Open Access Article

Ann. Acad. Med. Siles. (online) 2025; 79: 344–354
eISSN 1734-025X
DOI: 10.18794/aams/207117
www.annales.sum.edu.pl

PRACA POGLĄDOWA
REVIEW

Takotsubo syndrome – A review of recent reports

Zespół takotsubo – przegląd najnowszych doniesień

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ABSTRACT

Takotsubo syndrome is a poorly understood but increasingly diagnosed condition, especially in postmenopausal women. It affects patients of all ages. It is divided into two types according to its triggering factor. In the primary type, chronic stress is the main cause, while in the secondary type the trigger is physical – iatrogenic. The name of the disease comes from the Japanese word for a octopus trap with a narrow neck and wide base. In imaging studies, this cardiomyopathy resembles such a device. The aim of the study is to comprehensively analyze the available literature in scholarly databases such as PubMed, Google Scholar, and ScienceDirect. The work examines historical background, possible risk factors, clinical course, classification of the disease, preventive measures, and treatment strategies. The current lack of consensus regarding the definition used by prestigious cardiology societies has been highlighted in the context of recent scientific research. Further research is necessary to better understand the pathomechanism of the disease, in order to choose the best pharmacological treatment and improve the quality of life of patients with this condition.

KEYWORDS

takotsubo syndrome, cardiomyopathy, acute coronary syndrome, myocardial structural disorder, catecholamines, beta-blockers, prevention, pharmacological treatment

Received: 06.03.2025 **Revised:** 12.06.2025 **Accepted:** 13.06.2025 **Published online:** 29.09.2025

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Publisher: Medical University of Silesia, Katowice, Poland

STRESZCZENIE

Zespół takotsubo jest jednostką chorobową słabo poznaną, lecz coraz częściej diagnozowaną, zwłaszcza w grupie kobiet po menopauzie. Dotyczy pacjentów w każdym wieku. Ze względu na czynnik wyzwalający wyróżnia się dwa typy choroby – pierwotny oraz wtórny. W typie pierwotnym główną przyczyną jest przewlekły stres, natomiast w typie wtórnym czynnik fizyczny – jatrogenny. Nazwa choroby pochodzi od japońskiego słowa oznaczającego naczynie rybackie z wąską szyjką i szerokim dnem, używane do chwytania ośmiornic. W badaniu obrazowym kardiomiopatia ta przypomina kształtem owo naczynie. Celem pracy jest kompleksowa analiza dostępnej literatury w bazach danych takich jak PubMed, Google Scholar i ScienceDirect. W pracy uwzględniono rys historyczny, możliwe czynniki ryzyka, przebieg kliniczny oraz klasyfikację schorzenia. Omówiono także postępowanie profilaktyczne i strategie leczenia. Obecny brak jednomyślności w definicji stosowanej przez prestiżowe towarzystwa kardiologiczne został uwidoczniony w kontekście najnowszych badań naukowych. Konieczne są dalsze badania w celu lepszego zrozumienia patomechanizmu choroby, aby dobrać jak najlepsze leczenie farmakologiczne i poprawić jakość życia pacjentów z tym schorzeniem.

SŁOWA KLUCZOWE

zespół takotsubo, kardiomiopatia, ostry zespół wieńcowy, zaburzenie budowy mięśnia sercowego, katecholaminy, beta-blokery, profilaktyka, leczenie farmakologiczne

Introduction

Takotsubo syndrome (TTS) is recognized as a stress--induced cardiomyopathy and as a form of cardiomyopathy with paraclinical and clinical symptoms similar to acute myocardial infarction (AMI) [1]. The exact pathogenesis of the syndrome remains unclear. Typical presenting symptoms reported by patients are chest pain and dyspnea. On physical examination, an electrocardiogram may show transient ST elevation and a small rise in cardiac troponin T [2]. It progresses to an acute clinical state characterized by reversible systolic and diastolic dysfunction of the left ventricle. In this condition, one segment of the ventricle contracts very strongly, while another segment becomes dilated and ceases to function. The InterTAK registry distinguishes four TTS subtypes. The most prevalent type is apical [1]; it is associated with hypokinesis or akinesis of the mid and apical segments of the left ventricle. The disease is often preceded by a physical or emotional stressor that causes a sudden increase in catecholamines, leading to heart dysfunction [3,4]. Over the past few years, there has been a noticeable upward trend in the number of cases reported. Despite the growing awareness among physicians about TTS, the syndrome still represents a significant diagnostic challenge. A study published in 2019 indicated that the in-hospital mortality risk is similar to that in patients presenting with acute coronary syndrome. One group which is particularly often diagnosed with TTS is postmenopausal women who have experienced an episode of psychological stress [5].

The authors conducted a detailed analysis of the available scientific literature found in the databases PubMed, Google Scholar, and ScienceDirect. To refine the selection of relevant sources, narrow the results, and increase accuracy, the specific keywords in the search were "takotsubo syndrome," "management in takotsubo syndrome," "stress cardiomyopathy," "broken heart syndrome," and the Boolean operators AND and OR. The selection criteria included original research articles and reviews published in peer--reviewed journals in both Polish and English. Publications without full-text access and non-specialist articles lacking scientific evidence were excluded. The sources were also selected by publication date, as articles published within the last ten years were favored. Initially, 10,629 articles were identified based on the search strategy. After removing duplicates and reviewing titles and abstracts, 780 articles remained for further evaluation. Ultimately, after the full texts were analyzed and the inclusion and exclusion criteria were applied, 91 studies were selected for the study (Figure 1).

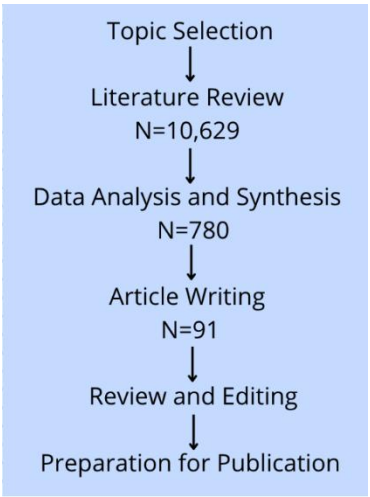

Fig. 1. Flowchart.

Historical background

The majority of newly classified disease entities undergo a name change over time as knowledge about them increases. This was also the case with the syndrome described in the 1990s by Japanese

cardiologist Hikaru Sato, especially in the first few years following his description [6]. Initially, the term "takotsubo cardiomyopathy" was commonly used in the literature on the subject, but over the years, the following names were used to define the disease: "takotsubo cardiomyopathy," "left ventricular ballooning syndrome," "broken heart syndrome," and "stress cardiomyopathy" (Figure 2). Due to the different nomenclature, various abbreviations of these names have appeared in the literature. In the past few years, most experts have agreed that the abbreviation for takotsubo syndrome should be "TS" or "TTS" [7,8]. These abbreviations inform the reader that only the left ventricle of the heart is affected by transient regional systolic dysfunction, dilation, and swelling.

There is also controversy over the classification of the disease as cardiomyopathy, which is defined by persistent structural and functional abnormalities of the heart muscle. The syndrome does not have a genetic basis and is not a primary cardiomyopathy; individuals with cardiomyopathy cannot spontaneously recover. Furthermore, this term cannot be used when abnormalities in the functioning or morphology of the coronary arteries lead to acute myocardial ischemia and are responsible for systolic dysfunction in the left ventricle. Considering the transient nature of this phenomenon, the guidelines for cardiomyopathy published in 2023 by the European Society of Cardiology do not recommend classifying TTS as cardiomyopathy; however, most scientific reports still do so [9,10,11].

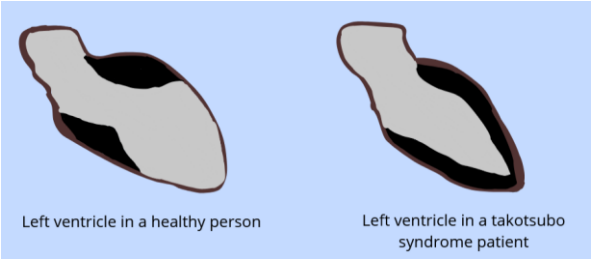

Fig. 2. Comparison of the appearance of the left ventricle (based on [1]).

Epidemiology

A precise determination of its incidence rate is not currently possible, due to the lack of sufficient knowledge among medical professionals and its similarity to acute coronary syndrome (ACS), resulting in the possibility that many cases may be missed and undocumented [12,13]. Continued medical advances, increasing awareness of the symptoms of this disease, and widespread access to invasive coronary angiography are increasing the percentage of accurate diagnoses. Clinical diagnoses are increasingly being reported on all continents, but are relatively rarely detected among Latin American and African-American populations [14,15]. According to the currently available information, the prevalence in the population is less than 2%. These data may be misleading due to an undersampled study group that is not representative of the entire population. In addition, studies conducted in one region may not be generalizable to other areas.

The vast majority of diagnoses, up to 90%, are in women aged 65–70. This syndrome is less frequently diagnosed in women below this age, as well as in men and children, including even premature babies. Few cases of TTS in men have been described in the literature, mainly associated with medical procedures such as bladder catheterization, gastroscopy, or knee joint arthroscopy. Men diagnosed with TTS are statistically younger than women, and in their case, physical triggers are more likely than in women, among whom emotional triggers are predominant. Cardiogenic shock and ventricular fibrillation occur more frequently in men, while chronic heart failure is most common in women. Japan is the only country where a higher percentage of the patients are men, although the reason for this finding has not been determined so far. Younger patients often have an atypical clinical picture characterized by the absence of comorbidities or neurological and psychiatric disorders; they are also less likely to develop complications. The occurrence in children was mainly related to mental illnesses, the use of psychoactive substances, and the development of sepsis. TTS described in the pediatric population presents a characteristic clinical profile, with a higher frequency of atypical symptoms and physical factors, as well as higher prevalence of cardiogenic shock and a similar mortality and recurrence rate to the adult population.

The frequency varies by gender as well as ethnicity, as it is less common in Latinos and African Americans, who more often experience complications such as respiratory failure or stroke, as opposed to Caucasians, in whom TTS is more commonly seen [13,16,17]. Increasing awareness among healthcare workers increases the number of accurate diagnoses in men, mainly residing in Japan, due to men's predisposition to physical rather than psychological stress [18,19].

In 2011, the Zurich Heart House, based in Switzerland, established the International Takotsubo Registry (ITR) to study clinical features, analyze study results, and determine the best options for treatment and prevention of this disease. Currently, it is studied at 26 research centers in Europe and the United States, including two in Poland: the 1st Department and Clinic of Cardiology of the Medical University of Gdańsk and the 1st Department and Clinic of Cardiology of the Medical University of Warsaw (MUW) [13,20]. Due to the limited availability of Polish patients, researchers from the center at MUW created a Polish registry of people diagnosed with TTS under the patronage of the Polish Cardiology Society's Heart Failure Association and the Interventional Cardiology Association of the

Polish Cardiology Society. The aim of the registry is to analyze the clinical profile of hospitalized patients in Poland. Data are provided by all hemodynamic laboratories affiliated with the Interventional Cardiology Association. The main facility is the Independent Public Central Clinical Hospital in Warsaw, which is the University Clinical Center MUW, while the coordinating center is the aforementioned 1st Department and Clinic of Cardiology at MUW, which described the first case of TTS in Poland in 2006 [21,22,23].

Etiology

In nearly one third of patients diagnosed with TTS, the triggering factor cannot be determined. Possible stress--related factors can be categorized as psychological or physical. The former may include strong emotions such as those connected with financial losses, arguments, domestic violence, or speaking before a large audience. Physical stressors classified as iatrogenic triggers include a fear of undergoing numerous medical procedures, which may be accompanied by unpleasant sensations such as varying levels of pain and merely staying in a medical facility [24,25]. Most stress factors are associated with discomfort, but not all. Positive stress (eustress) mobilizes and motivates the human body. This additional, albeit short-term, energy boosts activity. This interesting type of TTS is called the "happy heart syndrome," but unfortunately, it is not often described in the literature [26,27]. Gender also plays a role in the potential occurrence of this condition. Men react more strongly to physical events than women, who are naturally more sensitive and less resistant to situations causing a lot of emotions [28].

Pathophysiology

The syndrome was identified and classified 35 years ago by a cardiologist in Japan, but its exact pathophysiological mechanism is still unknown. It is suspected to be related to catecholamine cardiotoxicity, coronary artery spasm, microvascular dysfunction, and estrogen deficiency. A potential triggering system may also be excessive stimulation of the nervous system, changes in its functioning and structure, and changes in the balance and distribution of adrenergic receptors, including adrenaline and noradrenaline [29,30,31]. The most commonly discussed mechanism is the release of catecholamines due to stress and activation of the sympathetic autonomic part of the nervous system, which is responsible for preparing the body for defense or evacuation in case of danger. Endogenous catecholamines released by the body have a detrimental effect on the heart muscle, causing transient myocardial hypokinesis with local wall motion abnormalities [32].

Classification

Various types of stress-induced cardiomyopathies have been described in the specialist literature (Figure 3). We can divide them into primary TTS and secondary TTS. In the first case, the cause of psychological stress or the absence of such stress can be determined, so most people seek specialized medical help when they experience acute chest pain. The secondary form occurs much more frequently and is triggered by physical factors, such as an existing illness, past trauma, or current medical procedure. In these individuals, sudden activation of the sympathetic nervous system or a sudden increase in catecholamines may be the triggering factor [13].

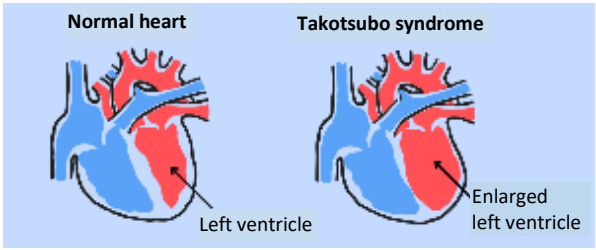

Fig. 3. Left ventricle (based on [34,37]).

Due to differences in anatomical presentation, we can distinguish four subtypes of typical and atypical cases, based on abnormalities seen in the angiogram of the left ventricle or echocardiographic deviations. Typical cases include the most common (81.7%) apical ballooning, also known as TTS, characterized by hypokinesis or dyskinesis of the apical and mid segments of the anterior, septal, inferior, and lateral walls of the left ventricle and associated with hyperkinesis of the basal segments. Atypical subtypes entail mid-ventricular, basal, and focal abnormalities. The mid-ventricular subtype (14.6%) is recognized when there is a ring-like hypokinesis or dyskinesis of the mid-ventricular segments, with normokinesis or hyperkinesis of the basal and apical segments. Patients with the basal subtype (2.2%) have hypokinesis or dyskinesis of the basal segments and normokinesis or hyperkinesis of the mid-ventricular and apical segments, resulting in left ventricular motion abnormalities outside the perfusion area of the coronary artery. The least common variant is the focal subtype (1.5%), which is diagnosed only in cases of focal hypokinesis or dyskinesis of any segment of the left ventricle according to laboratory, electrocardiographic, and clinical criteria [15,33,34].

Reversed takotsubo cardiomyopathy (rTTC) is also distinguished in the literature. It is one of the rarest types of stress-induced cardiomyopathies, characterized by hypokinesis of the basal and mid-ventricular

segments. It has similar pathophysiological causes but affects a different group of patients, as it is diagnosed in young people with reduced hemodynamic compromises. Patients with the reversed type of the syndrome present with chest pain, which may be accompanied by dyspnea and additional symptoms such as indigestion, abdominal pain, and syncope. There may also be moderate heart failure due to reduced ejection fraction and hypotension due to obstructive outflow. Treatment involves beta-blockers, angiotensin-converting enzyme (ACE) inhibitors, and angiotensin receptor blockers (ARBs). Proper identification of this type is important due to the potential negative health consequences [30,35,36,37,38].

Diagnostic criteria

Differential diagnosis is recommended with symptoms that are also present in ACS in populations at high risk of an episode. It is most commonly detected in postmenopausal women experiencing acute chest pain and respiratory symptoms, often associated with emotional stress and an abnormal electrocardiogram. However, such symptoms occurring in men should not be underestimated. Various laboratory, electrocardiographic, and imaging studies are used in the diagnosis. Coronarography is most commonly used to exclude coronary artery disease (CAD). Ventriculography can be used to assess left ventricular function (left ventricular apical dilatation is seen in TTS). In contrast, transesophageal echocardiography with longitudinal strain assessment and acoustic marker tracking allows a detailed assessment of myocardial function, including the degree of left ventricular dysfunction [2,39].

One of the most commonly used diagnostic criteria is the one proposed by the Mayo Clinic in 2004 and updated in 2008. The updated criteria include the four key symptoms reported by patients with suspected TTS: transient hypokinesis, akinesis, or dyskinesis of the mid-segments of the left ventricle with or without involvement of the apex; local abnormalities in contractility caused by a triggering factor; the absence of diagnosed CAD; and angiographic evidence of a myocardial infarction plaque rupture, new ECG abnormalities such as ST elevation or T wave inversion, and elevated troponin levels, with no evidence of a thrombus or myocarditis [40,41] (Table I).

The most up-to-date diagnostic criteria are those published by the European Society of Cardiology in 2018, which incorporate the latest medical findings based on a consensus between conflicting information. The International Takotsubo Diagnostic Criteria, known as the InterTAK diagnostic criteria, include the following eight points: transient left ventricular dysfunction with possible contractility abnormalities, a psychological or physical triggering factor, neurological disturbances, new ECG changes, elevated troponin and creatine kinase levels, elevated B-type natriuretic peptide, absence of infectious myocarditis, and menopause. Additionally, the coexistence of CAD is not an obstacle to diagnosing the syndrome [27,42]. Typically, patients present with textbook symptoms, but there is a group of patients in whom the ECG may be normal or their young age may raise doubts about the presence of this disease. These are challenges faced by clinicians on a daily basis.

Table I. InterTAK Diagnostic Score (based on [41])

Criteria	Points
Female sex	25
Emotional trigger	24
Physical trigger	13
Absence of ST-segment depression	12
Psychiatric disorders	11
Neurologic disorders	9
QTc prolongation	6
> 70 points – high probability of TTS	
≤ 70 points – probability of TTS	

TTS – takotsubo syndrome.

Differential diagnosis

TTS is a medical condition that is difficult to correctly identify due to its similarities in course and symptoms to many other cardiac diseases. Despite its resemblance to ACS, as mentioned above, it also shows similarities to various types of cardiomyopathy [43,44]. Dilated cardiomyopathy (DCM) is characterized by a progressive increase in the volume of the heart chamber with impaired contractility, causing symptoms that appear suddenly, such as ventricular arrhythmias or sudden death of the myocardium [45]. The opposite of DCM is non-dilated left ventricular cardiomyopathy (NDLVC), which does not meet all diagnostic criteria because the left ventricle has only subtle systolic dysfunction and its walls are not enlarged [46]. One of the diseases that should be differentiated from TTS is hypertrophic cardiomyopathy (HCM), which manifests as an asymmetrically thicker left ventricular wall (hypertrophy) and interventricular septum; in about 30% of people, there is also obstructed outflow of blood from the left ventricle, resulting in myocardial ischemia and rhythm disturbances. It is the most common cause of sudden cardiac death in people under 35 years of age living in the United States [47]. Another condition that can be confused with TTS is genetically determined arrhythmogenic cardiomyopathy, usually of the right ventricle, but the disorder can also affect the left ventricle, leading to arrhythmias and heart failure [48]. Restrictive cardiomyopathy (RCM) involves severe diastolic dysfunction with a limited ability to fill with blood. RCM can involve the left, right, or both ventricles, with typically normal to slightly increased

ventricular thickness, but atrial enlargement is characteristic due to the increased pressure in them [49]. The symptoms in each of these dysfunctions include heart failure, chest pain, and rhythm disturbances. Nevertheless, a detailed medical interview can facilitate the correct differentiation of the above-mentioned cardiomyopathies due to differences in imaging diagnostics and different clinical courses, leading to the correct diagnosis for a patient presenting with this condition.

Symptoms

In most patients, the clinical picture is indistinguishable from ACS due to the similar symptoms. Most patients report an initial symptom of shortness of breath or gradually increasing chest pain of a squeezing nature during rest, often accompanied by shortness of breath. The occurrence of chest pain resembling that in ACS is reported by about 70% of all patients. The presence of ST-segment deviations on the electrocardiogram and abnormal cardiac biomarkers should be indications for conducting a differential diagnosis of TTS. Currently, cardiac catheterization is the only method that can distinguish TTS from ACS and myocardial infarction with nonobstructive coronary arteries (MINOCA). Other reported symptoms include dizziness, syncope, or circulatory arrest. There is also a group of patients in whom such symptoms are not observed. The varying intensity of individual symptoms depending on the type of syndrome is intriguing. In patients with primary TTS, chest pain with associated vegetative symptoms – such as heart palpitations, rapid heart rate, and a feeling of tightness in the chest – is more frequently observed than syncope, cardiogenic shock, and palpitations, which are more commonly seen in patients representing the secondary type [21,50,51,52,53].

Risk factors

The main risk factor is often intensely stressful situations, divided into negative and positive events. Negative life events, such as the death of a loved one, divorce, or diagnosis of a serious illness (e.g., asthma, chronic obstructive pulmonary disease, or pancreatitis) are widely recognized factors. Positive strong emotions modulate the autonomic nervous system in a similar way to negative ones. Further research on the impact of positive emotions can lead to a paradigm shift in how we understand TTS [54,55]. Gender is an undeniable risk factor, as the majority of those affected are postmenopausal women; hormonal changes due to the lack of estrogen can significantly increase the likelihood of developing the disease. Currently, there are not enough reports related to genetic factors, but five cases of familial occurrence of this disease are well described. Two of them were mother-daughter relationships, while the other three involved sisters. The coexistence of neurological and psychiatric disorders can also predispose one to this syndrome [13]. Detailed descriptions of newly diagnosed patients show the possible multi-faceted nature of the disease, thus expanding the spectrum of factors that may predispose one to it.

Clinical course

The majority of patients have a transient nature of the disease, but they are still at risk of developing serious complications. In most patients, the disease is transient, but there is still a risk of serious complications from hospitalization, cardiac arrest, heart failure, cardiogenic shock, or the effects of comorbidities such as renal failure or stroke. In some patients, the disease may have an acute course, characterized by severe symptoms and cardiac dysfunction. In the acute phase, patients experience chest pain similar to that of a heart attack, difficulty breathing, and pulmonary edema. Relapse and long-lasting functional consequences such as depression and fatigue are also possible. The next phase is the recovery phase, in which symptoms gradually subside, and the left ventricular contractility returns to normal [24,56].

Prevention

To prevent a subsequent episode of TTS, it is essential to focus on the triggering factors. Lifestyle modifications and targeted interventions can play a crucial role in mitigating the risk. The primary emphasis should be on reducing exposure to chronic stress and incorporating relaxation techniques such as yoga and meditation, which have proven effective. Emotional stress can negatively impact the circadian rhythm, leading to difficulties in falling asleep, waking up at night, and making poor dietary choices. It is important to choose foods rich in unsaturated fatty acids, whole grains, and a variety of vegetables and fruits in order to create diverse and well-balanced meals, tailored to individual nutritional needs. The foundation of prevention should include balanced physical activity that is preferred and enjoyed, and avoiding any substances like alcohol or cigarettes.

Educating patients and their loved ones about the disease, its course, food-drug interactions, possible implications, and the importance of monitoring their body is crucial to achieving optimal outcomes and preventing future incidents. Likewise, a strategic approach should aim to optimize an individualized, coordinated treatment plan and minimize any adverse effects, such as controlling symptoms, ensuring quality of life, slowing progression, and preventing life-threatening complications like arrhythmias. Ethical considerations must guide the decision-making process, ensuring informed consent and respecting patient autonomy in therapeutic choices, especially

considering the unpredictable and uncharted effects of this disease in later years.

We should focus on previous comorbidities – monitoring test results and the body's response to medications and maintaining regular specialist visits – as they can exacerbate the disease's course. Nevertheless, regular screenings play a significant role in prevention, allowing for the early detection of abnormalities in the body. Appropriate coordination of healthcare plays a key role in minimizing risks and improving patient safety.

Treatment

In the treatment of TTS, medications commonly used for ACS are initially administered, but only after a proper differential diagnosis is symptomatic treatment introduced. It is also important to remember that CAD and TTS can often coexist, which should be considered when choosing pharmacotherapy. Various drug groups can reduce the likelihood of the disease recurring [34]. Diuretics and vasodilators are used in treatment. To reduce myocardial overload and control blood pressure, ACE inhibitors and ARBs are used. Intravenous administration of diuretics is associated with an increased mortality rate within 30 days, in contrast to ACE inhibitors, which were associated with reduced long-term mortality. ACE inhibitors should not be used in individuals with normal cardiac output, as this can result in impaired peripheral nervous system function related to low peripheral vascular resistance.

Given the likely mechanism related to catecholamine hyperstimulation, it is justified to use beta-blockers, which can improve blood flow through the heart, thus preventing patient death. In the analysis of an Italian registry of individuals diagnosed with TTS (the Takotsubo Italian Network), it was found that the use of beta-blockers was associated with a lower risk of death from all causes, including non-vascular diseases, though the probability of recurrence was comparable with or without beta-blockers. It should be noted that these drugs do not prevent the development of the disease, as more than 30% of people were regularly taking therapeutic doses due to other diseases [57,58,59]. ARBs and beta-blockers did not statistically correlate with patient mortality [60].

In patients with large areas of hypokinesis of the heart or reduced left ventricular contractility, anticoagulant therapy should be initiated. Aspirin acts both as an anticoagulant and an anti-inflammatory agent, inhibiting the production of prostaglandins and thromboxane, and reducing the concentration of inflammatory proteins in plasma. It was administered to 1,533 individuals out of 1,750 in the InterTAK registry. Comparing these patient groups led to the conclusion that the use of aspirin was not associated with a reduced risk of cardiovascular events after 30 days or after 5 years of patient observation. Additionally, the use of statins did not affect the short- or long-term possibility of serious complications and was not associated with lower mortality among the group using these drugs [61,62,63,64].

According to data and recommendations from a Swedish research registry, the majority of patients were treated with beta-blockers (77.8% orally and 8.3% intravenously) and antiplatelet drugs such as aspirin (66.7%). Less frequently, oral anticoagulants (11.3%), ACE inhibitors (55.5%), ARBs (15.3%), statins (55.1%), and diuretics (19.5% orally and 17.2% intravenously) were administered [15,60,65,66] (Table II).

Table II. Medications used in takotsubo syndrome management (based on [35,58,63])

Medication	Indication
Loop diuretics	Elimination of excess fluids
ACE inhibitors	Reduction of blood pressure
ARBs	Heart failure
Beta-blockers	Reduction of heart rate

ACE – angiotensin-converting enzyme; ARBs – angiotensin receptor blockers.

Currently, there are no established treatment guidelines due to the relatively rare occurrence, which significantly limits the possibility of large clinical trials, as well as the complexity of the pathophysiology, including the lack of a defined etiology of disease episodes. All actions taken to alleviate the course of the disease are based on the consensus of cardiology experts through a detailed analysis of previously described patients. The main goal of the therapy is to reduce serious brain and blood vessel damage, which can result in significant hemodynamic complications such as stroke and AMI.

In patients with TTS and comorbidities, the prevention and treatment of coexisting diseases should be continued until the patient is admitted to the hospital and treated for TTS. The condition for such therapy is an absence of adverse clinical reactions in the patient to whom it was administered. Therefore, it is necessary to continue administering medications for hypertension, heart failure, arrhythmias, diabetes, and hyperlipidemia, although their dosages can be adjusted according to the patient's current condition.

An episode of cardiomyopathy requires appropriate care from healthcare professionals. Such a patient should be hospitalized in a cardiology department equipped with the necessary facilities, and after discharge should be under cardiological observation, including preventive echocardiography to assess the degree of recovery and detect any complications of the disease. We cannot predict long-term effects. In many people, the heart returns to its normal function within a month, and routine ECGs only confirm this.

There is a possibility of TTS recurring in the same person. About 5% of patients experience a recurrence, most commonly within 3 months to 3 years of the first disease episode. A recurrence can occur at any age, in both women and men, even if it was first diagnosed in early childhood. A different triggering factor may be involved, which may also be a different anatomical type visible on imaging [40,58,67].

The prognosis is favorable, as left ventricular contractility returns to normal within 30 days of an episode and recovery soon follows. However, unavoidable complications may occur after the disease. The most commonly observed complications are arrhythmias, valve abnormalities, clot formation, pulmonary edema, and hypotension, which can lead to tissue hypoxia [68,69].

Invasive treatment of complications

ACS is the most serious complication of TTS and CAD. It is differentiated into ACS with ST-segment elevation (STEMI) and without ST-segment elevation (NSTEMI) on a 12-lead electrocardiogram. Unstable angina, where clinical symptoms suggest a heart attack but without biochemical evidence, is also included in ACS. Globally, STEMI currently accounts for 30% of all ACS cases, while NSTEMI accounts for 70%. The latter has a worse prognosis. STEMI poses an immediate life-threatening condition and requires the fastest possible intervention in the catheterization laboratory. Percutaneous reperfusion of the coronary artery within 120 minutes of symptom onset reduces mortality from 9% to 7%. In percutaneous coronary intervention (PCI), the use of drug-eluting stents or bare-metal stents is recommended for STEMI. Adjunctive pharmacotherapy is also very important. The European Society of Cardiology guidelines recommend the use of acetylsalicylic acid in the form of oral or chewable tablets at a dose of 150–300 mg and a strong P2Y12 inhibitor – mainly prasugrel or ticagrelor, possibly clopidogrel, in the case of primary PCI. Antiplatelet therapy should be accompanied by anticoagulant therapy. A class I recommendation drug is unfractionated heparin, and bivalirudin in patients with low platelet count. The patient's age, body weight, and comorbidities, such as stroke, should be considered so as to exclude possible adverse effects from individual drugs. Anticoagulant therapy should be continued for 12 months after the procedure. High-sensitivity troponin measurement is the preferred test for diagnosing NSTEMI. In high-risk patients, coronary angiography is recommended and percutaneous or surgical revascularization of the closed coronary artery should be performed within 24 to 48 hours to confirm the diagnosis. The surgical procedure is aortocoronary bypass (CABG), which involves creating a connection between the main artery – the aorta – and the remaining coronary arteries, bypassing the narrowing site. However, it is associated with a higher risk of stroke than PCI [70,71,72,73, 74,75,76,77] (Figure 4).

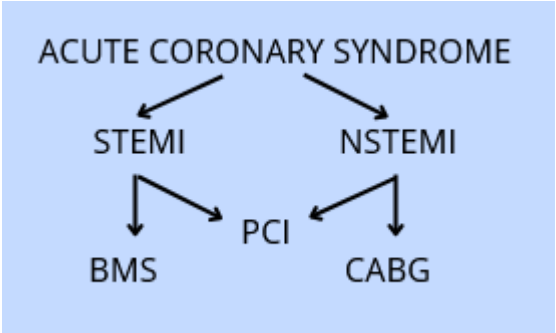

Fig. 4. Types of acute coronary syndrome (based on [73,74,75,77]). STEMI – ST-segment elevation myocardial infarction; NSTEMI – non-ST-segment elevation myocardial infarction; BMS – bare metal stent; PCI – percutaneous coronary intervention; CABG – coronary artery bypass grafting.

Ventricular arrhythmias, such as ventricular tachycardia – including torsades de pointes – are common electrophysiological disturbances that can be caused by TTS. Torsades de pointes is a type of ventricular tachycardia with a multifactorial etiology. It is characterized by electrolyte disturbances – hypokalemia and hypomagnesemia. The primary treatment for this disturbance is intravenous administration of magnesium, regardless of whether its level is reduced in the blood. In cases resistant to treatment, it is recommended to shorten the repolarization period by increasing the heart rate with isoproterenol. Stopping the heart's action or severe hypotension should be managed with electrical cardioversion. To improve outcomes in patients who have experienced an episode of ventricular arrhythmia, an implantable cardioverter-defibrillator (ICD) should be implanted. The device does not completely eliminate episodes of arrhythmia, but it often proves to be a life-saving therapy [78,79,80] (Figure 5).

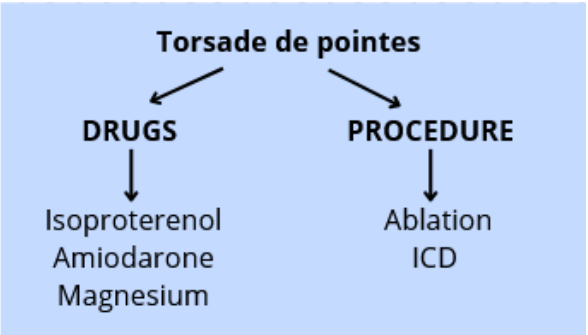

Fig. 5. Management of torsade de pointes (based on [80,81,82,83]). ICD – implantable cardioverter-defibrillator.

Treatment of ventricular arrhythmia initially involves the use of antiarrhythmic drugs, such as amiodarone,

which is considered the most effective antiarrhythmic in ventricular tachycardia pharmacotherapy. In the case of recurrent arrhythmia resistant to antiarrhythmic drugs, ablation therapy is the treatment of choice. It was once performed on an open heart, but there is now a safe, effective percutaneous method for treating rhythm disturbances. The procedure is performed in the electrophysiology laboratory and begins with electroanatomic mapping to detect the structure initiating the rhythm disturbance. The doctor then creates lesions with an ablation catheter inserted via the femoral artery that uses electrical current or low temperature. This disrupts conduction through the structure causing the rhythm disturbance, thereby eliminating the disturbance [81,82,83].

Clinical trials

There is a visible deficit of adequate scientific research on TTS treatment methods. Currently, two significant phase IV clinical trials are being conducted in Europe, studying the side effects caused over time by a new treatment method after its approval and introduction on the market. Due to the large scale of these studies, it is possible to detect the rarest side effects. Long-term side effects are monitored in different patient groups to assess their effectiveness in various clinical aspects. Both studies aim to evaluate the effectiveness of different pharmacological treatment methods and their impact on heart function and patient survival. The first study, named BROKEN-SWEDEHEART, or officially "Optimized Pharmacological Treatment of Broken Heart Syndrome (Takotsubo)," is being conducted in three Scandinavian countries: Denmark, Sweden, and Norway. It assesses left ventricular ejection fraction after 48–96 hours, the frequency of thromboembolic incidents and heart thrombosis, the need for heart-supporting devices within 30 days of a disease episode, and detailed patient examination results. The study focuses on the effects of adenosine, an antiarrhythmic drug, and dipyridamole, an antiplatelet drug. The second study is titled "Beta-blockers in Takotsubo Syndrome: A Randomized Clinical Trial (β-TAKO)" and is being conducted by hospitals in Spain. It concerns the effects of beta-blockers with alpha activity or nitric-oxide-releasing properties, which may improve left ventricular function. It assesses changes in left ventricular activity using the left ventricular ejection fraction method and global longitudinal strain. The systolic function of the left ventricle is analyzed using the echocardiogram wall motion score index over a period of 7 days. The results of these studies will provide valuable information on TTS management by comparing different therapeutic strategies and their impact on heart function and patient survival [84,85].

Conclusions

This review has presented the latest cardiological findings on TTS. In further research, it is important to not only to establish the exact pathophysiology of the disease and its long-term health effects after an episode, but also to determine why the disease primarily affects women. It is suspected that this may be due to the hormonal changes occurring after menopause. Ongoing research in this area is essential. By adjusting the pharmacotherapy, the risk of potential complications that reduce quality of life can be minimized. Another important element is to disseminate knowledge about this disease among specialists from various fields in order to enable proper diagnosis. Collaboration between a family doctor, cardiologist, gynecologist, and endocrinologist at the early stages of TTS can significantly improve prognosis and enhance the prospects of maintaining good quality of life in these patients.

Important limitations in the available studies were noted, such as the lack of long-term data, the limited number of studies involving men, and the small number of clinical trials. The paucity of data on long-term effects makes it difficult to assess the potential implications of the results in a broader clinical context. In addition, the dominance of studies on female populations leads to a gap in the knowledge regarding the effects of specific factors on men's health and limits the possibility of generalizing conclusions for both sexes. The small number of clinical trials further hinders the verification of the efficacy and safety of proposed solutions, which may limit their practical application.

Authors' contribution

Study design – W. Ficoń, M. Dobosz
Data collection – W. Ficoń, M. Dobosz
Manuscript preparation – W. Ficoń, M. Dobosz
Literature research – W. Ficoń, M. Dobosz
Final approval of the version to be published – W. Ficoń, M. Dobosz

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