Because catecholamine excess is considered the hallmark of TTS, β -blockers have become one of the most frequently prescribed long-term therapies despite limited supporting evidence. However, observational studies have yielded conflicting results regarding their impact on survival and recurrence [8].

METHODOLOGY

This narrative review summarizes the current evidence regarding the role of β -blocker therapy in Takotsubo syndrome. A comprehensive literature search was performed in the PubMed and Google Scholar databases during May 2026, covering articles published between January 2008 and April 2026. The search strategy included combinations of the following keywords: Takotsubo syndrome, stress cardiomyopathy, broken heart syndrome, β -blockers, beta-adrenergic blockade, heart failure, left ventricular outflow tract obstruction, ventricular arrhythmias, atrial fibrillation, arterial hypertension, mortality, recurrence, and long-term outcomes. English-language original research articles, systematic reviews, meta-analyses, clinical guidelines, expert consensus statements, and relevant observational studies were evaluated for inclusion. Studies were excluded if they were duplicates, lacked perti-

nent clinical data, or did not directly address β -blocker utility in TTS. Priority was given to landmark studies and recent high-quality evidence to provide a comprehensive and up-to-date synthesized overview.

TOPIC

Given the central role of sympathetic hyperactivation and catecholamine excess in the pathophysiology of Takotsubo syndrome (TTS), β -blockers have been widely adopted in clinical practice as a theoretically appealing therapeutic strategy [9]. In routine clinical practice, patients with TTS are initially managed as having acute coronary syndrome (ACS) until obstructive coronary artery disease is excluded by coronary angiography [10]. Consequently, acute-phase management remains largely empirical, frequently following established acute coronary syndrome protocols. Because left ventricular (LV) function recovery typically initiates early post-symptom onset, it remains uncertain whether currently available therapies modify the natural course of the disease or primarily provide supportive management and prevent complications [11]. Therefore, contemporary treatment strategies are directed mainly toward stabilization of the acute clinical presentation, management of complications, and treatment of coexisting cardiovascular conditions rather than delivering disease-specific intervention [12]. Long-term management strategies also remain empirical. Both β -blockers and renin-angiotensin-aldosterone system (RAAS) inhibitors—such as ACE inhibitors and angiotensin receptor blockers (ARBs)—are routinely prescribed despite a lack of definitive randomized trial data confirming their prognostic benefit [8]. Observational studies have yielded conflicting results regarding the association between β -blocker therapy and long-term outcomes in Takotsubo syndrome. Several registry-based analyses have reported improved survival among patients treated with β -blockers [13–17]. In contrast, other studies have failed to demonstrate a statistically significant association between β -blocker therapy and mortality, underscoring persistent uncertainty regarding their true prognostic impact. Importantly, meta-analytic data have suggested a potential in-hospital benefit; however, results remain heterogeneous across studies and clinical settings [8,18–21]. Accordingly, rather than ques-

tioning whether all patients should receive β -blockers, a more clinically pertinent issue is identifying specific patient subgroups most likely to derive benefit. The following sections outline the rationale for β -blocker administration across key clinical scenarios.

In patients who have recovered from the acute phase of TTS, are asymptomatic, hemodynamically stable, and have no persistent left ventricular dysfunction or concomitant cardiovascular disease, the routine initiation or continuation of long-term pharmacological therapy remains questionable [22,23]. Although β -blockers are frequently prescribed based on the presumed role of catecholamine excess in TTS pathophysiology, there is currently no convincing evidence that prolonged sympathetic activation persists after the acute phase or that long-term β -blocker therapy improves outcomes in otherwise low-risk patients [24,25]. Moreover, observational studies have shown that TTS may occur despite chronic β -blocker treatment, suggesting that these agents do not reliably prevent disease recurrence [26]. Therefore, in asymptomatic patients without established cardiovascular indications for β -blocker therapy, routine long-term treatment should be approached cautiously, with decisions individualized according to the patient's overall clinical profile rather than the diagnosis of TTS alone.

Arterial hypertension is a prominent comorbidity in patients with Takotsubo syndrome and frequently represents an established indication for antihypertensive therapy. In the acute phase, β -blockers may be considered in patients with elevated blood pressure and/or sinus tachycardia. Short- or ultra-short-acting agents, such as esmolol or landiolol, may be preferred during the early phase because they allow rapid dose titration and prompt discontinuation. Once the patient is clinically stable, longer-acting cardioselective agents, such as metoprolol or bisoprolol, may be used when continued β -blockade is indicated [27]. Ivabradine may be considered as an alternative for the management of persistent sinus tachycardia when β -blockers are contraindicated or poorly tolerated. However, in patients with well-controlled blood pressure and no other indication for β -blocker therapy, current evidence does not support their routine long-term use solely on the basis of a previous episode of TTS.

Left ventricular outflow tract ob-

struction represents a distinct subgroup of patients with Takotsubo syndrome in whom β -blockers have a well-established pathophysiological rationale [28,29]. By reducing heart rate and myocardial contractility, β -blockers prolong diastolic filling, improve left ventricular preload, and decrease the dynamic intraventricular pressure gradient. In the acute setting, short-acting intravenous agents such as esmolol or landiolol are generally preferred because they allow careful dose titration according to the patient's hemodynamic status, while cardio selective oral β -blockers may be continued once clinical stability is achieved [30,31]. However, in the presence of cardiogenic shock accompanied by severe left ventricular dysfunction, β -blockers should be administered with extreme caution or avoided because of their negative inotropic effects.

Heart failure in Takotsubo syndrome is most commonly observed in patients with reduced left ventricular ejection fraction, advanced age, or in the setting of physical stressors. Management is primarily supportive and similar to that of acute heart failure due to other causes. In this context, the role of β -blockers is limited during the acute phase, particularly in patients with low cardiac output or signs of hypoperfusion, where their negative inotropic effects may lead to clinical deterioration. If initiated early, β -blocker therapy should be carefully re-evaluated and discontinued if heart failure worsens. Conversely, reintroduction of β -blockers may be considered during the recovery phase, once left ventricular function improves and hemodynamic stability is achieved, or when there is a concomitant indication such as chronic heart failure [32,33].

Atrial fibrillation is the most common supraventricular arrhythmia complicating Takotsubo syndrome, with a reported incidence ranging from 7% to 33%. Beyond its frequency, accumulating evidence indicates that atrial fibrillation is associated with a significantly worse prognosis [34]. In a recent meta-analysis including more than 4,000 patients with Takotsubo syndrome, atrial fibrillation was associated with an approximately threefold higher risk of adverse outcomes. In this clinical setting, β -blockers represent an appropriate therapeutic option for ventricular rate control. Thus, in patients with concomitant atrial fibrillation, the indication for β -blocker therapy is primarily driven by the arrhythmia itself rather than by Takotsubo

syndrome [35].

Ventricular arrhythmias, including ventricular fibrillation and torsades de pointes, are an important but relatively under-recognized complication of Takotsubo syndrome, occurring in more than 8% of patients. These arrhythmias are frequently associated with significant QTc prolongation, which is most pronounced during the early phase of the disease and correlates with an increased risk of life-threatening events. Takotsubo syndrome should therefore be considered a form of acquired long QT syndrome, particularly in the context of hyperadrenergic activation. Continuous electrocardiographic monitoring is recommended in patients with marked QT prolongation until normalization of repolarization is documented. In this setting, β -blockers are often used to attenuate sympathetic overactivity; however, their role remains primarily supportive, while management is driven by prevention and treatment of malignant arrhythmias according to established electrophysiological principles [36,37].

TTS recurrence is relatively low, with an estimated cumulative incidence of approximately 5% at 6 years and an annual recurrence rate of around 1–2%. In addition to true recurrence, a proportion of patients experience persistent or recurrent symptoms such as chest pain or dyspnea without clear evidence of recurrent left ventricular dysfunction. Pooled observational data cohort analyses suggest that the use of β -blockers at discharge is not associated with a reduced risk of recurrence. Instead, initial presentation severity remains the primary determinant of long-term recurrence [38].

Artificial intelligence and machine learning approaches are increasingly being applied to the analysis of large clinical datasets, including those derived from patients with Takotsubo syndrome [39,40]. These emerging technologies may contribute to improved risk stratification, identification of novel diagnostic and prognostic markers, and a more personalized approach to patient management.

CONCLUSION

The pathophysiology of Takotsubo syndrome [TTS] remains incompletely understood, and its management is currently based largely on therapeutic strategies established for other cardiovascular diseases. A definitive diag-

nosis of TTS is typically made after a certain delay, once acute conditions that may mimic its clinical presentation have been excluded. Consequently, initial management should focus on supportive therapy. Furthermore, regardless of the underlying pathophysiological mechanism, TTS is characterized by transient and typically fully reversible ventricular dysfunction. While this justifies the use of pharmacological therapy during the acute phase, it does not necessarily support its continuation during long-term follow-up. Given the spontaneous recovery observed in the majority of patients, a conservative approach with careful clinical monitoring may be appropriate in asymptomatic individuals without other comorbidities. Pharmacological therapy prescribed for pre-existing comorbidities should not be routinely discontinued but rather adjusted according to the patient's clinical status. In particular, the use of β -blockers should be guided by established cardiovascular indications and individual patient characteristics rather than by the diagnosis of Takotsubo syndrome alone. Future prospective studies, including randomized controlled trials, are needed to better define the role of β -blocker therapy in specific patient subgroups and to identify individuals who may derive the greatest clinical benefit.

CONFLICT OF INTEREST

All authors declare no conflict of interest.

REFERENCES

1. Crea F, Iannaccone G, La Vecchia G, Montone RA. An update on the mechanisms of Takotsubo syndrome: 'At the end an acute coronary syndrome'. *J Mol Cell Cardiol.* 2024 Jun;191:1-6.
2. Ravindran J, Brieger D. Clinical perspectives: Takotsubo cardiomyopathy. *Intern Med J.* 2024 Nov;54(11):1785-95.
3. Kato K, Lyon AR, Ghadri JR, Templin C. Takotsubo syndrome: aetiology, presentation and treatment. *Heart Br Card Soc.* 2017 Sep;103(18):1461-9.
4. 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.
5. Shadmand M, Lautze J, Md AM. Takotsubo pathophysiology and complications: what we know and what we do not know. *Heart Fail Rev.* 2024 Mar;29(2):497-510.
6. Singh T, Khan H, Gamble DT, Scally C, Newby DE,

- Dawson D. Takotsubo Syndrome: Pathophysiology, Emerging Concepts, and Clinical Implications. *Circulation*. 2022 Mar 29;145(13):1002-19.
7. Isogai T, Matsui H, Tanaka H, Fushimi K, Yasunaga H. In-hospital Takotsubo syndrome versus in-hospital acute myocardial infarction among patients admitted for non-cardiac diseases: a nationwide inpatient database study. *Heart Vessels*. 2019 Sep;34(9):1479-90.
 8. Raposeiras-Roubin S, Santoro F, Arcari L, Vazirani R, Novo G, Uribarri A, Enrica M, Lopez-Pais J, Guerra F, Alfonso F, Pätz T. B-blockers and long-term mortality in takotsubo syndrome: results of the multicenter GEIST registry. *Heart Failure*. 2025 May 1;13(5):815-25.
 9. Madias JE. Blood norepinephrine/epinephrine/dopamine measurements in 108 patients with takotsubo syndrome from the world literature: pathophysiological implications. *Acta cardiologica*. 2021 Nov 22;76(10):1083-91.
 10. Lyon AR, Bossone E, Schneider B, Sechtem U, Citro R, Underwood SR, Sheppard MN, Figtree GA, Parodi G, Akashi YJ, Ruschitzka F. 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. *European journal of heart failure*. 2016 Jan;18(1):8-27.
 11. Madias JE. Insulin and short acting iv beta blockers: A "new" proposal for the acute management of takotsubo syndrome. *International Journal of Cardiology*. 2021 Jul 1;334:18-20.
 12. Dias A, Gil JJ, Santoro F, Madias JE, Pelliccia F, Brunetti ND, Salmoirago-Blotcher E, Sharkey SW, Eitel I, Akashi YJ, El-Battrawy I. Takotsubo syndrome: State-of-the-art review by an expert panel-Part 1. *Cardiovascular Revascularization Medicine*. 2019 Jan 1;20(1):70-9.
 13. Jain H, Soni K, Odat RM, Agrawal SP, Pushparaji B, Levine DJ, Salmoirago-Blotcher E, Abbott JD, Vallabhajosyula S. Impact of B-blockers on long-term mortality in Takotsubo syndrome: a real-world analysis of the TriNetX Global Collaborative Network database. *European Heart Journal: Acute Cardiovascular Care*. 2026 Mar;15(3):206-11.
 14. Silverio A, Parodi G, Scudiero F, Bossone E, Di Maio M, Vriz O, Bellino M, Zito C, Provenza G, Radano I, Baldi C. B-blockers are associated with better long-term survival in patients with Takotsubo syndrome. *Heart*. 2022 Sep;108(17):1369-76.
 15. Silverio A, Citro R, Bossone E, Bellino M, Zito C, Provenza G, Protà C, Iuliano G, Radano I, Polito MV, Baldi C. 5037 Drug treatment with B-blockers and long-term outcome in patients with takotsubo syndrome: results from the takotsubo Italian network. *European Heart Journal*. 2019 Oct 1;40(Supplement_1):ehz746-0040.
 16. Rodriguez Mejia RA, Singh A, Bahekar A, Gupta A. Efficacy of B-blocker therapy in Takotsubo cardiomyopathy: A systematic review and meta-analysis. *Int J Cardiol*. 2025 Oct 15;437:133483.
 17. Komamura K, Fukui M, Iwasaku T, Hirotani S, Masuyama T. Takotsubo cardiomyopathy: Pathophysiology, diagnosis and treatment. *World J Cardiol*. 2014 Jul 26;6(7):602-9.
 18. Santoro F, Sharkey S, Citro R, Miura T, Arcari L, Urbano-Moral JA, Stiermaier T, Nuñez-Gil JJ, Silverio A, Di Nunno N, Ragnatela I. B-blockers and renin-angiotensin system inhibitors for Takotsubo syndrome recurrence: a network meta-analysis. *Heart*. 2024 Apr 1;110(7):476-81.
 19. Kummer M, El-Battrawy I, Gietzen T, Ansari U, Behnes M, Lang S, Zhou X, Borggreve M, Akin I. The use of beta blockers in takotsubo syndrome as compared to acute coronary syndrome. *Frontiers in Pharmacology*. 2020 May 14;11:681.
 20. Ahsan MJ, Ahmad S, Dvalishvili M, Yousaf A, Sigurdsson G, Goldsweig AM. Effect of B-Blocker Use on Long-Term Mortality in Takotsubo Cardiomyopathy: A Systematic Review and Meta-Analysis. *Am J Cardiol*. 2023 Oct 1;204:168-70.
 21. Inban P, Thai A, Parekh RS, Kudaiarov D, Thalamanchi K, John J, Sharma R, Aravazhi PS. Takotsubo cardiomyopathy: Advances in pathophysiology, diagnostic biomarkers, genetic insights, multisystemic involvement, treatment updates & multidisciplinary interventions. *Disease-a-Month*. 2025 Oct 30:102029.
 22. Omerovic E, Redfors B. Takotsubo syndrome: pathophysiological insights and innovations in patient care. *Nature Reviews Cardiology*. 2026 Apr;23(4):239-54.
 23. Matta AG, Carrié D. Epidemiology, pathophysiology, diagnosis, and principles of management of Takotsubo cardiomyopathy: a review. *Medical science monitor: international medical journal of experimental and clinical research*. 2023 Mar 6;29:e939020-1.
 24. Santoro F, Mallardi A, Leopizzi A, Vitale E, Rawish E, Stiermaier T, Eitel I, Brunetti ND. Current knowledge and future challenges in Takotsubo syndrome: part 2—treatment and prognosis. *Journal of Clinical Medicine*. 2021 Jan 26;10(3):468.
 25. Akashi YJ, Goldstein DS, Barbaro G, Ueyama T. Takotsubo cardiomyopathy: a new form of acute, reversible heart failure. *Circulation*. 2008 Dec 16;118(25):2754-62.
 26. Lau C, Chiu S, Nayak R, Lin B, Lee MS. Survival and risk of recurrence of takotsubo syndrome. *Heart*. 2021 Jul 1;107(14):1160-6.
 27. Schultz T, Shao Y, Redfors B, Bergmann Sverrisdóttir Y, Råmunddal T, Albertsson P, Matejka G, Omerovic E. Stress-induced cardiomyopathy in Sweden: evidence for different ethnic predisposition and altered cardio-circulatory status. *Cardiology*. 2012 Jul 27;122(3):180-6.

28. Di Vece D, Silverio A, Bellino M, Galasso G, Vecchione C, La Canna G, Citro R. Dynamic left intraventricular obstruction phenotype in Takotsubo syndrome. *Journal of clinical medicine*. 2021 Jul 22;10(15):3235.
29. Santoro F, Ieva R, Ferraretti A, Fanelli M, Muscaico F, Tarantino N, Martino LD, Gennaro LD, Caldarella P, Biase MD, Brunetti ND. Hemodynamic effects, safety, and feasibility of intravenous esmolol infusion during Takotsubo cardiomyopathy with left ventricular outflow tract obstruction: results from a multicenter registry. *Cardiovascular Therapeutics*. 2016 Jun;34(3):161-6.
30. Yoshioka T, Hashimoto A, Tsuchihashi K, Nagao K, Kyuma M, Ooiwa H, Nozawa A, Shimoshige S, Eguchi M, Wakabayashi T, Yuda S. Clinical implications of midventricular obstruction and intravenous propranolol use in transient left ventricular apical ballooning (Tako-tsubo cardiomyopathy). *American heart journal*. 2008 Mar 1;155(3):526-e1.
31. Sato Y, Matsumoto K, Sakai J, Nakamura T, Tanaka H, Hirata K. 'Incompatible Housemates': Hypertrophic Obstructive Cardiomyopathy and Takotsubo Syndrome. *Intern Med*. 2020 Apr 1;59(7):957-62.
32. Citro R, Radano I, Parodi G, Di Vece D, Zito C, Novo G, Provenza G, Bellino M, Protà C, Silverio A, Antonini-Canterin F. Long-term outcome in patients with Takotsubo syndrome presenting with severely reduced left ventricular ejection fraction. *European journal of heart failure*. 2019 Jun;21(6):781-9.
33. Akashi YJ. Long-term prognosis in patients with Takotsubo syndrome. *Eur J Heart Fail*. 2019 Jun;21(6):790-1.
34. Prasitlumkum N, Kittipibul V, Limpruttidham N, Rattanawong P, Chongsathidkiet P, Boondarikpornpant T. The presence of atrial fibrillation in Takotsubo cardiomyopathy is predictive of mortality: Systematic review and meta-analysis. *Ann Noninvasive Electrocardiol Off J Int Soc Holter Noninvasive Electrocardiol Inc*. 2018 Jun 25;24(1):e12566.
35. Osiecki A, Sarwiński K, Gąsior J, Kochman W, Michałkiewicz D, Pawlak A. Incidence and prognostic significance of atrial fibrillation in patients with tako-tsubo syndrome-systematic review and meta-analysis. *Casp J Intern Med*. 2025;16(3):405-16.
36. Madias C, Fitzgibbons TP, Alsheikh-Ali AA, Bouchard JL, Kalsmith B, Garlitski AC, Tighe DA, Estes III NM, Aurigemma GP, Link MS. Acquired long QT syndrome from stress cardiomyopathy is associated with ventricular arrhythmias and torsades de pointes. *Heart Rhythm*. 2011 Apr 1;8(4):555-61.
37. Stiermaier T, Eitel C, Denef S, Desch S, Schuler G, Thiele H, Eitel I. Prevalence and clinical significance of life-threatening arrhythmias in takotsubo cardiomyopathy. *Journal of the American College of Cardiology*. 2015 May 19;65(19):2148-50.
38. Singh K, Carson K, Usmani Z, Sawhney G, Shah R, Horowitz J. Systematic review and meta-analysis of incidence and correlates of recurrence of takotsubo cardiomyopathy. *Int J Cardiol*. 2014 Jul 1;174(3):696-701.
39. Agrawal A, Bhagat U, Arockiam AD, Haroun E, Faulx M, Desai MY, Jaber W, Menon V, Griffin B, Wang TK. Machine learning risk-prediction model for in-hospital mortality in Takotsubo cardiomyopathy. *International Journal of Cardiology*. 2025 Jul 1;430:133181.
40. De Filippo O, Cammann VL, Pancotti C, Di Vece D, Silverio A, Schweiger V, Niederseer D, Szawan KA, Würdinger M, Koleva I, Dusi V. Machine learning-based prediction of in-hospital death for patients with takotsubo syndrome: The InterTAK-ML model. *European journal of heart failure*. 2023 Dec;25(12):2299-311.

Primena β-blokatora u Takotsubo sindromu: aktuelni dokazi i buduće perspektive

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KRATAK SADRŽAJ

Uvod: Takotsubo sindrom (TTS) predstavlja akutni, stresom indukovani oblik prolazne disfunkcije leve komore, najčešće izazvan emocionalnim ili fizičkim stresom i može da imitira različita akutna kardiološka stanja pri inicijalnoj prezentaciji. Optimalno dugoročno farmakološko lečenje ostaje neizvesno, naročito u pogledu uloge terapije beta-blokatorima.

Metodologija: Ovaj narativni pregled zasnovan je na pretrazi literature u bazama PubMed i Google Scholar tokom maja 2026. godine. U obzir su uzeti radovi objavljeni u periodu od januara 2008. do aprila 2026. godine, uključujući originalne istraživačke studije, meta-analize, podatke iz registara, kliničke vodiče i ekspertske konsenzuse koji se odnose na ulogu beta-blokatora u Takotsubo sindromu.

Tema: Beta-blokatori predstavljaju široko primenjivanu grupu lekova u kliničkoj praksi kod bolesnika sa dijagnostikovanom Takotsubo sindromom. Opravdanje za primenu beta blokatora se zasnima na hipotezi kateholaminskog viška u njegovoj patofiziološkoj osnovi. Međutim, dostupni dokazi o njihovom uticaju na mortalitet i rekurencu su nekonzistentni. Iako su beta-blokatori indikovani u određenim kliničkim situacijama, kao što su atrijska fibrilacija, hipertenzija, srčana slabost, opstrukcija izlaznog trakta leve komore, prevencija i lečenje aritmija njihova rutinska primena kod svih bolesnika sa TTS nije podržana aktuelnim dokazima iz studija. Sve više dokaza ukazuje da terapiju treba individualizovati u skladu sa kliničkim fenotipom i pridruženim kardiovaskularnim stanjima, a ne samom dijagnozom TTS.

Zaključak: Terapija beta-blokatorima u Takotsubo sindromu treba da bude vođena postojećim kardiovaskularnim indikacijama i individualnim kliničkim karakteristikama bolesnika. Rutinska dugoročna primena kod svih bolesnika nije podržana aktuelnim dokazima, što naglašava potrebu za randomizovanim kontrolisanim studijama koje bi definisale njihovu tačnu ulogu u ovoj populaciji.

Ključne reči: Takotsubo sindrom, beta-blokatori, stres kardiomiopatija, farmakoterapija, kardiovaskularne bolesti

Received: July 10, 2026

Accepted: July 28, 2026