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RIGHT VENTRICULAR FUNCTIONAL AND ECHOCARDIOGRAPHIC EVALUATION IN PATIENTS UNDER FOLLOW-UP AFTER T-TEER

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ABSTRACT

Introduction

Severe tricuspid regurgitation is associated with reduced functional capacity and potential right ventricular impairment. In elderly or high-surgical-risk patients, transcatheter tricuspid valve repair with TriClip represents an important therapeutic option. Exercise stress echocardiography combined with the six-minute walk test (6MWT) may help assess functional and haemodynamic response after treatment.

Methods

Thirty-two patients with severe tricuspid regurgitation treated with TriClip were analyzed. In 2025, all patients underwent exercise stress echocardiography with concurrent 6MWT. Clinical, functional, and echocardiographic parameters were collected, including heart rate, left ventricular ejection fraction, pulmonary artery systolic pressure (PAPs), tricuspid regurgitation velocity (TRV), TAPSE, TAPSE/PAPs ratio, 6MWT distance, NYHA class, Borg score, and maximum workload. In 2026, a one-year follow-up was performed. Given the limited number of paired observations, longitudinal analyses were descriptive.

Results

The cohort was elderly, with a mean age of 79 years (range 63–94). At baseline, heart rate increased from 71 ± 11 bpm at rest to 118 ± 15 bpm at peak stress. Left ventricular systolic function was preserved, with an ejection fraction of 55 ± 1% at rest and 65 ± 2% after stress. Mean PAPs increased from 26 ± 7 mmHg at rest to 37 ± 6 mmHg at peak exercise, with corresponding TRV values of 2.46 ± 0.47 m/s and 2.86 ± 0.37 m/s. Mean TAPSE was 20 ± 3 mm. Mean 6MWT distance was 384 ± 70 m, with a Borg score of 12 ± 2, median NYHA class II, and maximum workload of 78 ± 8 W.

At follow-up, only 3 of 32 patients completed a comparable reassessment; 6 patients were unable to complete the stress protocol and were classified as clinically worsened, while 23 were lost to follow-up. Among the three patients with paired data, three different trajectories were observed: clinical and echocardiographic stability;

functional improvement with increased TAPSE and 6MWT distance; and deterioration with rising pulmonary pressures, reduced TAPSE, and a decline in the TAPSE/PAPs ratio, consistent with right ventricular–pulmonary arterial uncoupling**.**

Conclusions

In patients with severe tricuspid regurgitation treated with TriClip, exercise stress echocardiography combined with 6MWT may provide useful information on functional and haemodynamic response after treatment. One-year follow-up showed heterogeneous trajectories, although the high proportion of patients who were not reassessed or were lost to follow-up limits cohort-level conclusions. The TAPSE/PAPs ratio may represent a useful parameter for monitoring right ventricular–pulmonary arterial coupling after treatment.

SUMMARY

LIST OF ABBREVIATIONS

Abbreviation	**Definition**
6MWT	6-Minute Walk Test
BMI	Body Mass Index
COPD	Chronic Obstructive Pulmonary Disease
NYHA	New York Heart Association
PASP/sPAP	Pulmonary Artery Systolic Pressure
PVR	Pulmonary Vascular Resistance
RA	Right Atrium
RAP	Right Atrium Pressure
RIMP or MPI	Right Ventricular Index of Myocardial Performance
RV	Right Ventricle
RVOT	Right Ventricular Outflow Tract
RVRR	Right Ventricle Reverse Remodeling
TAPSE	Tricuspid Annulus Plane Systolic Excursion
TDI	Tissue Doppler Imaging
TEE	TransEsophageal Echocardiography
TEER	Transcatheter Edge-to-Edge Repair
TR	Tricuspid Regurgitation
TRV	Tricuspid Regurgitation Velocity
TTE	Trans-Thoracic Echocardiography
TV	Tricuspid Valve

INTRODUCTION

A. ANATOMY OF THE HEART AND THE TRICUSPID VALVE

The right ventricle is the most anteriorly located cardiac chamber, situated behind the sternum and delimited by the tricuspid valve annulus and the pulmonary valve(1).

The tricuspid valve is the largest of the cardiac valves and its function depends on the complex interaction between the annulus, leaflets, chordae tendineae and papillary muscles.

The Tricuspid Valve Orifice is located in the mid-anterior portion of the right atrial (RA) floor, with a mean circumference of 11.4 cm in men and 10.8 cm in women.(2)

The valvular orifice is surrounded by the Tricuspid annulus, a dynamic fibromuscular structure with a saddle-shaped spatial conformation. (3,4).

Normally the annulus contracts reducing its area to facilitate leaflet coaptation; however, in the setting of right ventricular or atrial dilation, the annulus flattens and dilates, predominantly along its lateral and posterior margins(3,4).

Although the tricuspid valve typically consists of three leaflets (anterior, posterior and septal), some patients present with more than three leaflets(3).

Four main types of tricuspid configuration can be identified: Type I, a valve with the classic three leaflet morphology; Type II, a two-leaflet morphology resulting from incomplete separation; Type III, a valve featuring an extra leaflet, and Type IV, characterized by the presence of five or more leaflets.(5)

Consequently, a thorough understanding of valve morphology is fundamental for the execution of edge-to-edge repair procedures(3)

Macroscopically the right ventricle features a distinct crescent shape and is divided into three components: the inflow tract, consisting of the tricuspid valve, chordae tendineae and papillary muscles, the myocardial apex and the outflow tract, a tubular structure that supports the pulmonary valve leaflets(6,7).

Unlike the left ventricle, the right ventricle has thin walls composed of deep and superficial muscle layers(8), in which intermediate layers are difficult to identify(6).

While this architecture allows for the adequate management of blood volumes at low pressure, in the presence of chronic volume overload, such as in cases of severe tricuspid regurgitation, the ventricle undergoes dilative remodeling, losing its contractile capacity(6).

The arterial blood supply to the right ventricle is continuous through both diastole and systole. It is provided by the right coronary artery and the conus artery, the latter being responsible for the perfusion of the infundibulum(7,8).

Despite their structural and functional differences, the two ventricles share the interventricular septum and they are enclosed by epimyocardial fibers within a semi-flexible pericardium. Consequently, the distension of one ventricle directly affects the distensibility and filling of the other. This concept is defined as ventricular interdependence(7,9,10).

B. RIGHT HEART FAILURE

1. Pathophysiology of right ventricular failure

For many years, cardiological research focused almost exclusively on left ventricular pathophysiology. It was only between the 1950s and 1970s that the importance of right heart function and its role in various cardiovascular diseases was recognized(1).

The primary function of the right ventricle is to receive systemic venous return and pump it into the pulmonary arteries(1). Being connected in series with the left ventricle, it must pump the same effective stroke volume(1). However, the right ventricle is connected to a highly distensible low-impedance pulmonary vascular resistance compared to the systemic circulation(1).

Consequently the right ventricular stroke work (defined as the product of stroke volume and mean pulmonary artery systolic pressure) is one-seventh that of the left ventricle, directly reflecting a mean pulmonary pressure that is proportionally lower than the systemic one(1,11).

For this reason, the right ventricle features thinner walls and greater compliance compared to the left ventricle; it is highly capable of accommodating volume variations but remains sensitive to acute increases in pressure(1).

Right ventricular function depends on three interconnected factors: contractility, afterload and preload.

Right ventricular dysfunction can arise from primary myocardial disease or secondary to an increase in afterload (pressure) or preload (volume)(12).

An impairment of pump function, and consequently contractility, can be caused by primary right ventricular cardiomyopathies, ischemic/infarction events or myocardial inflammatory processes. A reduction in right ventricular ejection fraction (indicator of myocardial contractility) is an independent predictor of decreased survival and major adverse cardiac events(13).

The workload imposed on the right ventricle during the ejection phase, known as afterload, is a critical determinant of its function(1).

Compared to the left ventricle, the right heart exhibits marked sensitivity to pressure changes, adapting less effectively to pressure overload than to volume overload. Pressure overload, which is evident in pathological processes affecting the pulmonary circulation and the left heart, is directly attributable to an increased afterload(12).

It is typically caused by pulmonary hypertension, which can be pre-capillary (idiopathic, secondary to lung diseases such as COPD or fibrosis, or pulmonary embolism) or post-capillary (associated with left heart diseases such as aortic or mitral stenosis, myocarditis or left ventricular dysfunction). Increased pulmonary vascular resistance elevates right ventricle wall stress: if the load increases slowly, the ventricle triggers hypertrophic adaptation mechanisms; however, if the increase is rapid or persistent, the ventricle undergoes dilation in an attempt to maintain output, ultimately developing right ventricular failure(14).

As a determinant of myocardial shortening, right ventricular preload represents the degree of diastolic stretch before contraction(1).

While the heart utilizes the Frank-Starling law to optimize output in response to increased volume, excessive loading can become detrimental. Due to the phenomenon of ventricular interdependence, a massive right ventricular dilation can mechanically compress the left ventricle, causing a significant decline in overall heart function(1). Pathological volume overload generally develops secondary to left-to-right shunts or severe tricuspid regurgitation(1).

In this context, tricuspid regurgitation is both a consequence and a cause of the progression of ventricular dysfunction, following the 'TR begets TR' paradigm: regurgitation imposes a chronic volume overload on the ventricle, which increases dilation and alters the geometry of the annulus, worsening the degree of tricuspid regurgitation(1,4). Interrupting the vicious cycle by restoring valvular competence, possible through edge-to-edge repair with TriClip, represents the pathophysiological rationale for arresting negative remodeling of the right chambers(1,4).

Regardless of the initial trigger, a vicious cycle is created in which each alteration exacerbates the others: volume overload causes right ventricular dilation(15,16). Since space within the pericardium is limited, the expansion of the right ventricle shifts the interventricular septum toward the left(15,16). This leads to a reduction in the compliance and contractile capacity of the left ventricle (interdependence). The reduction in left ventricular preload, combined with contractile impairment, causes a decrease in cardiac output and consequently systemic arterial pressure(15,16). Since coronary perfusion depends on the pressure gradient between the aorta and the ventricle itself, systemic hypotension reduces blood flow and thus oxygen supply to the right ventricular myocardium(15,16). This generates ischemia that further reduces right ventricular contractility. The reduced contractility of the right ventricle leads to upstream blood stagnation, causing systemic venous congestion(15,16). This damages peripheral organs (liver and kidneys), leading to multi-organ failure that aggravates the shock state(15,16).

In summary, pressure overload increases wall tension, favoring ischemia, which reduces contractility, which in turn increases dilation and volume overload, leading to reduced cardiac output and systemic hypotension due to the alteration of left ventricular geometry resulting from interventricular septal shift(15,16).

2. Epidemiology

Heart failure represents a public health concern, being a leading cause of mortality and hospitalization worldwide(17). Right heart failure is a clinical consequence of the right ventricle's inability to pump an adequate volume of blood into the lungs(18).

Estimating the epidemiological burden of this condition is difficult, but its causes are common(16). The clinical presentation and prognosis of right ventricular failure depend on the underlying cause and the presence of comorbidities(16).

The primary objective remains early recognition and risk stratification(16).

3. Diagnosis

The diagnostic workup relies on clinical history and physical examination: patients frequently present with symptoms overlapping with those of left heart failure (exertional dyspnea, anginal-like chest pain) and classic signs of intravascular volume expansion and elevated central venous pressures (jugular venous distension with a positive Kussmaul sign, bilateral dependent edema with a positive pitting sign, hepatomegaly, ascites and a loss of lean body mass)(19).

The diagnostic framework incorporates laboratory evaluations (complete blood count, atrial natriuretic peptide, renal and hepatic function; that highlights hyperbilirubinemia and altered cholestasis markers, secondary to congestive hepatopathy(19)), non-invasive investigations (electrocardiogram, echocardiography, cardiac magnetic resonance imaging) and minimally invasive modalities (cardiac catheterization)(19).

This comprehensive approach is essential to accurately characterize ventricular dysfunction and guide the selection of the most appropriate therapeutic strategy(16).

To capture the complexity of the patient's clinical presentation and assess its impact on daily life, the New York Heart Association (NYHA) functional classification stratifies patients into four severity groups, based on symptoms elicited by physical exertion(20,21).

C. TRICUSPID REGURGITATION

1. Epidemiology

In recent years, clinical and scientific interest in the ‘forgotten valve’’ has grown exponentially. Tricuspid Regurgitation (TR) is associated with increased hospitalization rates for heart failure and reduced survival(22,23). Although isolated surgical intervention on the tricuspid valve can resolve valvular incompetence, it remains a largely underutilized therapeutic option, due to the high in-hospital mortality associated with it(23).

Mild or trace tricuspid regurgitation is a relatively common and benign condition, whereas significant TR represents a pathology with a major epidemiological impact. According to the 2025 ESC/EACTS guidelines, significant TR has an age- and sex-adjusted prevalence of 0.55% in the general population: this percentage tends to increase in women and with advancing age, reaching 4% in individuals over 75 years (24).

Several studies have demonstrated that female and/or elderly patients with moderate or severe tricuspid regurgitation have a significantly higher mortality risk compared to patients with absent or mild tricuspid regurgitation(25).

The clinical determinants of Tricuspid Regurgitation include age, female sex, and Body Mass Index (BMI). Other predictive factors are: Atrial Fibrillation (AF), implantable devices, atrial or ventricular dilation, left heart disease.(26,27)

The prognosis of Tricuspid Regurgitation depends on the severity of the clinical case: the higher the grade of regurgitation, the greater the mortality rate(24).

2. Etiology

Tricuspid regurgitation (TR) is classified into two main categories based on its etiology. Primary tricuspid regurgitation is caused by congenital or acquired structural abnormalities that directly affect the valvular apparatus, such as chordal tethering or rupture, commissural fusion or leaflet fusion(28). The most frequent causes include infective endocarditis, rheumatic heart disease, congenital anomalies and iatrogenic injury(3).

Secondary or functional tricuspid regurgitation, present in 85% of cases, does not directly involve the valvular apparatus, which is anatomically normal, but stems from a geometric alteration of the right ventricle or right atrium(3).

Atrial functional regurgitation is triggered by atrial fibrillation and manifests with dilation of the right atrium and the tricuspid annulus, while right ventricular size and function are preserved(3).

Ventricular functional regurgitation is a consequence of a left-sided ventricular or valvular pathology, that causes dilation and dysfunction of the right ventricle; this subsequently leads to annular dilation and tethering of the valve leaflets, which fail to coapt properly(3).

Tricuspid Regurgitation (TR) can be classified also according to its severity. A multiparametric approach allows for an accurate assessment of the condition, categorizing it into mild, moderate, and severe grades. However, given the escalating mortality risk associated with worsening regurgitation, the classification has been expanded to include two additional grades, massive and torrential, to better differentiate the most extreme forms of the disease(29).

3. Diagnosis

Patients with mild or moderate tricuspid regurgitation (TR) are often asymptomatic; in severe cases they report fatigue and exertional dyspnea, due to reduced cardiac output.

On physical examination, they present signs of peripheral edema and abdominal distension, congestive hepatomegaly and ascites, jugular venous distension and holosystolic murmur(30,31).

The diagnostic workup proceeds with an electrocardiogram, echocardiography, cardiovascular magnetic resonance and cardiac catheterization with angiography(30,31).

The identification of advanced stages is fundamental, as these are strongly correlated with severe right ventricular dysfunction and a state of advanced congestive heart failure(22,30,31). In this scenario, the patient presents an elevated risk profile; therefore early diagnosis and proper risk stratification are strictly necessary to select the optimal therapy(22)

Echocardiography represents the fundamental diagnostic tool for the evaluation of the right ventricle and the diagnosis of tricuspid regurgitation. Historically, attempts were made to apply the same evaluation methods used for the left ventricle to the right ventricle; however, the irregular geometry of the latter severely limits their direct application(32).

To overcome these geometric constraints and avoid underestimation, a well-aligned RV-focused four chamber view is required. This approach allows for the accurate assessment of the linear dimensions, areas, volumes, wall thickness and the dimensions of the Right ventricular outflow tract (RVOT)(33). Linear RV measurements, evaluated at end-diastole, comprise the basal diameter, the mid-diameter and the longitudinal diameter(20,33–35).

Furthermore, quantifying wall thickness, utilizing 2D or M-mode imaging to enhance contrast in the four chamber view is fundamental for identifying right ventricular hypertrophy(20,35).

Moreover, it is necessary to evaluate the morphology of the interventricular septum: in case of right ventricular dilation, the septum flattens and acquires a D-shaped morphology(20,34).

Transthoracic echocardiography (TTE) provides crucial information regarding right ventricular systolic and diastolic function, by analyzing myocardial motion along the longitudinal axis.

The longitudinal shortening is evaluated using Tricuspid Annular Plane Systolic Excursion (TAPSE), obtained by placing the cursor in M-mode on the annulus in a four chamber view and quantifying the displacement relative to a fixed reference point during systole. A higher excursion corresponds to better right ventricular systolic function, with a normal value of >1.7 cm(20,35–37).

The S'-wave velocity, measured using Tissue Doppler Imaging (TDI) on the Tricuspid Annulus, represents the peak systolic excursion velocity. The higher the velocity, the greater the ventricular systolic function(20,35).

Additionally, global right ventricular performance can be integrated using the Myocardial Performance Index (MPI or RIMP): lower values suggest superior functionality(35).

Right Ventricular diastolic function is evaluated via pulsed-wave and Tissue Doppler, estimating transvalvular filling velocities in early diastole (E) and atrial systole (A), myocardial tissue velocities (E', A') and their respective ratios(20,35).

In addition to structural and functional parameters, echocardiography allows for hemodynamic assessment, including Right Atrial (RA) pressure, systolic Pulmonary Artery Pressure (sPAP) and Pulmonary Vascular Resistance (PVR).

sPAP corresponds to right ventricular systolic pressure, if there are no right ventricular outflow tract gradients. Values exceeding 35 mmHg in young adults and 40 mmHg in older adults indicate a pathological elevation(20,35,38).

However echocardiography frequently underestimates pulmonary pressures in cases of severe tricuspid regurgitation; thus, right heart catheterization is strongly recommended for all patients undergoing an intervention to accurately assess the

hemodynamic consequences on the right atrium and the systemic venous circulation(24).

To integrate hemodynamics and contractility, a key pathophysiological parameter assessable via echocardiography is right ventricular-pulmonary arterial (RV-PA) coupling, which describes the capacity of right ventricular systolic performance to adapt to a given pulmonary afterload while maintaining an adequate cardiac output. This coupling is estimated through the ratio of tricuspid annular plane systolic excursion to systolic pulmonary artery pressure (TAPSE/sPAP ratio). RV-PA uncoupling occurs when a significant increases in afterload can no longer be compensated for by the contractile reserve of the ventricle. The presence of clinical and echocardiographic signs of right ventricular dysfunction and secondary end-organ damage represents a key element in estimating the peri-procedural risk in patients with severe tricuspid regurgitation undergoing intervention(24).

While Transthoracic echocardiography (TTE) serves as the initial screening tool, transesophageal echocardiography (TEE) and three-dimensional imaging have proven essential in enhancing the study of valvular morphology and the quantification of the regurgitant orifice(31).

Furthermore, echocardiography is crucial for long-term follow-up; patients with moderate or severe tricuspid regurgitation must be monitored clinically and echocardiographically every 6 months(24). Imaging allows for an assessment of the ventricle to predict the long-term clinical outlook(24).

Specifically, the parameters derived from echocardiography, such as TAPSE, functional area change and longitudinal strain, enable a precise post-operative analysis of the right ventricle systolic function, documenting hemodynamic recovery and the cardiac response to the relief of volume overload(24). The landmark TRILUMINATE Pivotal trial confirms that the reduction of regurgitation achieved via TriClip system is associated with significant reverse remodeling of the right-sided heart chambers(39)

4. Treatment evolution: from medical therapy to surgery and transcatheter therapy

The treatment of tricuspid regurgitation has represented a complex clinical challenge for decades, in which the choice of therapy depends on the etiology and severity of the defect(24). Initially, tricuspid regurgitation was managed conservatively with medical therapy, aimed exclusively at controlling congestive symptoms (primarily through diuretics); however, this approach remained ineffective in correcting the underlying anatomical defect(24). Regarding the surgical approach, current guidelines indicate intervention on the tricuspid valve predominantly in concomitance with left-sided cardiac surgeries (aortic or mitral valve procedures), as it has been widely demonstrated that primary or functional alteration of the tricuspid valve doesn't spontaneously regress after correcting the left-sided lesion alone(24).

In the case of isolated tricuspid regurgitation, the approach varies: in severe tricuspid regurgitation without concomitant left-sided valvular disease surgical intervention is recommended for symptomatic patients with primary tricuspid regurgitation and should be considered for those with symptomatic secondary tricuspid regurgitation or asymptomatic patients with signs of right ventricular dilation or dysfunction(24).

Despite the increase in procedural volume over time, isolated tricuspid valve surgery has historically been burdened by a high in-hospital mortality rate (8-10%), making it a high-risk option(24,40).

Based on the scientific evidence, interest in transcatheter correction techniques for tricuspid regurgitation has increased, following the pathway already established for aortic and mitral valvopathies. The TriClip system (Abbott) emerges as the first device enabling transcatheter edge-to-edge repair (TEER) on the tricuspid valve(41).

Technologically derived from the MitralClip system, a percutaneous treatment now incorporated into European and American recommendations, the device has been specifically optimized to treat the tricuspid valve(41).

The procedure, performed percutaneously via femoral venous access under transesophageal echocardiographic and fluoroscopic guidance, consists of grasping the valve leaflets (frequently the septal and anterior or posterior leaflets). This transforms

the single valvular orifice into a multiple orifice, thereby reducing the regurgitant area(39,41).

The efficacy and safety of this device have been established by the TRILUMINATE clinical trial program. Following an initial prospective study on 85 patients with symptomatic moderate tricuspid regurgitation, which demonstrated its feasibility(41), the international randomized trial (TRILUMINATE Pivotal Trial) demonstrated the efficacy and safety of the device in patients with severe, symptomatic tricuspid regurgitation(39). Furthermore, the study demonstrated that the repair was associated with reduction in the degree of regurgitation and an improved quality of life. Follow-up data have confirmed the sustained clinical benefit and the stability of the device over time, highlighting low mortality and high safety at the 2-year mark(42).

Based on this information, the purpose of this study is to evaluate the long-term functional and echocardiographic outcomes in patients under follow-up after tricuspid edge-to-edge repair.

METHODS

A. STUDY DESIGN AND OBJECTIVES

The present research is configured as a monocentric, longitudinal, observational clinical study. The integrated clinical pathway involved Hesperia Hospital for the execution of the transcatheter tricuspid valve edge-to-edge repair (T-TEER) procedure and peri-procedural management and the Cardiology Department of University Hospital of Modena (AOU Policlinico di Modena) for the diagnostic work-up and long-term clinical and instrumental follow-up.

The primary objective of this study is to quantify the long-term functional and echocardiographic changes in a cohort of patients undergoing tricuspid valve edge-to-edge repair (TEER) using the TriClip system.

The research analyses variations in systolic pulmonary artery pressure (sPAP) and the associated risk of pulmonary hypertension, and evaluates the occurrence of early right ventricular reverse remodeling (eRVRR) through specific echocardiographic parameters.

The target population for this study was selected using a convenience sampling method.

The group consists of patients with severe, massive or torrential tricuspid regurgitation, presenting with signs of right-sided heart failure who underwent transcatheter edge-to-edge (TEER) for tricuspid regurgitation, in accordance with the official clinical indications of the Heart Team.

The inclusion criteria applied in this study were:

- Age ≥ 18 years
- Echocardiographically documented diagnosis of severe or superior (massive/torrential) tricuspid regurgitation;
- Presence of right-sided heart failure symptoms (peripheral congestion, ascites, congestive hepatopathy) and functional limitation (NYHA functional class I-IV);

- Availability of a resting echocardiographic evaluation and at least one functional test (either a Six Minute Walking Test or stress echocardiography) performed at both baseline and follow-up

The exclusion criteria for the study included:

- Failure to provide informed consent for the use of personal clinical data for research purposes;
- Presence of conditions that impair the interpretability of functional tests, such as severe motor, orthopedic or neurological limitations;
- Poor echocardiographic acoustic window quality, rendering it unsuitable for a reliable quantification of key parameters at rest and at peak stress.

B. CLINICAL PROCEDURES PROTOCOL AND DATA COLLECTION

Data collection for research purposes was conducted by retrieving information from electronic medical records, as well as outpatient and inpatient documentation from the participating centers. The data sources comprised reports and digital imaging archives from both resting and stress echocardiography and reports from the six minutes walking test (6MWT) with vital parameters.

Where available in the patients' medical history, the clinical pathway included the serial repetition of resting and stress echocardiographic examinations and the 6MWT.

The observation period ranged from 1 year, depending on the availability of scheduled follow-up visits.

Within this long-term follow-up framework, the patient cohort underwent stress echocardiography and the 6MWT; the protocols and parameters of which are detailed below.

1. Echocardiography

Stress echocardiography is an established imaging technique used for the diagnosis and prognosis of valvular heart diseases as well as for evaluating the stress response in patients with cardiovascular pathologies(43).

For patients capable of performing an exercise test, physical exercise stress testing, utilizing treadmill or a bicycle ergometer, is recommended over pharmacological stress testing with dobutamine or vasodilators, because exercise capacity serves as potent predictor of clinical outcomes(44).

In this study, exercise echocardiography is performed using a dedicated bicycle ergometer in a semi-reclined position.

A symptom-limited protocol is adopted: patients are instructed to pedal at a constant cadence of 60 revolutions per minute against an initial workload of 25 watts, which is increased by 25 watts every 2-3 minutes(44).

Throughout the test, echocardiographic images are captured at rest and immediately at peak exercise during the recovery phase. Continuous electrocardiographic monitoring and blood pressure measurements are performed at each workload increment.

The entire examination lasts 30 minutes, but it could be prematurely terminated upon reaching the age-predicted maximal heart rate or muscular exhaustion, joint pain or the onset of symptoms and clinical signs(44).

The parameters evaluated during the study are:

- Clinical parameters: resting and peak exercise heart rate (bpm); resting and peak systolic and diastolic blood pressure (mmHg);
- Exercise tolerance parameters: maximum workload achieved (Watt); presence of baseline symptoms and the type of clinical response to exercise; occurrence and specific indication for premature test termination;
- Right ventricular morphology and function parameters: baseline and post-stress right ventricular ejection fraction (RVEF,%); tricuspid annulus plane systolic excursion (TAPSE, expressed in mm); post-stress right ventricular end-systolic volume (RVESV, expressed in mL);

- Tricuspid valve and pulmonary circulation parameters: indexed diastolic tricuspid annulus diameter, severity grade of residual tricuspid regurgitation (TR), baseline and exercise peak tricuspid regurgitation velocity obtained via continuous-wave Doppler (TVR, expressed in m/s); estimated baseline and peak systolic pulmonary artery pressure (sPAP, mmHg) and sPAP variation (ΔsPAP= peak sPAP – baseline sPAP);
- Right ventricular-pulmonary arterial coupling parameters: the hemodynamic ratio between TAPSE and sPAP at baseline (expressed in mm/mmHg)

2. Six-Minute Walking Test (6MWT)

The objective evaluation of global functional capacity is performed using the six-minute walking test (6MWT), conducted in strict accordance with the American Thoracic Society (ATS) guidelines(45).

Patients are instructed to walk along a flat corridor at their maximum possible pace to record the total distance covered, expressed in meters, over a six minute period(45).

At the end of the test, patients are questioned regarding their perceived exertion and any symptoms that arose during the walk.

If a patient needs to stop, rest or slow down, the timer is maintained running continuously and both the final distance covered and the duration of the pause are recorded(45).

The specific outcomes evaluated through this test include:

- Total distance covered (expressed in meters);
- NYHA functional class of the patient at the time of the follow-up control;
- Severity of dyspnea or fatigue perceived at the end of the walk, quantified using the Borg Scale(46);
- Peak heart rate achieved during the test (bpm);
- Presence of cardiac symptoms during exertion (including angina, worsening dyspnea, palpitations or dizziness);

- Premature test termination before the expiration of the six-minute time limit;
- Improvement in functional performance evaluated in relation to the dynamic parameters recorded during the corresponding stress echocardiography session.

C. STATISTICAL ANALYSIS

The collected data are systematically analyzed.

Categorical variables are expressed as absolute frequencies (n).

Continuous variables are presented as mean and standard deviation (SD) for normally distributed data or as median with the corresponding maximum and minimum values for non-normally distributed data.

Regarding the longitudinal analysis, given the limited number of paired observations, as only 3 of 32 patients completed the full one-year follow-up protocol, results are presented descriptively. No inferential statistical testing was performed for group comparisons between baseline (2025) and follow-up (2026).

The high proportion of patients who did not complete the re-evaluation constrained the ability to draw statistically significant conclusions for the entire group.

For the patients who completed the follow-up, the evolution of echocardiographic, hemodynamic and functional parameters was analyzed by assessing absolute individual variations (Δ).

This approach enabled the identification and description of distinct clinical and hemodynamic response profiles (stability, improvement or deterioration) and the monitoring of specific markers, specifically the TAPSE/sPAP ratio, as an indicator of right ventricular-pulmonary arterial (RV-PA) coupling

Given the study's aim to generate real-world evidence, a convenience sampling approach is adopted, corresponding to the number of eligible patients available during the observation period. Missing data are reported as such, without the application of any imputation techniques.

For incomplete functional tests (e.g. a prematurely terminated six-minute walking test), the value or distance achieved at the time of interruption is considered valid for the analysis, in strict accordance with the technical procedure.

RESULTS

A. BASELINE COHORT CHARACTERISTICS (2025)

A total of 32 patients underwent exercise stress echocardiography with concurrent six-minute walk testing (6MWT) at baseline in 2025. The cohort was elderly, with a mean age of 79 years (range 63–94). Resting heart rate averaged 71 ± 11 bpm and increased to 118 ± 15 bpm at peak stress. Left ventricular systolic function was preserved in all patients, with a resting ejection fraction of 55 ± 1% rising to 65 ± 2% after stress, consistent with a normal contractile reserve.

Resting pulmonary pressures were normal to mildly elevated. Pulmonary artery systolic pressure (sPAP) at rest averaged 26 ± 7 mmHg (median 24, range 18–52 mmHg) and rose to 37 ± 6 mmHg (median 36, range 22–55 mmHg) at peak exercise, with corresponding tricuspid regurgitation velocities (TRV) of 2.46 ± 0.47 m/s at rest and 2.86 ± 0.37 m/s at peak. Right ventricular (RV) longitudinal function, assessed by TAPSE, averaged 20 ± 3 mm (range 16–28 mm). Functionally, patients covered 384 ± 70 m on the 6MWT (range 200–500 m) with a Borg score of 12 ± 2, the median NYHA class was II (range I–III), and the maximum achieved workload was 78 ± 8 W. Summary statistics are reported in *Table 1* and per-patient baseline values for the full cohort in *Table 3.*

Table1: Clinical and echocardiographic characteristics of the cohort at baseline (2025) and summary at follow-up (2026): the table summarizes the mean values and standard deviations for hemodynamic and functional parameters of the entire initial cohort and of three patients, who completed follow-up

Descriptive statistics of data for patients in 2025 cohort and patients who completed follow-up in 2026					
	2025			**2026**	
Parameter	**n**	**Mean ± SD**	**Median [min–max]**	**n=3 and value**	**mean**
HR rest (bpm)	29	71 ± 11	73 [50–96]	84, 68, 65	72
HR max (bpm)	29	118 ± 15	115 [100–150]	105, 110, 121	112
EF% rest	29	55 ± 1	55 [52–55]	55, 55, 55	55
EF% post-stress	29	65 ± 2	65 [57–65]	65, 65, 65	65
sPAP rest (mmHg)	30	26 ± 7	24 [18–52]	23, 25, 31	26
sPAP max (mmHg)	29	37 ± 6	36 [22–55]	38, 49, 50	46
TRV rest (m/s)	30	2.46 ± 0.47	2.40 [1.74–4.00]	2.00, 2.50, 2.78	2.43
TRV max (m/s)	29	2.86 ± 0.37	2.80 [1.94–3.53]	2.80, 3.80, 3.53	3.38
TAPSE (mm)	30	20 ± 3	20 [16–28]	22, 29, 19	23
Max workload (W)	29	78 ± 8	75 [75–100]	100, 75, 100	92
6MWT distance (m)	29	384 ± 70	400 [200–500]	425, 520, 520	488
6MWT Borg	29	12 ± 2	12 [9–15]	13, 13, 15	14
NYHA class	30	2 ± 1	2 [1–3]	1, 1, 2	1

B. FOLLOW-UP DISPOSITION (2026)

At one-year follow-up in 2026, only 3 of the 32 patients (ID 1, 4, and 16) completed a comparable stress echocardiographic and 6MWT re-assessment. Six patients (ID 3, 5, 7, 11, 15, and 17) were unable to complete the follow-up stress protocol and were classified as clinically worsened; of these, three (ID 3, 7, and 15) had successfully performed the test in 2025, indicating a genuine loss of exercise tolerance over the interval, whereas patients 5 and 11 had no evaluable data in either year. The remaining 23 patients were lost to follow-up (*Table 3*).

Owing to the small number of paired observations, longitudinal findings are presented descriptively, without inferential testing (*Table 1, right-hand columns; Table 2).*

Table 2: Longitudinal Comparison of Echocardiographic, Hemodynamic and Functional Parameters between 2025 and 2026: detailed analysis of individual changes for patients ID 1,4 and 16

Longitudinal Comparison of Echocardiographic, Hemodynamic, and Functional Parameters Between 2025 and 2026 in Three Patients									
	patient ID 1			**patient ID 4**			**patient ID 16**		
Parameter	**2025**	**2026**	**Δ**	**2025**	**2026**	**Δ**	**2025**	**2026**	**Δ**
HR rest (bpm)	85	84	-1.0	80	68	-12.0	96	65	-31.0
HR max (bpm)	100	105	+5.0	110	110	0	120	121	+1.0
EF rest %	55	55	0	55	55	0	54	55	+1.0
EF post-stress%	65	65	0	65	65	0	62	65	+3.0
PAPs rest (mmHg)	22	23	+1.0	24	25	+1.0	24	31	+7.0
TVR rest (m/s)	2.2	2	-0.2	2.2	2.5	+0.3	2.1	2.78	+0.68
sPAP max (mmHg)	34	38	+4.0	49	49	0	46	50	+4.0
TVR max (m/s)	2.6	2.8	+0.2	3.31	3.8	+0.49	3.2	3.53	+0.33
Annular diam (mm)	38	41	+3.0	39	43	+4.0	41	40.5	-0.5
Ins TR (severity)	1	1	0	2	2	0	2	2	0
Work Load Max (W)	75	100	+25.0	75	75	0	75	100	+25.0
TAPSE (mm)	21	22	+1.0	23	29	+6.0	28	19	-9.0
ΔPAPs (mmHg)	12	15	+3.0	25	24	-1.0	22	19	-3.0
TAPSE/PAPs rest	0.95	0.96	+0.01	0.96	1.16	+0.2	1.17	0.61	-0.56
6MWT distance (mt)	425	425	0	425	520	+95.0	500	520	+20.0
6MWT Borg	14	13	-1.0	13	13	0	12	15	+3.0
6MWT NYHA	1	1	0	1	1	0	1	2	+1.0

Table 3: Individual baseline values for total cohort and patient status at one-year follow-up: the table presents data collected in 2025 and in 2026 for each patient.

Per-patient values — 2025 (all 32) + 2026 follow-up block (ID 1,4,16 only)																					
		2025 — all patients													2026 (ID 1,4,16)						
ID	Age	HR	HR max	EF% rest	EF% post-stress	sPAP rest	sPAP max	TRV rest	TRV max	TAPSE	TAPSE/PAPs rest	Max workload	6MWT distance	6MWT Borg	NYHA class	sPAP rest	sPAP max	TAPSE	TAPSE/sPAP	6MWT dist	NYHA
1	82	85	100	55	65	22	34	2.2	2.6	21	0.95	75	425	14	1	23	38	22	0.96	425	1
2	72	76	110	55	65	29	40	3	3.4	19	0.66	75	350	13	2	Lost to follow-up					
3	80	60	110	55	65	52	55	3.46	3.53	21	0.4	75	475	10	1	Worsened					
4	86	80	110	55	65	24	49	2.2	3.31	23	0.96	75	425	13	1	25	49	29	1.16	520	1
5	82	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	Worsened					
6	92	86	120	55	65	24	30	2.4	2.5	19	0.79	75	350	13	2	Lost to follow-up					
7	78	50	120	55	65	28	38	2.6	3.4	23	0.82	75	300	12	2	Worsened					
8	79	75	150	52	57	18	22	1.91	1.94	21	1.17	100	450	10	1	Lost to follow-up					
9	69	70	150	55	65	26	36	2.3	2.9	19	0.73	100	500	10	1	Lost to follow-up					
10	80	60	100	55	65	22	34	2.1	2.7	21	0.95	75	300	9	1	Lost to follow-up					
11	82	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	Worsened					
12	85	80	120	55	65	28	36	2.4	2.5	20	0.71	100	300	12	2	Lost to follow-up					
13	63	72	130	55	65	20	36	1.9	2.8	21	1.05	75	425	13	1	Lost to follow-up					
14	80	73	125	55	65	23	38	2.6	2.8	17	0.74	75	350	11	2	Lost to follow-up					
15	81	80	110	55	65	35	35	2.2	2.2	17	0.49	75	n/a	n/a	2	Worsened					
16	84	96	120	54	62	24	46	2.1	3.2	28	1.17	75	500	12	1	31	50	19	0.61	520	2
17	81	n/a	n/a	n/a	n/a	20	n/a	1.74	n/a	18	0.9	n/a	200	15	3	Worsened					

18	66	60	150	55	65	32	38	2.6	2.9	21	0.66	75	450	12	1	Lost to follow-up
19	66	60	150	55	65	32	38	2.6	2.9	18	0.56	75	450	12	1	Lost to follow-up
20	74	55	115	55	65	22	36	2	2.6	17	0.77	75	400	13	2	Lost to follow-up
21	73	66	105	55	65	38	46	2.8	3.2	16	0.42	75	400	11	3	Lost to follow-up
22	84	76	120	55	65	22	36	2.4	3.2	20	0.91	75	350	13	2	Lost to follow-up
23	73	86	115	55	65	27	34	2.1	3	23	0.85	75	450	11	2	Lost to follow-up
24	94	78	110	55	65	32	34	2.8	2.8	19	0.59	75	300	14	2	Lost to follow-up
25	75	73	125	55	65	20	38	3	2.8	17	0.85	75	350	12	3	Lost to follow-up
26	87	52	115	55	65	18	42	2.4	2.6	21	1.17	75	400	10	2	Lost to follow-up
27	79	64	105	55	65	30	36	2.8	3.4	23	0.77	75	350	14	2	Lost to follow-up
28	87	58	115	55	65	22	34	4	3	19	0.86	75	300	13	2	Lost to follow-up
29	82	74	110	55	65	26	36	2.2	3	19	0.73	75	425	10	1	Lost to follow-up
30	84	64	100	55	65	18	30	2.2	2.8	21	1.17	75	400	10	2	Lost to follow-up
31	68	68	120	55	65	24	38	2.4	2.6	23	0.96	75	350	11	2	Lost to follow-up
32	86	74	100	55	65	28	36	2.4	2.5	21	0.75	75	400	11	2	Lost to follow-up

C. LONGITUDINAL CHANGES IN PATIENTS WITH PAIRED DATA

Among the three patients with complete paired data, three divergent one-year trajectories were observed *(Table 2; Figures 1–3).*

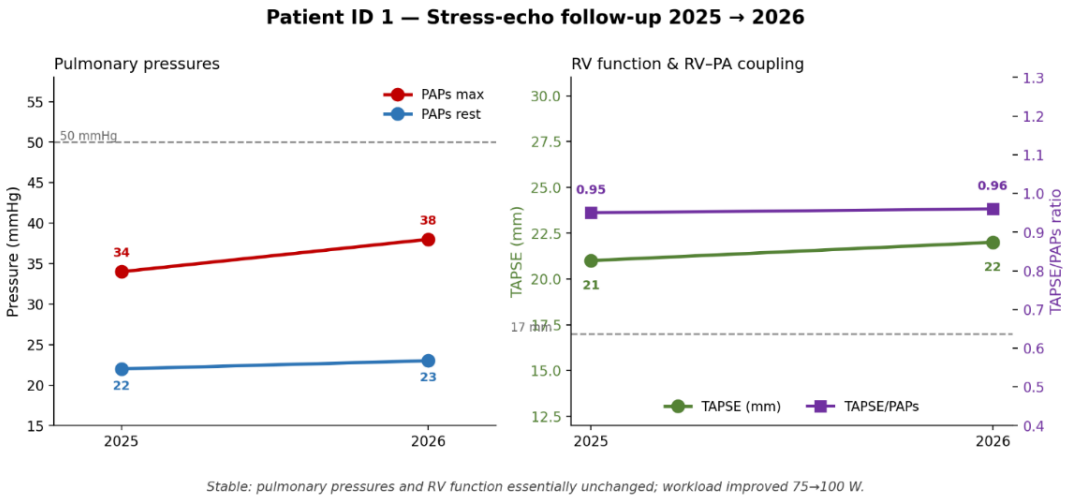

Figure 1: Longitudinal evolution of functional and hemodynamic parameters in Patient 1

Patient 1 remained clinically and echocardiographically stable (Figure 1). Ejection fraction was unchanged (55% at rest and 65% post-stress in both years), and RV function was preserved (TAPSE 21→22 mm; TAPSE/sPAP ratio 0.95→0.96). Resting and peak pulmonary pressures varied minimally (sPAP rest 22→23 mmHg; sPAP max 34→38 mmHg). Exercise capacity was maintained, with an unchanged 6MWT distance (425 m) and stable NYHA class I, while the maximum achieved workload improved from 75 to 100 W.

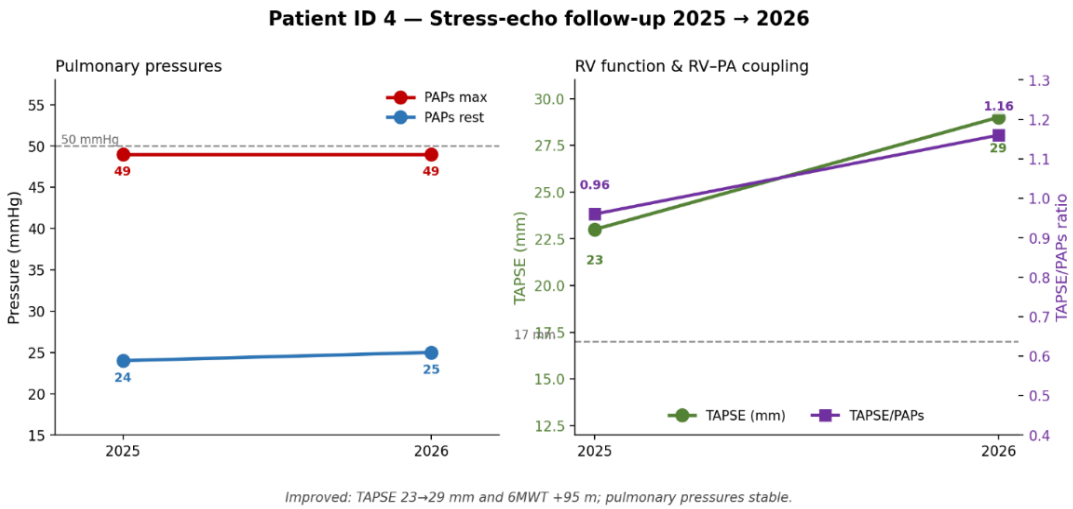

Figure 2: Longitudinal evolution of functional and hemodynamic parameters in Patient 4

Patient 4 showed functional improvement (*Figure 2).* RV longitudinal function increased (TAPSE 23→29 mm), with a corresponding rise in the TAPSE/sPAP ratio from 0.96 to 1.16, indicating improved RV–pulmonary arterial (RV–PA) coupling. Pulmonary pressures were stable (sPAP max 49→49 mmHg), ejection fraction remained preserved, and the 6MWT distance increased markedly from 425 to 520 m (+95 m) with maintained NYHA class I.

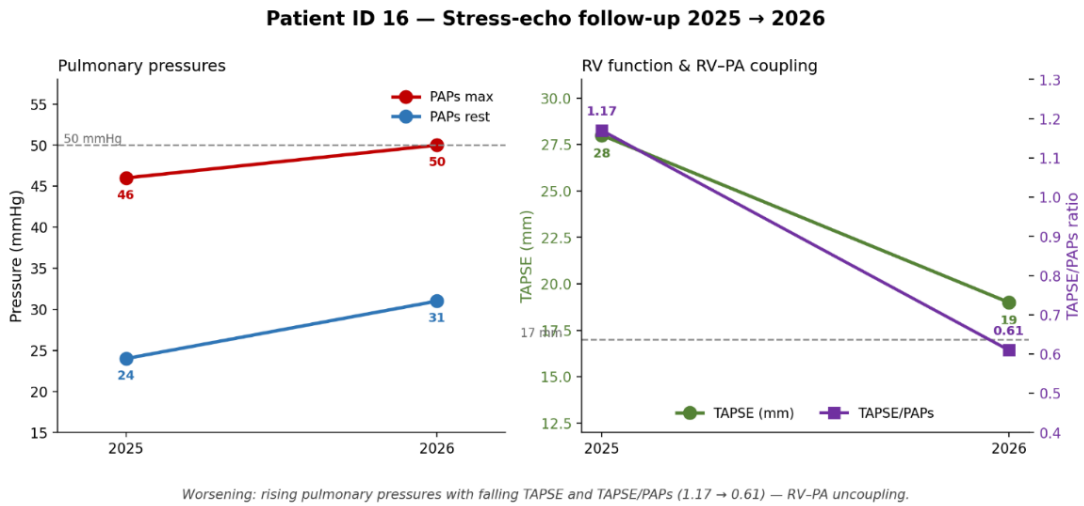

Figure 3: Longitudinal evolution of functional and hemodynamic parameters in Patient 16

Patient 16, in contrast, demonstrated clear deterioration (*Figure 3*). Pulmonary pressures rose at both rest and peak (sPAP rest 24→31 mmHg; sPAP max 46→50 mmHg), accompanied by an increase in resting TRV from 2.1 to 2.78 m/s. RV function declined substantially, with TAPSE falling from 28 to 19 mm and the TAPSE/sPAP ratio decreasing from 1.17 to 0.61, consistent with RV–PA uncoupling. This was paralleled by a one-class worsening of symptoms (NYHA I→II) and a higher exertional Borg score (12→15), despite a modest increase in 6MWT distance (500→520 m).

D. SUMMARY

Taken together, the paired data illustrate three distinct one-year trajectories: stability (Patient 1), improvement in RV function and exercise capacity (Patient 4), and combined RV functional decline with rising pulmonary pressures and RV–PA uncoupling (Patient 16). The high proportion of patients who could not complete or were lost to follow-up (29 of 32) constrains cohort-level inference and is itself a noteworthy observation.

DISCUSSION

A. STUDY FRAMEWORK AND CLINICAL CONTEXT

The present single-center, longitudinal study aimed to evaluate the long-term functional and echocardiographic outcomes in a cohort of patients affected by severe or torrential tricuspid regurgitation (TR) who underwent transcatheter edge-to-edge repair (TEER) using the TriClip system.

For several years in cardiological field, the Tricuspid valve has been defined as the 'forgotten valve', since its pathology was considered merely a reflection of left heart conditions(22,23). Consequently, the clinical management of Tricuspid Regurgitation (TR) was limited to medical therapy, which did not always prove to be sufficient. Today, the recognition of severe TR as a progressive condition, associated with increased mortality and hospitalization rates, has led to a shift(23).

Isolated tricuspid surgery, despite resolving valvular incompetence, remains an underutilized option due to high in-hospital mortality, estimated at around 8-10%(40).

However, transcatheter techniques such as the TriClip offer a fundamental, minimally invasive therapeutic alternative. The efficacy and safety of this device have been validated by the TRILUMINATE Trial, which demonstrated a reduction in regurgitation and an improvement in quality of life at a two-year follow-up(39,42).

Nevertheless, translating these findings from randomized clinical trials to daily clinical practice raises critical questions, regarding patient selection and long-term post-procedural hemodynamic evolution.

B. ANALYSIS OF RESULTS

The initial cohort evaluated in 2025 reflected the real-world epidemiological profile: it comprised 32 patients with a mean age of 79 years (63-94), affected by severe, massive or torrential Tricuspid Regurgitation with right heart failure symptomatology, objectified by a median NYHA class of II. This was accompanied by limitation in global functional capacity, evidenced by a mean distance covered in the Six-Minute Walk Test (6MWT) of only 386 meters.

In this cohort, left ventricular systolic function was preserved (resting LVEF 55%); however stress echocardiographic measurements revealed a significant hemodynamic overload on the right chambers: systolic pulmonary artery pressure (sPAP) increased from a mean resting value of 26 mmHg to 37 mmHg at peak exercise, with tricuspid regurgitation velocities reaching 2.86 m/s during exertion.

These data highlight a vulnerable population, presenting a clinical picture of tricuspid regurgitation and advanced heart failure, wherein a precarious equilibrium between volume overload and dynamic pulmonary afterload is observed.

The analysis of the three patients who completed the entire evaluation process at a one-year follow-up allowed for the observation of the heterogenous pathophysiological response to the correction of Tricuspid Regurgitation (TR) via the TriClip procedure.

Patient ID 1 represents the profile of stability. Morphological and functional parameters remained unchanged compared to the previous year: right longitudinal function (TAPSE from 21 mm to 22 mm) and pulmonary pressure values (sPAP from 22 mmHg to 23 mmHg) remained stable. From a functional perspective, the maintenance of the distance covered in the 6MWT (425 m) and the increase in the maximum tolerated workload on the ergometer (from 75 to 100 W) suggest that, in a percentage of subjects, the primary benefit of the TEER procedure lies in interrupting the vicious cycle whereby "TR begets TR".

Patient ID 4 is the ideal expression of the TriClip device's efficacy, representing therapeutic success. One year after the last follow-up, an increase in right ventricular longitudinal function was observed (TAPSE from 23 mm to 29 mm). This finding, coupled with the stability of pulmonary pressure, led to a clear increase in the TAPSE/sPAP ratio, rising from 0.96 to 1.16 mm/mmHg. This ratio is the echocardiographic index used to estimate right ventricular-pulmonary arterial (RV-PA) coupling. The improvement of this index serves as evidence of early reverse remodeling of the right ventricle, which, having been freed from the chronic volume overload of the regurgitation, recovered its contractile reserve and capacity to adapt to pulmonary afterload. This progress also led to a marked improvement in overall functional capacity, demonstrated by a 95 m increase in the distance covered during the 6MWT (from 425 to 520 meters).

Patient ID 16 embodies deterioration and uncoupling. In this case, the clinical course was the opposite, as a worsening occurred. An increase in systolic pulmonary pressure was recorded both at rest (from 24 to 31 mmHg) and at peak exercise (from 46 to 50 mmHg), alongside a collapse in contractile function (TAPSE from 28 to 19 mm) and an increase in resting tricuspid regurgitation velocity (TRV from 2.1 to 2.78 m/s). Furthermore, the evident decrease in the TAPSE/sPAP ratio, dropping from an optimal value of 1.17 to a pathological value of 0.61 mm/mmHg, indicates a distinct RV-PA uncoupling. This led to clinical decline, evidenced by the transition from NYHA class I to NYHA class II and an increase in the Borg scale score (from 12 to 15), which is synonymous with an increased perception of dyspnea and fatigue during exertion.

The coexistence of such variable responses within a small number of longitudinal observations suggests the need to evaluate the methodological and clinical factors that influence post-operative evolution.

C. DROP-OUT RATE

The most impactful element of the study is the exceptionally high loss to follow up rate: out of a cohort of 32 patients, 23 were lost to follow-up, 6 manifested a clear clinical worsening that precluded re-testing and only 3 completed the evaluations in 2026**.**

Such losses hindered the application of statistical inference tests on the entire cohort and the ability to extrapolate the conclusions to the broader population. Nevertheless, in a real world context, the loss of patients is not exclusively a methodological limitation; rather, it constitutes clinical information itself, which can be attributed to two main justifications.

The first reason underlying the drop-out is based on the difficulty of executing the diagnostic protocol to which patients had to be subjected both at baseline and at follow-up. The workflow included performing the 6-Minute Walk Test and a stress echocardiogram using a semi-recumbent cycle ergometer with a symptom-limited protocol. The initial evaluation in 2025 was, for many patients, an experience burdened by exertional dyspnea, extreme muscle fatigue and osteoarticular discomfort; consequently, many lost to follow up patients refused the proposal to undergo a one-year control examination due to the difficulties encountered the previous year, preferring less invasive outpatient cardiological visits not associated with physical exertion.

The second motivation for the clinical drop-out relates to the timing of the valve correction. In clinical practice, it is possible that patients suffering from severe functional tricuspid regurgitation are referred for evaluation and subsequent TriClip intervention at a late stage in the natural history of the disease. When right heart failure has reached an advanced stage with evident clinical signs of peripheral congestion, ascites, and congestive hepatopathy, the right ventricular myocardium undergoes irreversible structural modifications, such that the contractile reserve of the ventricle is compromised. Intervention via TriClip placement at an already advanced stage, despite reducing the degree of regurgitation, fails to induce reverse remodeling or functional recovery of the already compromised myocardium. This justifies the clinical trajectory of the six patients who, despite being treated successfully, clinically

worsened, manifesting a loss of exercise tolerance severe enough to limit the execution of the tests.

D. THE REAL LIFE PERSPECTIVE

The dimensional limitation of the studied sample allows for additional reflections, as it provides a real life perspective rather than solely focusing on the application of a cutting-edge methodology.

Although the TEER procedure is a significant medical achievement, it should not be interpreted as a universal solution for all forms of advanced heart failure. Unlike large multicenter trials, which utilize strict inclusion and exclusion criteria to select an optimal patient cohort, this study highlights a more vulnerable and complex scenario, consisting of frail, elderly subjects with very advanced stages of the disease. The heterogeneity of the results emphasizes that the response to treatment is not uniform, underscoring that while it is an effective technique, careful patient selection is paramount.

Our observations of variable individual responses underscore the necessity of strict patient selection, a concept strongly supported by recent large-scale real-world data.

The BRIGHT registry (N=511) demonstrated that while TriClip is safe and effective in a broad population, 1-year survival is independently associated with preserved baseline right ventricular function, specifically a larger baseline RV TAPSE, rather than baseline TR severity(47).

This aligns with our finding in Patient 4, where adequate baseline RV-PA coupling and contractile reserve translated into therapeutic success.

Conversely, intervening when right heart failure has already caused irreversible structural modifications may negate the long-term survival benefits of T-TEER, highlighting the critical importance of procedural timing(47).

CONCLUSION

Despite the severe limitations related to the small sample size and high drop-out rate, these preliminary observations are consistent with the hypothesis that transcatheter edge-to-edge repair of the tricuspid valve via TriClip induces favorable clinical and hemodynamic responses, including disease stabilization and marked reverse remodeling associated with the optimization of ventricular-arterial coupling.

On the other hand, the high drop-out rate and cases of clinical deterioration highlight that the efficacy of the methodology may heavily depend on the timing of the intervention. Acting proactively, anticipating correction prior to the onset of irreversible alterations, could prove fundamental in maximizing the long-term success of these novel transcatheter techniques.

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