TRICUSPID REGURGITATION

Epidemiology

In the community, prevalence of moderate or severe tricuspid regurgitation (TR) is estimated to be as high as 0.55% and up to 4% after 75 years, a prevalence that is almost similar to that of aortic stenosis or mitral regurgitation.[1] Approximately 30% of patients with severe MR may also have severe TR, and up to 80% of patients referred for echocardiography have some degree of TR.[2]

Aetiology

Primary tricuspid regurgitation occurs as a result of intrinsic valve disease and is attributable to congenital anomalies, infective endocarditis, rheumatic disease, carcinoid heart disease, toxic effects of chemicals, tumors, blunt trauma, or myxomatous degeneration.[3] Congenital TR is usually due to Ebstein's anomaly. Carcinoid heart disease is frequently seen in patients with neuroendocrine tumors (2.5-5 cases per 100,000 population).[4] The tricuspid, mainly, and pulmonary valves are usually involved. Endocarditis-related TR is increasingly recognized fur to IV drug abuse.[5]

Secondary TR may be due to either ventricular or atrial dilatation, although both causes may co-exist.[5] Atrial secondary TR is characterized by normal-appearing leaflets that fail to coapt in the presence of marked annular and atrial dilatation, usually due to AF. The leaflets exhibit minimal tethering or tenting, and the right ventricular structure and function are typically normal. ***Ventricular secondary TR*** is due to dilatation of the right ventricle (mainly the midventricular free wall), leading to apical displacement of the papillary muscles and tethering of the leaflets. It occurs in response to any of several disease processes such as mitral and aortic valve disease and AF (Table 1).[6,7] Pacemaker and, especially ICD lead placement results in significant worsening of TR in 4-7 % of patients, and is a cause of secondary TR.[8-11] However device lead implantation in patients with a bioprosthetic TV has not always been associated with an increased incidence of significant prosthetic TR, and removal of the lead may or may not result in improvement.[5,12] ***Atrial secondary TR*** is a distinct phenotype of secondary tricuspid regurgitation with predominant dilation of the right atrium and normal right and left ventricular function. It occurs most commonly in elderly women with AF and in heart failure with preserved ejection fraction in sinus rhythm.[13] Atrial secondary TR is associated with pre-existing AF, but up to 50% of these patients do not have AF which, when develops, confers a higher risk.[14]

After surgery for ischaemic MR, progression of unrepaired nonsevere TR is uncommon. Progression of post-operative TR depends not only on pre-operative risk factors such as annular size or atrial fibrillation but also on post-operative factors such as residual or recurrent MR and the use of a permanent pacemaker or defibrillator.[15]

Pathophysiology and Natural History

The tricuspid valve apparatus consists of the annulus, the anterior (largest), septal, and posterior (or inferior) leaflets, chordae, and papillary muscles.[16] Most of the TV annulus lies on the atrioventricular junction; thus, its size depends on RV volume. Only 40% annular dilatation may result in clinically significant tricuspid regurgitation, whereas this was seen at 75% annular dilatation in mitral-valve studies.[5] Mild TR is common and usually is benign. However, moderate or severe TR can lead to irreversible myocardial damage and adverse outcomes.[2,3] When TR develops, there is a prolonged period of progressive RV and RA volume overload that engenders additional TR until right heart failure develops. PA pressure in functional TR is >55 mm Hg. RV failure with severe TR or constrictive pericarditis are possible causes of cardiac cirrhosis due to ischemic hepatopathy. Medical management of severe disease is associated with 1-year mortality rate of approximately 40% and even mild tricuspid regurgitation is associated with a nearly 30% increase in all-cause, long-term mortality.[5] Moderate and severe TR increase mortality independently of PV pressure or RV function. [17] However, RV dysfunction, but not significant TR, is independently associated with survival late after left heart valve procedure.[18] Fast development of TR is associated with older age, presence of a pacemaker or defibrillator lead, presence of mild (vs. no) TR, echocardiographic signs of RV dilation and dysfunction, and left-sided valve surgery without concomitant tricuspid surgery.[19] In patients with significant secondary TR, patients with RV systolic dysfunction have worse clinical outcome regardless of the presence of RV dilation.[20]

Presentation

Symptoms of TR are often nonspecific, and patients with severe TR may be asymptomatic (Table 2). Symptoms are usually of fatigue and decreased exercise tolerance as a result of low cardiac output. Elevated right atrial (RA) pressure leads to atrial arrhythmias (mainly AF), peripheral edema and hepatic congestion with decreased appetite and abdominal fullness. Eventually right-heart failure with ascites and anasarca develops.

Physical Findings

Elevated JVP with a prominent v wave in 35% to 75% of patients with severe TR.

Hepatomegaly is present in 90%, but pulsating liver is noted inconsistently.

The holosystolic murmur of TR is heard along the sternal border, increasing in intensity with inspiration as a result of increased systemic venous return (it is heard in <20% of patients with severe TR due to equalization of pressures between the RA and RV). AF is common.[21]

Investigations

Echocardiography (2-D or preferably 3-D) is the main diagnostic tool (Table 3 and Figure 1).[22-25] A novel TV leaflet nomenclature classification scheme can be used to identify 4 types of TV morphologies with the use of TEE imaging (Figure 2).[26] Vena contracta width (indirectly reflects the effective regurgitant orifice area) > 0.7 cm, large flow convergence (PISA radius > 0.9 cm at Nyquist limit of 40 cm/s) and systolic flow reversal in hepatic veins are specific signs of severe TR. An effective regurgitant orifice (ERO)> 40 mm^2 indicates adverse clinical outcomes, even for isolated TR.[27,28] Additional supportive signs are a dense "dagger-shaped", triangular, early-peaking systolic continuous-wave Doppler signal, IVC dilatation and respiratory diameter variation <=50%, a prominent E-wave, and RA/RV dilatation. The tricuspid annulus can be measured in diastole from the apical four-chamber view; in this view, an annular diameter >40 mm (or >21 mm/m^2 body surface area) is considered to be dilated.[29] There is major respiratory variation of TR and RV shape and echocardiographic assessment requires multiplying measurements throughout the cardiac cycle in order to appropriately assess TR and right ventricular pressure.[30]

CMR is considered the reference standard for assessing right ventricular chamber size and volumes and can be used to quantify the severity of tricuspid regurgitation.[31] Liver extracellular volume is a novel objective biomarker that provides a comprehensive and prognostic assessment of chronic TR and its consequences.[32]

Right ventriculography is not helpful because the catheter may induce TR, and cardiac catheterization for pressure measurements is rarely required. It may be needed in case of discordant symptoms and echocardiographic findings (Table 4).

Plasma or 24-h urinary ***5-hydroxyindoleacetic acid (5-HIAA)*** levels or plasma **chromogranin A (CgA)** are useful for identifying patients at risk of developing carcinoid heart disease.[4]

Dedicated ***clinical risk scores*** such as the TRI-SCORE (www.tri-score.com),[33] and the STS isolated TV risk calculator.[34] take into consideration clinical and echocardiographic signs of RV dysfunction, and secondary organ (particularly hepatic and renal) impairment. They guide the clinical decision-making process of patients with severe TR, especially as transcatheter therapies are emerging (Figure 3).

Therapy

Medical Therapy

The medical management of secondary TR is aimed at the underlying causes such as left ventricular failure and lung disease.[35] Diuretics is the only proven therapy for symptomatic relief but when excessive may decrease cardiac output (Table 4).

Surgical Therapy

Few data are available regarding surgical management. It is possible that the late presentation of patients with severe symptomatic tricuspid regurgitation is largely responsible for the 10-12% in-hospital mortality associated with isolated surgical tricuspid valve replacement.[5,36] Current indications for TV surgery are severe primary and TR are tricuspid annulus dilatation with symptomatic RV failure in patients undergoing left-sided valve surgery (Tables 5 and 6, Figures 4 and 5).[37,38]

Patients with ERO<40 mm^2 in the context of isolated TR can be treated conservatively.[27] Functional TR with an annular diameter <40 mm can also be treated medically. When the diameter exceeds 40 to 44 mm, tricuspid annuloplasty with or without leaflet augmentation is recommended.[5,29] The treatment of functional (secondary) TR in particular remains controversial because of high rates of residual or recurrent TR and poor outcomes following surgical intervention. However, functional TR may not resolve on its own if the underlying disease is successfully treated, and RV volume overload due to chronic TR may result in irreversible RV myocardial damage.[2] Functional tricuspid regurgitation is a strong determinant of survival after heart failure with reduced ejection fraction.[39] TV repair or replacement is the only effective treatment for symptomatic TR, and recently there has been evidence in favor of a more aggressive surgical approach to secondary (functional) TR.[40] Remodeling annuloplasty of the tricuspid valve based on tricuspid dilation (tricuspid annular diameter greater than twice the normal size (> or = 70 mm)) improves functional status of patients undergoing mitral valve repair irrespective of the grade of TR.[41] However, in patients with isolated severe TR, surgery has not been associated with improved long-term survival compared to medical management.[42]

Operative mortality for concomitant ***TV annuloplasty*** is doubled compared to that for isolated MV surgery but avoids the usual post-operative deterioration of existing TR. The presence of either >3.2-cm right ventricular midcavity diameter or >0.85-cm^2 tricuspid valve tethering area are associated with adverse events at 1 year after tricuspid annuloplasty.[43] In a randomized comparison (CTSN RCT), the addition of tricuspid repair at the time of mitral valve surgery resulted in less frequent progression to severe tricuspid regurgitation, at the cost of a higher risk of permanent pacemaker implantation, at 2 years follow-up. No effect

with respect to MACCE, quality of life, functional status, hospital readmission, and death was detected during this period.[44]

Surgery for ***isolated TV disease*** is rare, and the in-hospital mortality following repair or replacement is up to 10%.[36]

In patients with both primary and functional TV disease, ***TV repair*** is associated with better survival than TV replacement. Ring annuloplasty is preferable to purse-string annuloplasty (De Vega).[22,45]

TV replacement is indicated for patients whose valves are not amenable to repair. No difference has been found in survival among patients with mechanical versus biological TV prostheses, but a bioprosthesis is preferred due to the low incidence of thromboembolic complications. Right-sided bioprostheses have superior durability compared with left-sided bioprostheses. Pericardial bioprostheses are generally avoided in the tricuspid position because of leaflet stiffness and risk of obstruction.[23] Recommendations on pacing after TS surgery are provided in Table 7.

Transcatheter TV implantation, annuloplasty or ***repair*** using various valves and other percutaneous devices are new options as an alternative to surgery for high-risk patients with functional TR or for failing bioprostheses (Figure 6).[1,46-49]

In the TRIC-I-HF RCT in patients with heart failure and severe tricuspid regurgitation, transcatheter tricuspid-valve repair and medical therapy resulted in significantly better outcomes than medical therapy alone with a lower incidence of a composite of death from any cause or hospitalization for heart failure at 3 years.[50]

When severe symptomatic TR is secondary to leaflet perforation or lead impingement from a ***pacemaker lead*** that clearly interferes with leaflet mobility or coaptation, removal or repositioning of the lead with additional TV repair may decrease the degree of TR.[8]

Asymptomatic patients with severe ***carcinoid heart disease*** may require valve replacement, enabling partial hepatic resection or liver transplantation. Carcinoid heart disease patients represent a high-risk surgical subgroup primarily because of perioperative hemodynamic lability (carcinoid crisis) characterized by peripheral vasodilatation and hypotension that requires IV octreotide administration.[23] Somatostatin analogues are the mainstay of medical therapy for symptom control.[4] Interferon may also be considered.

In patients with ***pulmonary hypertension*** and severe TR secondary to pulmonary thromboembolic disease, pulmonary thromboendarterectomy alone has been shown to reduce pulmonary hypertension and usually reduces TR severity without the need for concomitant tricuspid annuloplasty. TR secondary to severe primary pulmonary hypertension usually is treated with pulmonary vasodilator and diuretic therapy alone because of the risk of cardiac surgical intervention and overall poor prognosis.

Although ***percutaneous balloon mitral valvuloplasty*** may result in less TR, TV

repair combined with mitral valve replacement is better than mitral balloon valvuloplasty alone in patients with severe functional TR, especially if atrial fibrillation or RV enlargement is present.[22]

Recommendations on **pregnancy** are presented in General Principles.

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