

Tricuspid Regurgitation in a Patient with Heart Transplant: Percutaneous Management

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Regurgitación tricuspídea en un paciente con trasplante de corazón: Manejo percutáneo

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ABSTRACT

Tricuspid regurgitation (TR) is the most frequent valvular complication after heart transplantation with different clinical sequelae. In its most severe form, it can cause right heart failure with a poor long-term prognosis. Its management is complex, both medical, surgical, and percutaneous. The TricValve system, a bicaval system with two self-expanding valves (superior vena cava and inferior vena cava), dedicated to treating symptomatic IT refractory to medical therapy, is safe and effective in improving quality of life. We present the first heart transplant patient with severe symptomatic TR who underwent successful bicaval valve (TricValve) implantation.

Keywords: Cardiology; Heart transplantation; Tricuspid valve insufficiency.

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RESUMEN

La insuficiencia tricúspide (IT) es la complicación valvular más frecuente después del trasplante cardíaco con diferentes secuelas clínicas. En su forma más grave puede generar insuficiencia cardíaca derecha con mal pronóstico a largo plazo. Su manejo es complejo, tanto médico, quirúrgico como percutáneo. El sistema TricValve, sistema bicava con dos válvulas autoexpandibles (vena cava superior y vena cava inferior), dedicado a tratar la IT sintomática refractaria a terapia médica, es segura y efectiva para mejorar la calidad de vida. Presentamos el primer paciente trasplantado cardíaco con IT severa sintomática sometido a implante de válvulas bicava (TricValve) exitoso.

Palabras clave: Cardiología; Insuficiencia de la válvula tricúspide; Trasplante de corazón.

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Tricuspid regurgitation (TR) is an incompetence of the tricuspid valve to close. When it is significant, it has been associated with right heart failure (HF), liver failure, and mortality¹.

Orthotopic heart transplantation (HT) continues to be the therapy for advanced HF refractory to medical treatment² and TR is the most common valve complication after HT with an incidence of up to 80%³. Although most are mild and clinically insignificant, one third is in the moderate to severe range² and 6% develop symptoms refractory to medical therapy (MT), generating the need for correction⁴. Surgical repair is a challenge, given the previous surgical context, where up to 50% require reoperation due to surgical failure⁴. We present the case of a 65-year-old man, a 12-year-old heart transplant recipient, humoral rejection treated 10 years after the transplant and subsequent symptomatic TR refractory to MT.

Clinical case

A 65-year-old male with a history of HF secondary to idiopathic dilated cardiomyopathy underwent HT 12 years ago. In the post-transplant period, he was maintained on chronic immunosuppressive therapy with tacrolimus and mycophenolate. Additionally, he required a dual-chamber pacemaker to manage tachycardia-bradycardia syndrome. Eleven years after transplantation, he experienced an episode of humoral rejection treated with plasmapheresis, intravenous immunoglobulin, and rituximab, along with multiple endomyocardial biopsies (EMB) to monitor and manage his condition. Subsequent to the rejection episode, he developed paroxysmal atrial fibrillation, for which he began anticoagulation therapy with acenocoumarol.

After hospitalization due to rejection, dyspnea progressively stands out, reaching functional capacity III according to the New York Heart Association Classification. With diuretic treatment, effective decongestion is achieved, however, at 6 months the patient developed jugular venous distention with a V wave on physical examination and congestion that is resistant to high-dose diuretics. ProBNP of 825 pg/dl.

The case is reviewed by the heart team. A TRI-SCORE of 5 points, 14% annual mortality rate

(Table 1). Technical difficulties identified include: A pacemaker carrier, immunosuppression, future need for mechanical circulatory support, and previous sternotomy. It is decided to proceed with percutaneous intervention using an appropriate device for the aforementioned conditions: TricValve.

Pre-implant planning was performed using cardiac catheterization (Table 2), cavography, and cardiac tomography (CT) (Figure 1). The CT allows for sizing of both cavas and the selection of suitable devices (in this case, a superior vena cava (SVC) #25 and an inferior vena cava (IVC) #35).

Local anesthesia with lidocaine. Access is obtained using an 8-26 Fr introducer in the right femoral (RF) vein and a 6 Fr introducer in the left femoral (LF) vein. Unfractionated heparin 5000 IU is given intravenously to achieve an activated clotting time (ACT) equal to or greater than 250. A 0.35 Fr guide wire is advanced through the RF access to the right jugular vein (RJV) a high-support guide wire (Figure 2). A pigtail catheter is placed in the right pulmonary artery (RPA). The RF venous access as used to advancement of a 25 Fr delivery system with the #25 SVC prosthesis. The prosthesis is positioned according to the marker in the RPA (Figure 2). The delivery system is withdrawn through the RF access while keeping at the RJV. Then, a pigtail catheter is positioned in the suprahepatic vein as a second marker. The second delivery system is advanced, and the second #35 prosthesis is deployed (Figure 2).

A post-implant cardiac catheterization is performed, which shows a reduction of the V wave to 15 mm Hg in to the cavus vein. Atrial angiography does not identify leaks. There is no transprosthetic gradient observed. After the deployment of the prosthesis the patient reports intense pain in right shoulder, which is controlled with opioids. The access sites are sutured.

The patient is hospitalized in the care unit for monitoring. The patient's condition remains stable without any complications. Vasodilators and diuretics are titrated. The patient is discharged 72 hours later without incidents.

The 3-month transthoracic echocardiogram (TTE) shows a right ventricle (RV) of similar dimensions, torrential TR, prostheses in the

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SVC and IVC without leaks or transprosthetic gradients, and no pericardial effusion. Diuretics are reduced.

At the sixth month the patient is asymptomatic without signs of right HF. Control ProBNP of 235 pg/ml.

Table 1. TRI-SCORE⁹.

Risk factors	Score	Patient
Age over 70 years	1	0
Functional class III-IV NYHA	1	1
Signs of right heart failure (presence of jugular venous distention, ascites, or peripheral edema)	2	2
Furosemide dose \geq 125 mg/day	2	2
Glomerular filtration rate < 30 ml/min	2	0
Total bilirubin elevation	2	0
Left ventricular ejection function less than 60% (estimated visually or by biplane Simpson)	1	0
Moderate to severe right ventricular dysfunction (right ventricular dysfunction is based on echocardiographic values of TAPSE, S' wave and fractional area change (CAF))	1	0
0 pts: intrahospital mortality (IM) 1%, 1: 2%, 2: 3%, 3: 5%, 4: 8%, 5: 14%, 6: 22%, 7: 22%, 8: 48%, > 9: 65%		
http://www.tri-score.com		

NYHA: New York Heart Association. Ptos: points.

Table 2. Right cardiac catheterization.

Pressures	Values (mm Hg)	Saturometry
Right atrium	Wave A 11 Wave V 35	62%
Right ventricle	25/12	63%
Pulmonary artery	12	62%
Pulmonary enclavation pressure	12	

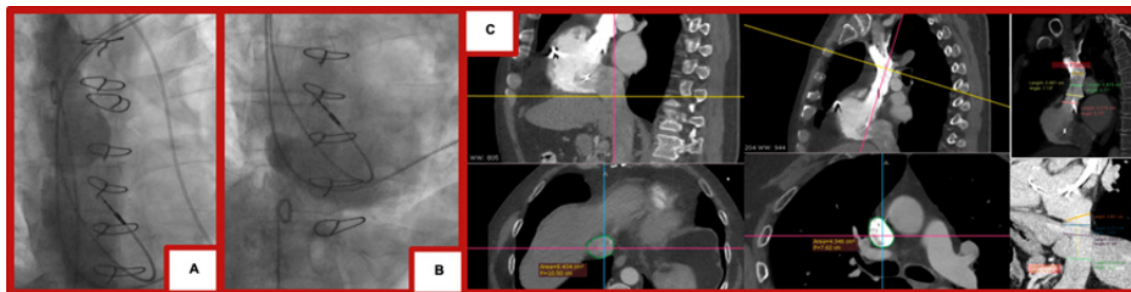


Figure 1: A. Cavography Vena Cava Superior in anteroposterior projection. B. Lower vena cavography and right atrium in anteroposterior projection. C. cardiac tomography in sagittal and transverse cut in upper and lower vena cava. Measurement of your area and diameters (distal, medium and proximal).

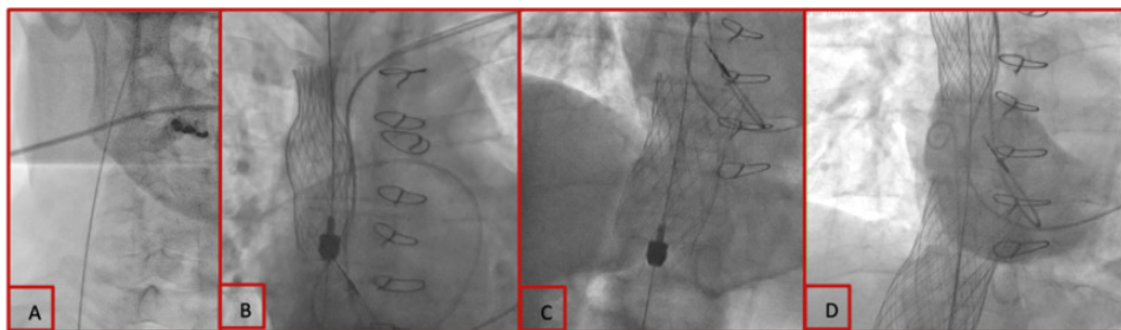


Figure 2: A. Anteroposterior projection. Guide in right jugular vein is observed. B. Anteroposterior projection. Self-expandable valve is observed in upper vena cava and pigtail caeter in right pulmonary artery. C. Anteroposterior projection. Self-expandable valve is observed in lower vena cava and pigtail caeter in suprahepatic vein. D. Auriculography in anteroposterior projection. Valves are observed in the upper vena cava and vena cava lower. No periprotthetic leaks are observed.

Discussion

The tricuspid valve is traditionally known as the “forgotten valve.” Its lesser importance is reflected in fewer published literature. Incidental TR without physiopathological consequences is frequently detected during TTE. The prevalence exceeding 3% in patients over 75 years old, similar to aortic stenosis or mitral insufficiency⁵.

Tricuspid apparatus disorders are classified as primary or secondary. In the latter case, there is an increasing number of patients developing significant TR due to iatrogenic causes: pacemaker leads and EMB⁶. The development of TR in this context has been reported in up to 23% of cases³. Unfortunately, fluoroscopy and TTE do not provide

optimal visualization of the valve apparatus⁷.

- Significant TR presents with signs and symptoms of right HF: ascites, fatigue, peripheral edema, painful hepatosplenomegaly, and abdominal distension. Untreated is associated with reduced survival due to hepatic and/or renal insufficiency⁸.
- Currently, the disease has limited treatment options, posing a challenge. Management includes medical, surgical, and percutaneous treatments. MT for severe TR is limited and mainly focuses on symptom relief.

The absence of accurate surgical risk assessment scales and high in-hospital mortality led to the

development of the TRI-SCORE scale⁹. This score consists of eight variables and a scoring range from 0 to 12 points, providing a simple, fast, sensitive, and reproducible risk model that can aid in decision-making (Table 1). It demonstrates an exponential increase in mortality as the risk score increases.

Surgical replacement or repair is associated with a high rate of complications and hospital mortality of up to 10%¹⁰, especially in patients with previous left-sided valve surgery or prior tricuspid repair. This motivated the development of percutaneous approaches, including transcatheter tricuspid valve intervention: annuloplasty devices, replacement devices, cava valve implantation, and coaptation devices¹¹. Fortunately, these transcatheter options have recently been incorporated into treatment algorithms according to guidelines¹².

Many patients with severe TR are considered suboptimal candidates for these new approaches¹³. Heterotopic valve implantation in the vena cava has emerged as a possible transcatheter strategy to indirectly treat the systemic effects of severe TR¹⁴. TricValve is an interventional approach for the treatment in patients who are not considered suitable for surgery. The concept behind this approach is to reduce caval reflux and, thus, decrease systemic venous congestion, promote RV remodeling, and increase cardiac output¹⁵. The first human experience was reported in 2011 and was successful¹⁶.

TR is a common condition after HT (during follow-up there is severe TR in up to 25% of cases¹⁷). Several mechanisms can explain this high rate, including RV dilation and repetitive EMB¹⁸. In most cases, the severity is mild to moderate and not associated with clinical sequelae requiring surgical intervention². However, when it is severe, it leads to the same poor long-term outcomes as in non-transplanted patients with severe TR. Unfortunately, surgical treatment in this setting carries a high risk (under immunosuppressive treatment and history of cardiac surgery)¹⁷. Percutaneous treatment has recently emerged as a treatment option in patients with high risk for cardiac surgery¹⁹. Its use therefore seems attractive in those patients and can provide an opportunity

to treat TR while avoiding a high-risk surgery in such fragile patients.

Currently, there is no data that compares the success rate between surgical treatment and percutaneous management, with isolated cases reported in both scenarios¹⁷. It is in this context that the heart team defined the possibility of a device TricValve as the most suitable option, ruling out surgery due to high surgical risk and limited experience in this type of scenario in our center.

Appropriate clinical, imaging and laboratory evaluation is necessary for its implantation. From a technical point of view, it is necessary to have an echocardiogram that does not show severe RV dysfunction (TAPSE <13 mm), at systolic pulmonary artery pressure >65 mm Hg²⁰. Furthermore, it is necessary to rule out renal dysfunction defined as serum creatinine >3mg/dl.

Procedure planning necessitates CT to ensure that the anatomy is suitable for the system implantation and to generate device measurements for both the IVC and SVC. Another noteworthy point in HT patients is the cavoatrial anastomosis zone, which could pose challenges in prosthesis expansion. The procedure is simple and avoids the challenges of navigating around the tricuspid valve apparatus. It requires local anesthesia with lidocaine, and femoral vein access is used. TTE is not required during the procedure; only fluoroscopy and cavography are necessary. It is crucial to avoid paravalvular leakage or obstruction of the hepatic vein, ensuring that the stent of the IVC does not protrude more than 20 mm into the right atrium. Postoperative monitoring the main drawback is post-implantation shoulder pain, which resolves within the first 24 hours post procedure.

Conclusions

The surgical/interventional management of severe symptomatic TR in transplanted patients is difficult, having a different confrontation with lower therapeutic options than in patient with severe non-transplanted IT. The bicava valve implant is a safe and effective option in this scenario.

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