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Right ventricular dysfunction following tetralogy of Fallot correction: anatomical determinants and therapeutic strategies

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Abstract

Right ventricular dysfunction following surgical correction of tetralogy of Fallot (TOF) remains a major determinant of long-term morbidity and mortality in survivors. Despite advancements in surgical techniques, residual anatomical abnormalities – including pulmonary regurgitation, right ventricular outflow tract obstruction, abnormal coronary artery anatomy, scar formation, and tricuspid regurgitation – synergistically drive ventricular remodeling and functional decline. This review synthesizes evidence on the pathophysiological interplay of these anatomical substrates. Key imaging modalities, such as 3D late gadolinium enhancement cardiac magnetic resonance, and artificial intelligence tools enhance risk stratification for ventricular arrhythmias. We propose a hierarchical management framework prioritizing hemodynamic stabilization, electrophysiological substrate modification and individualized strategies for concomitant lesions considerations. This work aims to bridge anatomical insights with therapeutic innovations, offering a roadmap for improving longevity and quality of life in repaired TOF patients.

Keywords: anatomical determinants, integrated management, right ventricular dysfunction, tetralogy of Fallot

Introduction

Tetralogy of Fallot (TOF) is the most common cyanotic congenital heart disease (CHD), representing approximately 10% of all CHD cases[1]. Surgical correction, first pioneered in the 1950s, has transformed TOF from a fatal condition into one with a 30-year survival rate exceeding 90%[2,3]. However, this success is tempered by the long-term burden of right ventricular dysfunction (RVD), which affects 20–40% of patients within 10–20 years post-repair and remains a leading cause of morbidity and mortality[4,5]. Chronic RVD is associated with a 3-fold increased risk of reoperation (e.g., pulmonary valve replacement), a 15% lifetime risk of ventricular assist device implantation or heart transplantation, and a 10-year mortality rate of 12–18% in severe cases[3,6]. These outcomes underscore the critical need to understand the anatomical drivers of RVD and refine therapeutic strategies.

HIGHLIGHTS

- Synergistic anatomical factors drive RVD post-TOF repair, requiring integrated management.
- 3D-LGE-CMR and AI tools enhance arrhythmia risk stratification and RV dysfunction prediction in rTOF.
- Integrated management prioritizes hemodynamic stabilization, electrophysiological substrate modification, and individualized strategies.

Surgical interventions for TOF, while life-saving, inherently alter cardiac anatomy. Residual lesions – such as pulmonary regurgitation (PR), right ventricular outflow tract obstruction (RVOTO), abnormal coronary artery (ACA) anatomy, right ventricular (RV) scar formation, and tricuspid regurgitation (TR) – interact synergistically to exacerbate ventricular remodeling and dysfunction[7,8]. For instance, transannular patch repair, though effective in relieving RVOTO, predisposes patients to severe PR and subsequent RV dilatation[9]. Conversely, valve-sparing techniques reduce PR but may leave residual gradients that paradoxically mitigate volume overload[10,11]. Such complexities highlight the limitations of a one-size-fits-all approach and emphasize the importance of individualized management.

This review synthesizes contemporary evidence on the anatomical substrates of RVD in repaired TOF (rTOF) patients, focusing on their pathophysiological interplay and clinical implications. We further propose a hierarchical framework for managing multifactorial RVD, integrating hemodynamic stabilization, electrophysiological risk stratification, and patient-specific factors. By bridging anatomical insights with therapeutic advancements, this work aims to guide clinicians in optimizing long-term outcomes for this growing patient population.

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Anatomical abnormalities

Pulmonary regurgitation

While the surgical correction of TOF is necessary for saving lives, it can often result in PR as a long-term complication. The occurrence of PR after TOF surgery is dependent on various factors, with the surgical approach being a key determinant of pulmonary valve function. Patients who undergo transannular surgery are more likely to develop PR compared to those who undergo valve-sparing techniques[5]. In a retrospective study spanning 30 years, it was determined that opting for valve-sparing surgery could lower the chances of requiring a pulmonary valve replacement (PVR) by 78.6% and enhance survival rates by 9% in contrast to utilizing a transannular patch[9]. The decision to use a valve-sparing technique in TOF surgery is contingent upon the preoperative z-score of the pulmonary valve annulus. The pulmonary valve annulus z-score quantifies the deviation of the measured annular diameter from the population mean, normalized to body surface area (BSA). It is calculated using the formula:

$$z = \frac{\text{Measured diameter} - \text{Mean diameter for BAS}}{\text{Standard deviation for BAS}}$$

A z-score of –3 indicates a diameter 3 standard deviations below the mean. Across studies there is a wide range in the reported z-score for valve-sparing patients[12]. Based on our review of the literature, we have found a relatively definitive answer. It appears that patients with a z-score ranging from –3 to –1 are suitable candidates for valve-sparing procedures, while those with a z-score below –3.5 should opt for transannular surgery[13-15]. Moreover, the likelihood of PR following TOF surgery is inversely correlated with preoperative RV parameters (such as RV length diameter and RV short diameter) and directly correlated with pulmonary artery parameters (including the McGoon ratio, Nakata index, and left PA diameter)[16].

PR impacts both the global and regional functions of the RV. In terms of global RV function, PR can lead to the gradual enlargement of the ventricle, which in turn can hinder RV contractility and overall function[17]. The structural changes in the RV will result in a lengthening of the QRS duration, thereby raising the risk of ventricular arrhythmias[18]. A regional functional perspective shows that PR leads to a reduction in RV peak systolic strain rate and end-systolic strain, along with a decrease in RV outflow track (RVOT) ejection fraction[19,20]. Cardiac magnetic resonance (CMR) imaging is considered the gold standard for RV function assessment. The parameters such as RV end diastolic volume index (RVEDVi) >60 ml/m^2, abnormal interventricular septal (IVS) movement, and corrected RV ejection fraction for PR (RVcEF) <30% indicate RVD, prompting the need for PVR even in asymptomatic patients[21]. Artificial intelligence (AI) models, developed using a large dataset of electrocardiogram and CMR, have provided new tools for evaluating RV function. These AI models can accurately predict the risk of RVD and long-term survival rates in rTOF patients[22]. Additionally, "QRS fragmentation" has been identified as a specific high-risk feature in TOF patients[23].

PVR is an effective treatment for eliminating PR whether performed through open heart surgery or a transcatheter approach (Table 1)[24]. It helps reduce RV volume overload and improves the quality of life for patients[6,25]. PVR also reduces the volume load of the RV and shortens the QRS duration, with a potential decrease of 6 ms in the QRS interval after three years post-PVR, which may lower the risk of ventricular arrhythmias[26]. However, it is important to note that while PVR addresses the hemodynamic challenges of PR, it may not be able to reverse RV remodeling caused by myocardial fibrosis or scarring in the RV[27].

Residual RVOT obstruction

The chronic pressure overload experienced by patients with TOF prior to surgical correction leads to RV hypertrophy as the myocardium adapts to the increased workload[31]. The hypertrophied RV myocardium becomes less compliant and more susceptible to dysfunction, increasing the risk of RVD after TOF surgery[27].

The primary goal is to relieve RVOT stenosis to the greatest extent possible and fix the ventricular septal defect (VSD). However, complete elimination of RVOT stenosis is challenging, and some degree of residual RVOT obstruction (RVOTO) is often present[32]. Neonatal correction aims to minimize pressure overload and hypoxemia, but it can lead to more severe residual RVOTO, particularly with valve-sparing techniques[13,33]. If correction is performed before the first year of age the risk of residual RVOTO would be much lower[34]. The obstruction site may be valvar, sub-valvar, supra-valvar, or multi-level.

Long-term follow-up is necessary to determine if re-intervention is needed for residual RVOTO in rTOF patients. The RVOT gradient may initially increase after surgery but will eventually decrease and stabilize as the pulmonary valve annulus and RVOT continue to grow[35]. Research shows that the rates of freedom from RVOT re-intervention are 97%, 94%, and 91% at 1, 5, and 10 years after valve-sparing surgery, respectively[36]. Mild residual RVOTO is frequently seen in rTOF and may be advantageous for RV volume and function, therefore routine monitoring is advised instead of immediate intervention[10]. Residual RVOTO is considered significant when the peak systolic RVOT gradient exceeds 25 mmHg[37]. A moderate RVOTO pressure gradient of 25–50 mmHg may be beneficial in preventing adverse RV remodeling. The paradoxical protective effect of moderate residual RVOTO stems from its ability to reduce PR-induced volume overload while maintaining physiological RV afterload. Specifically:

- Pressure-volume balance: Mild to moderate RVOTO increases RV systolic pressure, which counteracts the diastolic volume overload caused by PR. This mitigates RV dilatation and preserves contractility[11].
- Ventricular interdependence: A moderately elevated RV pressure improves septal geometry, enhancing left ventricular filling and overall biventricular efficiency[38].

However, moderate RVOTO also carries risks for cardiovascular events, so it is crucial to consistently monitor and manage it appropriately to minimize these risks[38,39]. If the pressure gradient surpasses 50 mmHg, a severe RVOTO might develop, necessitating further intervention to avoid complications and improve cardiac function[13].

Abnormal coronary artery anatomy

The coronary artery anatomy in patients with TOF can significantly differ from those with normal heart anatomy, with

Table 1

The outcomes of PVR and TPVR from latest researches

Population	Application	Valve type	Outcomes	Reference
4513 patients, the median age of patients was 19 years.	Regurgitant native RVOT anatomy.	57% receiving a Melody valve and 43% a SAPIEN valve.	Acute success was achieved in 95% of patients: 95.7% in homografts, 96.2% in bioprosthetic valves, 94.2% in native RVOTs, and 95.4% in Contegra conduits. Major adverse events occurred in 2.4% of procedures, with higher rates in patients with homografts (2.9%) and native RVOTs (3.4%).	[28]
243 patients, median age of 31 years.	Native right ventricular outflow tract PR, majority had tetralogy of Fallot.	Self-expanding Harmony valve	Acute technical success was achieved in 99.6% of cases. Procedural serious adverse events occurring in 4% of patients.	[29]
316 eligible patients with TOF.	58 (18.4%) underwent pediatric PVR.	—	Patients who received pediatric PVR experienced a higher rate of cardiac hospitalizations, with a rate of 0.50 versus 0.09 hospitalizations per 20 years, resulting in a hazard ratio of 4.71. The rates of all-cause hospitalizations (HR = 0.95) and cardiac interventions (HR = 1.13) were comparable between those who received PVR and those who did not.	[30]

PR, pulmonary regurgitation; PVR, pulmonary valve replacement; RVOT, right ventricle outflow track; TOF, tetralogy of Fallot.

a notable prevalence of ACA anatomy in TOF patients. These anomalies, present in 2–9% of TOF cases, with 72% of them crossing the RVOT[40,41]. ACA can complicate surgical interventions and affect postoperative outcomes such as PR, residual RVOTO and arrhythmias[41].

The typical types of ACA include: left anterior descending coronary artery (LAD) arising from right coronary artery (RCA), RCA arising from left coronary artery, RCA arising from LAD, accessory LAD arising from RCA, and major conal artery arising from RCA[42]. When the critical ACA crosses the RVOT, it can complicate TOF correction surgery. Complex surgical techniques such as RV-PA tube implantation or translocation of the main PA anterior to the ACA may be needed to avoid damaging the ACA[40,43]. However, these techniques come with risks such as tube stenosis, infection, residual RVOTO, and the need for reoperation[40]. The relationship between the ACA and the pulmonary annulus are the main anatomical factor to consider when choosing the surgical technique[40]. Proper selection of the implantation site and tube orientation is crucial to prevent ACA compression and myocardial ischemia, which can negatively impact postoperative cardiac function[44], and potentially lead to death[45]. TOF patients with ACA are at higher risk of long-term HF if they show signs of myocardial ischemia after surgery[40].

To prevent harm to the ACA during TOF correction surgery, a transatrial + transpulmonary approach may be employed. This approach has demonstrated superior long-term outcomes in minimizing the chances of residual RVOTO and the need for reoperation[46]. Another effective method to prevent damage to the ACA is by translocating the main PA anterior to it. This approach is especially useful when the ACA is located precisely at the level of the pulmonary annulus, making both commissurotomy and transannular patch procedures unfeasible[40]. It is important to note that no matter which surgical techniques are employed, the RV incision should be positioned at least 1 cm away from the ACA in order to establish a sufficient "safety zone"[45,47].

Scar formation in the RV

During the operation, a right ventriculotomy is necessary to reach the RVOT and VSD, however, it results in scar formation as a normal part of the healing process[48]. Myocardium scar is less elastic and more fibrotic than healthy myocardial tissue, which can impair the RV functions and lead to ventricular arrhythmias[49]. Up to now, different factors that influence scar formation have been identified. Total scar extension was significantly higher in patients with previous shunt and reintervention, scar size showed a positive correlation with age at TOF surgery and duration of post-surgical follow-up[50].

A patch is often stitched into the RVOT to widen it and relieve obstruction, but it may also result in scar tissue formation. The extent of scar formation is directly related to the size of the patch. Moreover, excising hypertrophic myocardium from the RVOT can lead to scarring in the RV, and the scar frequently extends to the anterior RV free wall and neighboring segments[51]. Research has indicated that the size of focal scar in the RV of rTOF patients is linked to decreased RV systolic function, while those who have had a PVR tend to have a larger area of focal scar in the RVOT[52]. The impairment of RV systolic function was more pronounced in an experimental animal study when RVOT and transannular patches were used, as opposed to PR alone, primarily due to scarring from the patches[53].

Severe ventricular arrhythmias are a leading cause of death in rTOF patients, primarily due to the extensive ventricular scar resulting from surgery[54]. There is notable variability in electrophysiological characteristics between the RVOT and RV wall, potentially due to morphological distinctions. The RVOT is a crucial target for radiofrequency ablation of ventricular tachycardia[51]. Scar tissue in the RVOT, RV anterior wall, and surrounding the patch plays a significant role in predicting ventricular tachycardia in rTOF[55]. Therefore, the extent of scarring in the RVOT could serve as a marker for adverse outcomes and provide a substrate for malignant ventricular arrhythmias[56].

Surgical techniques such as right ventricular infundibulum sparing (RVIS) have been shown to reduce the extent of scar formation and preserve RV function. This approach minimizes the size of the ventriculotomy, thereby reducing the area of myocardial injury and subsequent fibrosis[57]. The use of antifibrotic agents, such as pirfenidone, has been explored to reduce

fibrosis. However, while these agents can decrease fibrosis, their impact on improving RV function remains uncertain[58]. In cases of severe RVD post-surgery, mechanical circulatory support may be necessary to maintain hemodynamic stability and allow for recovery of RV function[59].

Tricuspid regurgitation

TR is a common occurrence after TOF surgery, affecting approximately 13–25% of rTOF patients[60]. Damage to the tricuspid valve during surgery can be a cause of TR, especially when closing the VSD through an atrial incision[61]. However, advancements in surgical techniques have reduced the risk of tricuspid valve damage. RV dilatation rTOF is common and can lead to tricuspid annulus expansion, contributing to functional TR. The relationship between RV dilatation and TR is not straightforward, as not all patients with severe RV dilation exhibit significant TR, with only 22% of these patients having moderate to severe TR[62]. On the contrary, Some studies suggest that TR may exacerbate RV dilation[63]. Additionally, research indicates that the maximum right atrial (RA) area, rather than RV area, is a more reliable predictor of TR severity, highlighting the role of RA dilation in TR pathophysiology[64].

Severe TR is significantly associated with adverse events after PVR[65], leading to a debate on whether TR should be addressed during PVR surgery. Before the availability of transcatheter PVR (TPVR), the effectiveness of PVR in improving TR was uncertain. Studies on simultaneous tricuspid valve repair during open-heart PVR surgery did not show significant improvements in postoperative TR[60]. However, research on TPVR has shown that it can improve both RV function and TR severity in rTOF patients[66,67]. It is now believed that TPVR can be used in rTOF patients with severe TR. While some TR may improve on its own, the decision to intervene on the tricuspid valve should be based on postoperative follow-up. It should be emphasized that the presence of significant TR can influence the timing of PVR, potentially advancing the need for intervention by approximately 2.5 years, which may increase the number of procedures a patient undergoes over their lifetime[61].

There are still a few unanswered questions regarding TR in rTOF patients, such as the true mechanism of TR and which cases require surgical intervention. It is evident that monitoring TR during long-term follow-up in rTOF patients is crucial to promptly address any potential adverse events.

RVD in rTOF patients is not an immediate postoperative phenomenon but rather a progressive consequence of dynamic interactions among residual anatomical abnormalities. The interplay of volume overload (e.g., pulmonary regurgitation), pressure overload (e.g., residual RVOTO), and structural alterations (e.g., ventriculotomy-induced scar formation, ACA anatomy) collectively disrupts RV mechanics (Fig. 1). Critically, prior studies have predominantly focused on isolated anatomical factors, overlooking their synergistic contributions to RVD progression. Our work bridges this gap by systematically analyzing how these abnormalities – whether hemodynamic (pressure/volume mismatch), electrophysiological (scar-related reentry circuits), or morphological (coronary anomalies) – converge to drive RVD over time. This integrative perspective not only clarifies the pathophysiology of late-onset RVD but also underscores the need for multifactorial risk stratification and tailored interventions in rTOF management.

Ventricular arrhythmia: electrophysiology consequences of anatomical abnormalities

Studies indicate that rTOF patients who progress to RVD are at a higher risk of experiencing ventricular arrhythmias. This type of arrhythmia is worrisome because it can greatly affect both long-term survival and quality of life. A study found that 43.3% of rTOF patients had either sustained arrhythmias or needed treatment for arrhythmias[68]. Those who developed arrhythmias had an 8.6% higher mortality rate than those who did not experience arrhythmias[69].

The incidence of ventricular arrhythmia in rTOF patients with RVD is primarily linked to alterations in anatomical structure rather than to the anatomical factors themselves being independent[70]. Scarring from RV incisions, fibrosis related to the VSD and RVOT patch, ACA configurations, the RV-PA conduit, and early PVR can contribute to the remodeling of conduction pathways[71,72]. This remodeling includes prolonged RV activation time, slower conduction velocities, and increased collagen content, which collectively contribute to the formation of slow conduction anatomical isthmuses (SCAIs). These SCAIs are associated with an elevated risk of ventricular arrhythmias, particularly ventricular tachycardia (VT)[73-76]. Studies have shown that timely intervention for SCAIs can effectively prevent the development of VT, thereby reducing the risk of sudden cardiac death caused by VT in rTOF patients with severe RVD.

Identification of SCAIs

Electroanatomical mapping (EAM) is the gold standard for identifying SCAIs, allowing precise localization of slow conduction zones and aiding in ablation procedures[76]. However, this technique is invasive and requires specific equipment and expertise that may not be easily accessible in every clinical environment. 3D late gadolinium enhancement cardiac magnetic resonance (3D-LGE-CMR) accurately identifies SCAIs by detecting areas of high signal intensity, with a sensitivity of 100% and specificity of 90%[77]. This technique can significantly reduce the need for invasive EAM procedures by approximately 70%. Novel ECG algorithms, such as analyzing the R wave in V1 and the terminal QRS vector in aVF, have been developed to identify SCAI3 in rTOF patients[78]. The integration of 3D-LGE-CMR and innovative algorithms provides non-invasive techniques for identifying delayed RV activation linked to SCAIs. Additionally, incorporating EAM with 3D-LGE-CMR, isochronal late activation mapping (ILAM), and multidetector computed tomography (MDCT) enhances the precision of SCAIs diagnosis in individuals with rTOF[74,76,77,79] (Table 2).

Management strategies

Risk stratification: Proposed risk scores demonstrated only moderate efficacy in distinguishing between patients with and without SCAIs, with area under the curve (AUC) values below 0.7. 3D-LGE-CMR provides enhanced accuracy in identifying SCAIs in rTOF patients, achieving an AUC exceeding 0.8[77]. The novel ECG algorithms attained a sensitivity of 83% and specificity of 80% for SCAIs detection, with an AUC surpassing 0.87[78]. This advancement facilitates risk stratification for VT in rTOF patients, potentially enhancing outcomes by pinpointing individuals at elevated VT risk.

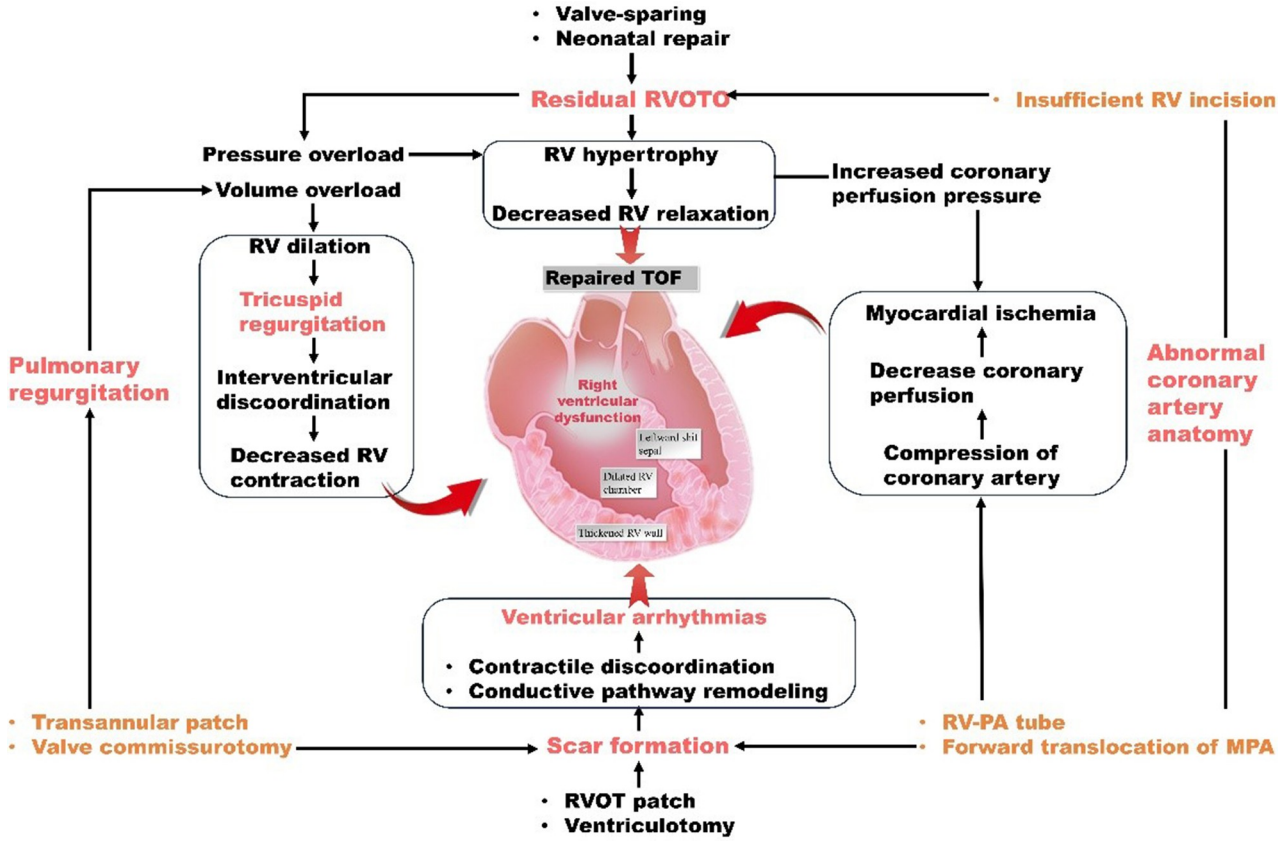

Figure 1. An illustration showing the anatomical-pathophysiological interactions leading to RVD in rTOF patients.

Prophylactic ablation: Prophylactic VT ablation targeting SCAIs was effective in diminishing the need for implantable cardioverter-defibrillator implantation and reducing subsequent arrhythmic occurrences, which encompass arrhythmic mortality, sustained VT, and cardiac arrest, with no ablation-related complications reported[76,80]. This suggests that the intervention was both safe and effective in mitigating VT in patients with rTOF.

Integrated management priorities in multifactorial RVD

Managing rTOF patients with concurrent anatomical abnormalities requires a personalized approach, focusing on interventions based on hemodynamic impact, arrhythmic risk, and long-term prognosis. This approach can help slow the progression of RVD, decrease the need for surgical reinterventions, and lower the risk of life-threatening arrhythmias. Below, we outline a systematic framework for making clinical decisions (Fig. 2).

Hemodynamic stabilization as the primary goal

Severe PR or residual RVOTO with a peak gradient >50 mmHg should be addressed first, as these conditions impose direct volume or pressure overload on the RV, accelerating ventricular remodeling and dysfunction. PVR remains the cornerstone for PR management, while RVOT reintervention (surgical or transcatheter) is indicated for significant RVOTO. In patients with both PR and RVOTO, mild-to-moderate RVOTO (25–50 mmHg) can paradoxically reduce RV dilatation by limiting the volume overload caused by PR[11]. This necessitates a careful balance in management, where the benefits of RVOTO in mitigating RV dilatation are weighed against the risks of pressure overload. Close monitoring with CMR imaging is advised to determine the optimal timing for intervention, ensuring that the benefits of addressing PR and RVOTO outweigh the potential risks[81].

Electrophysiological substrate modification

In rTOF patients with RVD who have a history of VT and extensive RV fibrosis, RV scarring, and hemodynamic tolerance, 3D EAM should be used to detect and identify SCAIs related to VT[82]. This information guides the implantation of an implantable cardioverter-defibrillator (ICD) and the use of antiarrhythmic drugs, along-side PVR. If VT is causing hemodynamic instability (e.g., LVEF < 35%) in these patients, VT substrate ablation should be performed to prevent VT, and prophylactic ICD implantation is also necessary to prevent sudden cardiac death[72].

Concomitant lesions considerations

Severity of TR: moderate-to-severe TR may improve after PVR due to RV reverse remodeling; thus, isolated TR without annular dilation can be monitored initially. Whether severe TR with annular dilatation (>40 mm) or right atrial enlargement requires surgical intervention depends on the PVR is an open-heart

Table 2

The methods used for detecting SCAI in rTOF patients

Population	Methods used	Findings	Clinical implication	Reference
55 consecutive patients with rTOF aged less than 30 years, median age of 15.8 years.	3D EAM and programmed electrical stimulation performed under sedation or anesthesia.	SCAIs was identified in 29% of patients. Factors associated with the presence of SCAIs included complex TOF variants, initial RV-to-pulmonary artery conduit placement, and ventriculotomy. 44% of patients with this substrate were inducible for VT.	The findings highlight the importance of early electrophysiological evaluation in young rTOF patients, particularly before PVR, to identify potential arrhythmogenic substrates that may become inaccessible after surgical interventions.	[74]
14 patients with rTOF, median age of these patients was 47 years. All patients had baseline SR, median QRS width was 170 ms.	3D-EAM used to create detailed maps of the heart's electrical activity, particularly during SR or RVOT. ILAM was performed to identify deceleration zones. Functional mapping was used to identify SCAIs by calculating conduction velocity and identifying deceleration zones.	ILAM accurately identified SCAI in patients with rTOF, SCAI was identified in 11 (40%) of patients and was related to VT inducibility.	This approach allows for precise targeting of SCAIs, which are critical for VT circuits, potentially leading to better clinical outcomes.	[76]
The derivation cohort consisted of 48 patients with a mean age of 34 years, the validation cohort had 53 patients with a mean age of 36 years.	RV EAM was performed to identify SCAIs.	In the derivation cohort, the sensitivity and specificity of 3D-LGE-CMR for identifying SCAIs were found to be 100% and 90%.	The ability to non-invasively identify SCAIs can aid in better patient selection for invasive procedures and improve overall management strategies for patients at risk of life-threatening arrhythmias.	[77]
All patients presenting with VT or undergoing planned PVR.	3D-LGE-CMR imaging to visualize myocardial scar and abnormal conduction areas. A specific signal intensity threshold was established based on comparisons between EAM findings and CMR data to differentiate between normal and abnormal myocardial areas.	In the validation cohort, these figures were slightly lower at 95% sensitivity and 91% specificity.		
130 patients aged more than 16 with rTOF were divided into two cohorts: a derivation cohort and a validation cohort, with 76 and 54 patients.	The study included a diagnostic algorithm combining R″ wave in V1 and negative terminal QRS portion (NTP) ≥80 ms in aVF for identifying SCAI3.	The study identifies a correlation between terminal QRS vector changes and SCAI3 in rTOF patients with RBBB. A diagnostic algorithm using R" wave in V1 and NTP ≥80 ms in aVF shows 74% sensitivity and 87% specificity for detecting SCAI3. In the validation cohort, the algorithm achieved 83% sensitivity and 80% specificity for identifying SCAI3.	The novel algorithm provides a non-invasive method for risk stratification of VT. It improves the accuracy of diagnosing SCAI3, which is a dominant VT substrate in rTOF patients, thereby aiding in better clinical decision-making.	[78]
A total of 61 patients with rTOF, with a mean age of 35 years.	MDCT studies were performed to measure anatomical isthmus dimensions associated with VT. EAM was performed to correlate MDCT findings with electrophysiological characteristics.	Calcification was identified in 75% of patients, allowing measurement of anatomical VT isthmuses. Catheter ablation was performed in 51% of patients targeting slowly conducting or narrow anatomical isthmuses	MDCT can effectively identify anatomical structures relevant to catheter ablation in patients with rTOF. This capability allows for a more detailed preoperative evaluation, assisting clinicians in understanding the anatomical landscape before transcatheter pulmonary valve placement, which is critical for planning safe and effective VT ablation procedures.	[79]
14 patients with rTOF, median age of 47 years.	Programmed electrical stimulation performed at the RV apex and RVOT to induce VT. High-density grid catheter was used to create 3D EAM of the RV during sinus rhythm. Automated ILAM was used to detect the SCAIs.	A total of 27 anatomical isthmuses were identified, with 11 classified as SCAI. The sensitivity and specificity of ILAM for identifying SCAIs were found to be 90% and 100%.	Automated ILAM effectively identifies SCAIs in rTOF patients, facilitating safer and quicker VT substrate characterization procedures.	[76]

rTOF, repaired tetralogy of Fallot; SR, sinus rhythm; EAM, electroanatomical mapping; SCAIs, slow conducting anatomical isthmuses; RV-PA, right ventricle-pulmonary artery; VT, ventricular tachycardia; PVR, pulmonary valve replacement; MDCT, multidetector computed tomography; 3D-LGE-CMR, 3D-late gadolinium enhancement-cardiac magnetic resonance; ILAM, isochronal late activation mapping.

procedure or a transcatheter approach. Severe TR may improve after TPVR[66,67]. If the patient is not a candidate for TPVR, then concomitant tricuspid valve repair is required as part of the PVR procedure[83,84].

ACA anatomy: open-heart surgical PVR is preferred over TPVR in patients with ACA crossing the RVOT, open-heart surgical PVR allows for direct visualization and preservation of the coronary artery, reducing the risk of compression to avoid coronary compression. The risk of coronary compression is a significant contraindication for TPVR in patients with ACA crossing the RVOT[85]. The rigid structure of the transcatheter device can compress the coronary artery, especially if the artery is in close proximity to the RVOT or conduit.

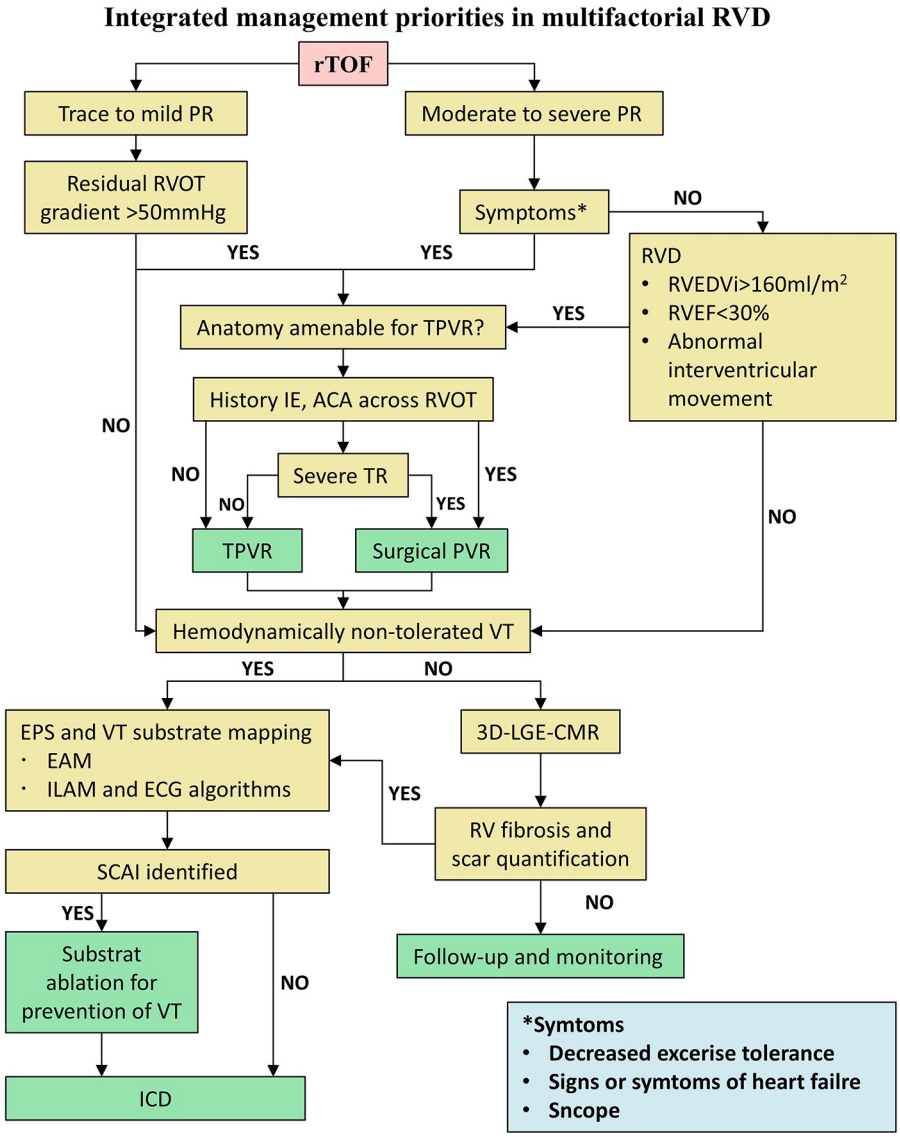

Figure 2. Multidisciplinary decision-making algorithm for multifactorial RVD in rTOF: integrating hemodynamic stratification, electrophysiological risk profiling, and precision therapeutics.

Individualized risk-benefit assessment

Patient-specific factors, including age, obesity (BMI ≥ 30 kg/m^2), exercise capacity (peak VO_2 ≤ 70%), and comorbidities (e.g., renal dysfunction), must refine decision-making[86]. For example, younger patients with preserved RV function (ejection fraction ≥ 50%) may benefit from early PVR to prevent irreversible fibrosis, whereas older patients with multiple comorbidities might prioritize symptom-directed therapy.

Conclusion

RVD remains a formidable challenge in rTOF patients, driven by a complex interplay of residual anatomical abnormalities. PR, residual RVOTO, scar formation, ACA anatomy, and TR collectively contribute to ventricular remodeling, arrhythmogenesis, and functional decline. Key advancements in imaging (e.g., 3D-LGE-CMR) and artificial intelligence have enhanced our ability to predict RVD progression and stratify arrhythmic risk, while transcatheter innovations like TPVR offer less invasive solutions for hemodynamic correction.

Future research must focus on refining risk prediction models, optimizing timing for reinterventions, and exploring antifibrotic therapies to reverse adverse remodeling. Collaborative efforts among surgeons, cardiologists, and electrophysiologists will be pivotal in translating anatomical insights into precision medicine. By embracing these principles, the goal of achieving near-normal longevity and quality of life for rTOF patients becomes increasingly attainable.

Ethical approval

The study and protocol were reviewed and approved by the Bioethics Committee of Sichuan University West China Hospital.

Consent

None.

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Author contributions

Q.F.: conceptualization, data curation, formal analysis, visualization, writing – original draft, review and editing; Y.W.: conceptualization, writing – review and editing; Q.A.: resources, writing – review; Y.L.: conceptualization, funding acquisition, resources, supervision, writing – review and editing, and project administration.

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