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RIGHT VENTRICULAR INVOLVEMENT IN TAKOTSUBO CARDIOMYOPATHY

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ABSTRACT: Takotsubo cardiomyopathy (TTC) is a transient cardiac condition characterized by acute left ventricular dysfunction. While traditionally considered a left ventricular disorder, growing evidence suggests that right ventricular (RV) involvement is more common and clinically significant than initially thought. This review provides a comprehensive overview of RV involvement in TTC, focusing on its prevalence, pathophysiology, clinical presentation, diagnostic approaches, and management considerations. Recent studies estimate the prevalence of RV involvement in TTC at 25-50% of cases. The pathophysiology is complex, involving catecholamine surge, microvascular dysfunction, and ventricular interdependence. RV involvement is associated with worse clinical outcomes, including higher mortality and complication rates. Diagnosis relies on multimodal imaging, primarily echocardiography and cardiac MRI, along with biomarkers and hemodynamic assessment. Management of TTC with RV involvement requires a tailored approach, balancing supportive care with potential need for mechanical circulatory support. Challenges in diagnosis and management include differentiating from other conditions and limited RV-specific therapies. Special populations, such as the elderly and those with chronic lung disease, present unique considerations. Recent advances in imaging techniques and biomarker research show promise for improving early detection and risk stratification. Future perspectives include developing standardized diagnostic criteria, exploring targeted therapies, and establishing large-scale registries to advance our understanding of this complex condition. In conclusion, RV involvement in TTC significantly influences patient prognosis and management. Clinicians should maintain a high index of suspicion for RV involvement in all TTC cases and tailor their approaches accordingly. Continued research is crucial for enhancing our understanding and improving care for this patient population.

INTRODUCTION: Takotsubo cardiomyopathy (TTC), also known as stress cardiomyopathy or broken heart syndrome, is a transient cardiac condition characterized by acute left ventricular dysfunction. First described in Japan in 1990 by Sato *et al.* [1], TTC has gained significant attention in the cardiovascular community due to its unique pathophysiology and clinical presentation. While traditionally considered a left ventricular disorder, increasing evidence suggests that right ventricular (RV) involvement is more common and clinically significant than initially thought [2].

This review aims to provide a comprehensive overview of right ventricular involvement in Takotsubo cardiomyopathy, focusing on its prevalence, pathophysiology, clinical presentation, diagnostic approaches, classification, hemodynamic impact, prognostic implications, management considerations, and future perspectives. By examining the current literature and recent advances, we seek to enhance

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understanding of this important aspect of TTC and its implications for patient care.

Prevalence and Recognition: The prevalence of RV involvement in TTC has been underestimated for years. Initial reports focused primarily on left ventricular abnormalities, with right ventricular dysfunction considered a rare occurrence. However, recent studies have challenged this notion, suggesting that RV involvement is far more common than previously believed.

A meta-analysis by Pelliccia *et al.* in 2017 reported that RV involvement occurs in approximately 25-30% of TTC cases [3]. However, some single-center studies have reported even higher rates, with RV dysfunction observed in up to 50% of patients with TTC [4]. The growing recognition of RV involvement is attributed to several factors:

1. **Improved Imaging Techniques:** Advanced echocardiographic methods and cardiac magnetic resonance imaging (MRI) have enabled better visualization and assessment of RV structure and function [5].
2. **Increased Awareness:** As knowledge of TTC has expanded, clinicians have become more attuned to the possibility of RV involvement, leading to more frequent evaluation and documentation [6].
3. **Standardized Criteria:** The development of diagnostic criteria for RV involvement in TTC has facilitated more consistent reporting and comparison across studies [7].

It is important to note that the true prevalence of RV involvement may still be underestimated, as some cases may be missed due to the transient nature of TTC and variability in imaging practices across institutions.

Pathophysiology: The exact mechanisms underlying RV involvement in TTC are not fully understood, but several theories have been proposed. The hallmark catecholamine surge affecting both ventricles is a primary factor, with the RV potentially more susceptible due to its thinner wall [1]. Coronary microvascular dysfunction may extend to the right coronary system, while ventricular interdependence can lead to RV impairment secondary to LV dysfunction [2, 3]. Acute pulmonary hypertension resulting from left-sided failure can strain the RV, and diffuse myocardial edema may affect both ventricles [4]. Some researchers propose an autonomic nervous system imbalance as a contributing factor [5]. Understanding these pathophysiological mechanisms is crucial for developing targeted therapies and improving outcomes in patients with RV involvement in TTC.

Clinical Presentation: The clinical presentation of TTC with RV involvement can be diverse and may differ from cases with isolated left ventricular involvement. The clinical manifestations are shown in **Fig. 1.**

Common features include:

- **Dyspnea and Fatigue:** These symptoms are often more pronounced in patients with biventricular involvement compared to those with isolated left ventricular TTC [15].
- **Right-Sided Heart Failure Signs:** Jugular venous distension, peripheral edema, and hepatomegaly may be observed in cases with significant RV dysfunction [16].
- **Hypotension and Cardiogenic Shock:** Biventricular involvement is associated with a higher risk of hemodynamic instability and cardiogenic shock [17].
- **Chest Pain:** Similar to classical TTC, patients may experience acute chest pain mimicking acute coronary syndrome [18].
- **Syncope or Pre-Syncope:** Reduced cardiac output due to biventricular involvement can lead to cerebral hypoperfusion and syncope [19].
- **Electrocardiographic Changes:** While ST-segment elevation or T-wave inversion in precordial leads is common in TTC, patients with RV involvement may show these changes in right precordial leads (V1-V3) as well [20].

Arrhythmias: Both atrial and ventricular arrhythmias can occur, with some studies suggesting a higher incidence in patients with RV involvement [21].

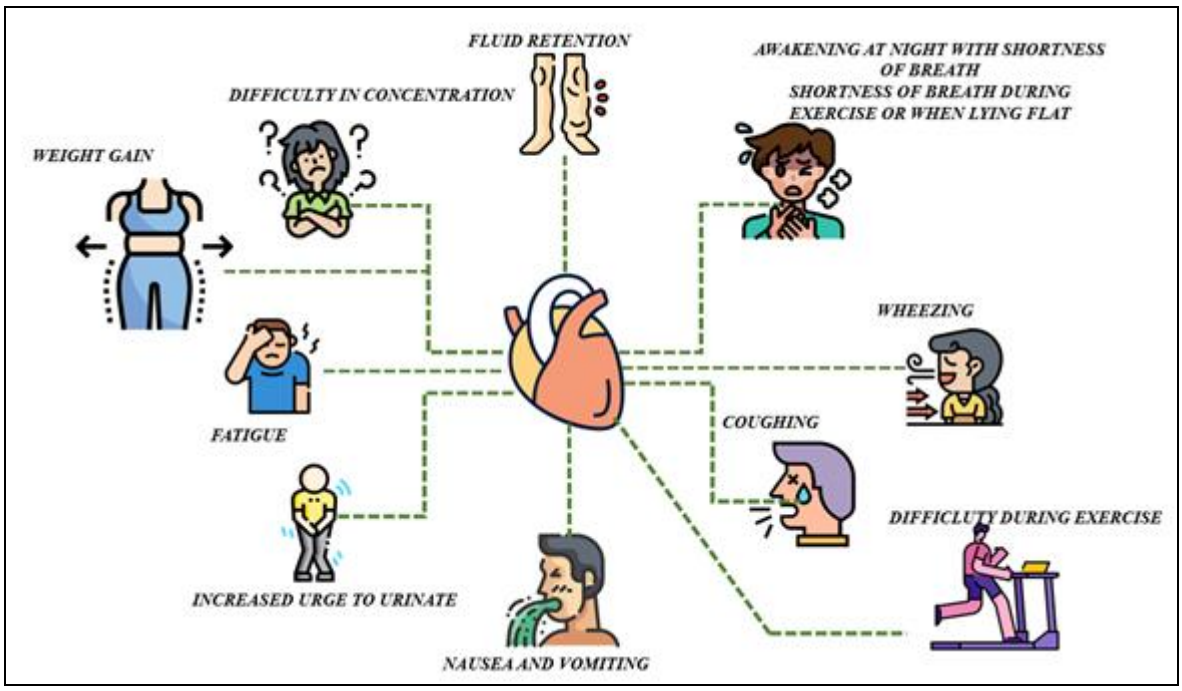

FIG. 1: CLINICAL MANIFESTATIONS OF RV INVOLVEMENT IN TTC

It's important to note that RV involvement may not always be clinically apparent and requires a high index of suspicion. Some patients may present with predominantly left-sided symptoms, and RV dysfunction may only be detected on imaging studies.

Diagnostic Approach: Diagnosing RV involvement in TTC requires a multimodal approach. Echocardiography remains the primary tool, assessing regional wall motion abnormalities, RV ejection fraction, TAPSE, and other parameters [6]. Cardiac MRI provides detailed evaluation of RV structure, function, and tissue characterization, including myocardial edema and fibrosis [7]. Coronary angiography is essential to rule out obstructive coronary artery disease [8]. Cardiac biomarkers, while not specific to RV involvement, may be more pronounced in biventricular cases [9]. Electrocardiography may show changes in right precordial leads, and hemodynamic assessment through right heart catheterization can reveal elevated right-sided pressures and reduced cardiac output [10, 11]. Nuclear imaging techniques, though less commonly used, can provide additional information on myocardial perfusion and metabolism [12]. The RV involvement in TTC diagnosed with different parameters is detailed in **Table 1** and **Fig. 2** illustrates TAPSE measurement.

TABLE 1: RV INVOLVEMENT IN TTC

Parameter	Echocardiography	Cardiac MRI	Normal Values	Significance in TTC
RV Ejection Fraction	Visual estimation, 3D echo	Short-axis cine images	>45%	<35% indicates significant RV dysfunction
TAPSE	M – mode measurement	4-chamber cine	>17 mm	<17 mm suggests RV systolic dysfunction
RV FAC	2D measurement	Short axis-cine	>35%	<35% indicates RV systolic dysfunction
S' Velocity	Tissue Doppler	Not applicable	>9.5 cm/s	<9.5 cm/s suggests RV systolic dysfunction
RV Free Wall Strain	Speckle tracking	Feature tracking	>-20%	>-20% (less negative) indicates RV dysfunction
RV Basal Diameter	2D measurement	4-chamber view	<41 mm	>41 mm suggests RV dilation
RVOT Fractional Shortening	2D or M-mode	Not routinely used	>30%	<30% indicates RVOT dysfunction
Tricuspid Regurgitation	Color doppler, CW Doppler	Phase contrast	Mild or less	Moderate-severe suggests RV dysfunction

IVC Diameter and Collapse	2D measurement	Coronal or axial views	<21 mm,>50% collapse	Dilated, non-collapsing IVC suggests elevated RA pressure
RV Wall Thickness	2D measurement	Short-axis view	<5 mm	>5 mm may indicate RV hypertrophy
Myocardial Edema	Not applicable	T2-weighted imaging	Absent	Presence indicates acute RV involvement
Late Gadolinium Enhancement	Not applicable	LGE sequences	Absent	Absence helps differentiate from MI or myocarditis

RV: Right Ventricle, TAPSE: Tricuspid Annular Plane Systolic Excursion, FAC: Fractional Area Change, RVOT: Right Ventricular Outflow Tract, IVC: Inferior Vena Cava, RA: Right Atrium, MI: Myocardial Infarction, LGE: Late Gadolinium Enhancement.

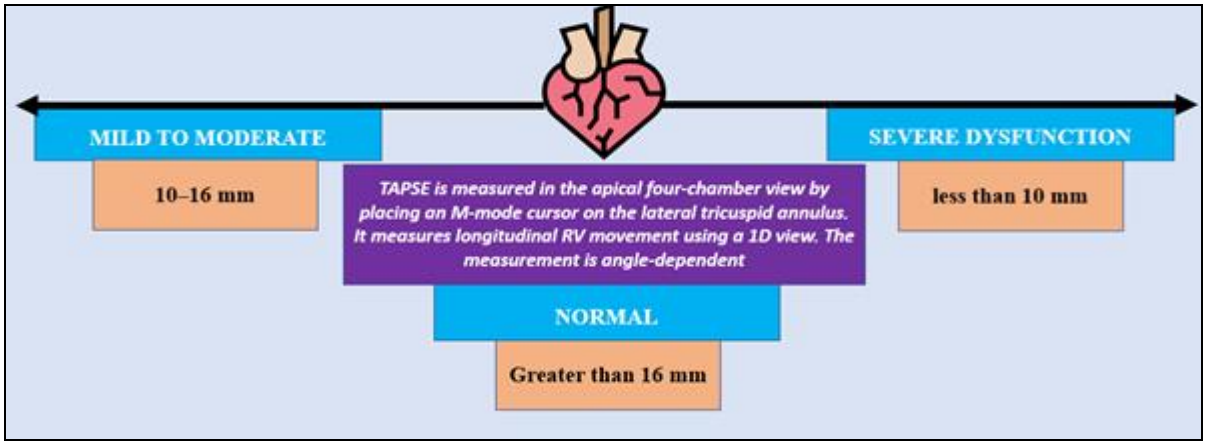

FIG. 2: TAPSE MEASUREMENT

Classification of Right Ventricular Involvement: RV involvement in TTC can be classified based on the extent and pattern of dysfunction and showed in **Fig. 3:**

1. **Isolated RV Involvement:** Rare cases where only the right ventricle is affected, with preserved left ventricular function [28].
2. **Biventricular Involvement:** Both left and right ventricles show characteristic wall motion abnormalities. This is the most common form of RV involvement in TTC [29].
3. **RV Sparing:** Classic left ventricular TTC without significant RV dysfunction [30].
4. **Focal RV Involvement:** Only specific segments of the RV (e.g., free wall or apex) are affected [31].
5. **Global RV Dysfunction:** The entire right ventricle shows reduced contractility [32].

Some researchers have proposed more detailed classification systems based on the combination of left and right ventricular involvement patterns, but these are not yet widely adopted in clinical practice [33].

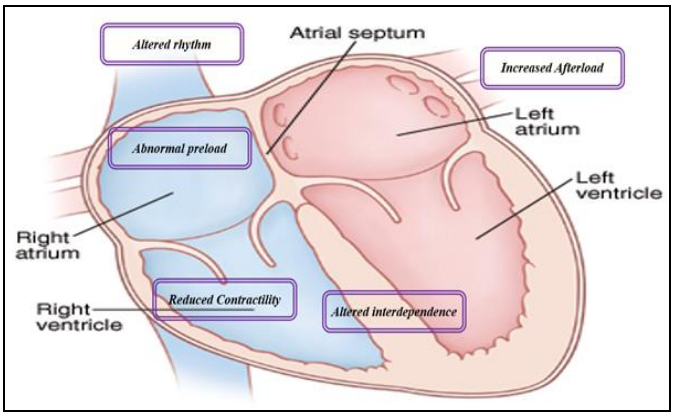

FIG. 3: INVOLVEMENT OF HEART CHAMBERS IN TTC

Hemodynamic Impact: RV involvement in TTC can have significant hemodynamic consequences. Biventricular dysfunction leads to a more pronounced reduction in overall cardiac output compared to isolated left ventricular TTC [13]. Elevated right-sided pressures can result in systemic venous congestion, affecting organ perfusion, particularly in the liver and kidneys [14]. Interventricular dependence can further impair left ventricular filling through septal shift, exacerbating the overall hemodynamic compromise [15]. Pulmonary hypertension, secondary to left ventricular dysfunction, increases RV afterload and

may worsen RV function [16]. Additionally, RV dilatation and dysfunction can lead to functional tricuspid regurgitation, further complicating the hemodynamic picture [17]. Understanding these alterations is crucial for appropriate management, particularly in cases requiring mechanical circulatory support.

Prognostic Implications: Several studies have demonstrated that RV involvement in TTC is associated with worse clinical outcomes. A meta-analysis found that RV involvement was associated with a 3.5-fold increase in short-term mortality [18]. Patients with RV involvement are more likely to experience complications such as cardiogenic shock, respiratory failure, and need for mechanical ventilation [19].

RV dysfunction is associated with prolonged hospitalization and increased resource utilization [20]. Some studies suggest a higher risk of TTC recurrence in patients with RV involvement, although data on this is limited [21]. While TTC is generally considered reversible, patients with RV involvement may experience a more prolonged recovery of ventricular function [22]. The impact on long-term outcomes remains an area of ongoing research, with some studies suggesting persistent subclinical cardiac dysfunction in a subset of patients [23].

Management Considerations: Managing TTC with RV involvement requires a tailored approach. Supportive care, including careful fluid management and treatment of precipitating factors, remains the mainstay [24]. Hemodynamic support may involve inotropes, although caution is advised given the potential role of catecholamines in TTC pathogenesis [25]. Severe cases may require mechanical circulatory support such as ECMO or biventricular assist devices [26]. Anticoagulation should be considered due to the risk of intracardiac thrombus formation [27]. Beta-blockers and ACE inhibitors/ARBs, while often used in TTC, should be administered cautiously in cases of significant RV dysfunction [28]. Pulmonary vasodilators may be beneficial in cases complicated by significant pulmonary hypertension [29]. Close monitoring and management of arrhythmias is essential, as is serial imaging to monitor ventricular function recovery [30].

Special Populations: Certain populations present unique challenges in RV involvement in TTC. Elderly patients have a higher likelihood of RV involvement and worse outcomes [31]. Those with chronic lung disease may have pre-existing pulmonary hypertension, predisposing to RV involvement and complicating management [32]. Postoperative patients may be at higher risk for TTC with RV involvement [33]. Some studies suggest a higher prevalence of RV involvement in TTC patients with underlying psychiatric conditions [34]. While rare, TTC can occur during pregnancy or the postpartum period, presenting unique management challenges in this population [35].

Challenges in Diagnosis and Management: Several challenges exist in diagnosing and managing RV involvement in TTC. Differentiating from conditions such as acute pulmonary embolism or right ventricular infarction can be difficult [36]. Limited RV-specific therapies and variability in imaging practices across institutions may lead to suboptimal management and potential underdiagnosis [37]. The transient nature of TTC means that the timing of assessment can significantly impact the detection of RV involvement [38]. Balancing hemodynamic support while avoiding excessive catecholamine stimulation is challenging, and the optimal duration and intensity of long-term follow-up remain unclear [39].

Recent Advances: Recent advances have improved our understanding and management of RV involvement in TTC. Novel imaging techniques, such as speckle tracking and 3D echocardiography, have enhanced detection and quantification of RV dysfunction [40]. Research into novel biomarkers, genetic susceptibility, and targeted therapies addressing catecholamine-induced cardiotoxicity shows promise for future management strategies. Artificial intelligence algorithms are being developed to improve early detection and risk prediction in TTC, including RV involvement.

Future Perspectives: Looking ahead, several areas warrant further investigation. Development of standardized diagnostic criteria for RV involvement in TTC is needed to facilitate consistent reporting and research comparisons.

Further studies into the pathophysiology, particularly at the cellular and molecular level, may identify potential therapeutic targets. Development of management protocols specifically addressing the unique hemodynamic challenges of biventricular TTC is crucial. Prospective studies to better understand long-term implications and potential preventive measures for high-risk populations are needed. Exploration of novel therapeutic approaches, including cell-based therapies and targeted molecular interventions, holds promise. Establishment of large-scale, international registries will facilitate data pooling and advance our understanding of this complex condition.

CONCLUSION: Right ventricular involvement in Takotsubo cardiomyopathy is a significant clinical entity that has gained increasing recognition in recent years. Its prevalence, estimated at 25-50% of TTC cases, underscores the importance of comprehensive cardiac evaluation in these patients. The complex pathophysiology, involving catecholamine surge, microvascular dysfunction, and ventricular interdependence, presents unique diagnostic and therapeutic challenges. RV involvement is associated with worse clinical outcomes, including higher mortality and complication rates, emphasizing the need for early recognition and targeted management strategies. Advances in imaging techniques, particularly echocardiography and cardiac MRI, have improved our ability to detect and characterize RV dysfunction in TTC. However, challenges remain in standardizing diagnostic criteria and optimizing management approaches. The hemodynamic impact of RV involvement necessitates careful consideration in therapeutic decision-making, particularly regarding fluid management and the use of inotropic support.

As our understanding of RV involvement in TTC evolves, several areas warrant further investigation. These include the development of RV-specific therapies, exploration of genetic and molecular mechanisms, and long-term follow-up studies to elucidate the lasting impact of this condition. The establishment of large-scale registries and collaborative research efforts will be crucial in advancing our knowledge and improving patient outcomes.

In conclusion, right ventricular involvement represents an important aspect of Takotsubo cardiomyopathy that significantly influences patient prognosis and management. Clinicians should maintain a high index of suspicion for RV involvement in all TTC cases and tailor their diagnostic and therapeutic approaches accordingly. Future research focusing on the unique aspects of RV involvement in TTC holds promise for enhancing our understanding and improving care for this complex patient population.

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