International Journal of Cardiology 168 (2013) 707–715

Contents lists available at ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard

Review

Functional tricuspid regurgitation: An underestimated issue

Michele Di Mauro [a,*,1], Gian Paolo Bezante [b,1], Angela Di Baldassarre [c,1], Daniela Clemente [a,1], Alfredo Cardinali [a,1], Angelo Acitelli [a,1], Sara Salerni [d,1], Maria Penco [a,1], Antonio M. Calafiore [e,1], Sabina Gallina [d,1], On behalf of the Italian Study Group on Valvular Heart Disease (Italian Society of Cardiology)

[a] Department of Cardiology, University of L'Aquila, L'Aquila, Italy
[b] Division of Cardiology, Department of Internal Medicine, University of Genoa, Genoa, Italy
[c] Department of Medicine and Aging Sciences, University "G. d'Annunzio", Chieti — Pescara, Italy
[d] Department of Imaging and Neuroscience, University "G. d'Annunzio", Chieti — Pescara, Italy
[e] Department of Adult Cardiac Surgery, Riyadh, Saudi Arabia

ARTICLE INFO

Article history:
Received 22 August 2012
Received in revised form 28 February 2013
Accepted 6 April 2013
Available online 3 May 2013

Keywords:
Functional tricuspid regurgitation
Tricuspid valve repair
Tricuspid valve surgery
Right ventricular dysfunction

ABSTRACT

This review article focuses on functional tricuspid regurgitation (FTR) that has long been a neglected and underestimated entity. FTR is defined as leakage of the tricuspid valve during systole in the presence of structurally normal leaflets and chordae. FTR may be secondary to several heart diseases, more commonly mitral valve disease, pulmonary hypertension, atrial fibrillation, cardiomyopathies, right ventricular dysplasia, and idiopathic annular dilatation. The reported prevalence of moderate or greater FTR is roughly 16%, but it rises up to 89% when considering FTR of any grade. According to the recommendations of the European Association of Echocardiography, two-dimensional transthoracic echocardiography (TTE) is the first-line imaging modality for the assessment of valvular regurgitation, whereas three-dimensional TTE may provide additional information in patients with complex valve lesions. Transesophageal echocardiography may be used when TTE results are inconclusive. The natural history of FTR is unfavorable, even in less than severe tricuspid regurgitation. Data from the literature suggest that moderate or greater FTR is a risk factor for worse survival. In addition, FTR of any grade may worsen over time, which makes it reasonable to consider the correction of FTR at an early stage, preferably at the time of mitral valve surgery. Tricuspid valve annuloplasty is the gold standard surgical treatment for FTR and is associated with a recurrence rate, defined as postoperative moderate or severe FTR, ranging from 2.5 to 5.5% at 1-year follow-up.

1. Introduction

Functional tricuspid regurgitation (FTR) has long been a neglected and underestimated entity. As it usually occurs secondary to mitral valve (MV) disease, cardiologists and cardiac surgeons have long argued that if regurgitation was "functional", then it should improve when the MV is treated. However, more recently, FTR has gained increasing recognition in both clinical and surgical settings. The purpose of this review was to discuss the insights of epidemiology, pathogenesis, natural history and surgery of FTR. A special focus was placed on the need for early identification and careful quantification of FTR in order to optimize surgical indications, because the clinical course of the disease may vary according to the several etiologies of FTR.

The literature search was performed using primarily the Medline database, but other databases were also considered (CTSNet, CASPUR, Ovid, ScienceDirect).

2. Definition

FTR is a complex valvular lesion in which the tricuspid valve (TV) leaks during systole in the presence of structurally normal leaflets and chordae. FTR is considered a "ventricular" disease.

3. Etiology and epidemiology

FTR can be secondary to several heart diseases, but it is usually associated with MV disease, pulmonary hypertension, atrial fibrillation, or cardiomyopathy [1].

Calafiore et al. [2] reported a prevalence of moderate to severe FTR of up to 63% of patients with mitral stenosis. The prevalence of moderate or severe FTR ranges largely from 8% to 45% in patients undergoing MV surgery for mitral regurgitation (MR). Dreyfus et al. [3] found that

* Corresponding author at: Department of Cardiology, University of L'Aquila, P.zza Tommasi 1, 67100 L'Aquila, Italy. Tel.: +39 3286687638.
E-mail address: michele.dimauro@univaq.it (M. Di Mauro).

[1] All authors take responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

0167-5273/$ – see front matter © 2013 Elsevier Ireland Ltd. All rights reserved.
http://dx.doi.org/10.1016/j.ijcard.2013.04.043

8% of patients had moderate to severe FTR at the time of surgery. Other studies reported a prevalence of moderate or severe FTR associated with MR of 14–37% [2,4–6] (14–27% for FTR + functional MR, 15% for FTR + MV prolapse [6], and 45% for FTR + primary MR [2]). FTR rate was 25–64% in patients with either ischemic or nonischemic cardiomyopathy [7,8]. Finally, among 5223 consecutive patients undergoing echocardiography, moderate or severe FTR was observed in roughly 16% but in up to 84% of patients when considering FTR of any grade [9].

4. Anatomical remarks

The TV apparatus is very complex. The recent introduction of real-time three-dimensional echocardiography (RT3DE) has allowed to obtain new important geometric insights into the pathophysiological mechanisms underlying FTR [10,11]. The TV consists of three leaflets and a non-planar, elliptical saddle-shaped annulus. The anterior leaflet is the largest, followed by the posterior leaflet, which arises from the posterior margin of the annulus from the septum to the infero-lateral wall, whereas the septal leaflet is the smallest and arises directly from the tricuspid annulus above the interventricular septum (Fig. 1). The normal septal tricuspid leaflet inserts at a position that is slightly apical to the insertion of the anterior MV leaflet. The tricuspid annulus has a complex 3D structure, with the postero-septal portion being the lowest (towards the right ventricular [RV] apex) and the antero-septal portion the highest (towards the right atrium) (Fig. 2). The tricuspid annular area varies from 3.9 cm^2 to 5.6 cm^2, with a percentage change of approximately 30% during the cardiac cycle [12].

The subvalvular apparatus of the TV consists of the chordae tendineae and two papillary muscles (anterior and posterior). A third papillary muscle is often present too. The anterior papillary muscle provides chordae to the anterior and posterior leaflets, whereas the posterior papillary muscle provides chordae to the posterior and septal leaflets. Some chordae tendineae also arise directly from the septum.

5. Pathophysiological mechanisms underlying functional tricuspid regurgitation

In FTR, the TV leaflets fail to coapt because of the geometrical distortion of the normal spatial relationships.

Dilatation of the tricuspid annulus occurs primarily in the anterior and posterior directions, as the small septal wall leaflet is fairly fixed [13] (Fig. 3). The annulus becomes more circular with a decreased medial-lateral/antero-posterior ratio (1.11 ± 0.09 versus 1.32 ± 0.09, $p < 0.001$) [13]. Both maximum (7.5 ± 2.1 versus 5.6 ± 1.0 cm^2/m^2, $p < 0.003$) and minimum (5.7 ± 1.3 versus 3.9 ± 0.8 cm^2/m^2, $p < 0.001$) tricuspid annular areas are significantly larger in patients with FTR [12]. Annular dilatation may become irreversible over time, as clearly demonstrated in patients with chronic thromboembolic pulmonary hypertension, in whom no significant changes in annular dimensions were observed after successful pulmonary thromboendarterectomy [14]. This finding is in contrast with previous studies that considered annular dilatation as the main determinant of FTR [15].

In addition, FTR results in loss of tricuspid annular contraction, especially in severe cases (from 29.6% to 14.6%). As determined by RT3DE studies, in healthy subjects the tricuspid annular area increases from mid-systole to early diastole, decreases during mid-diastole, and increases again in late diastole. Conversely, in patients with FTR, the early diastolic peak is less frequently observed [12]. Furthermore, annular flattening may also occur, by which the tricuspid annulus loses its bimodal shape [13] (Fig. 4).

FTR may also be related to RV enlargement or dysfunction, which results in papillary muscle displacement and increased tethering forces with subsequent leaflet malcoaptation (Fig. 5). In the study of

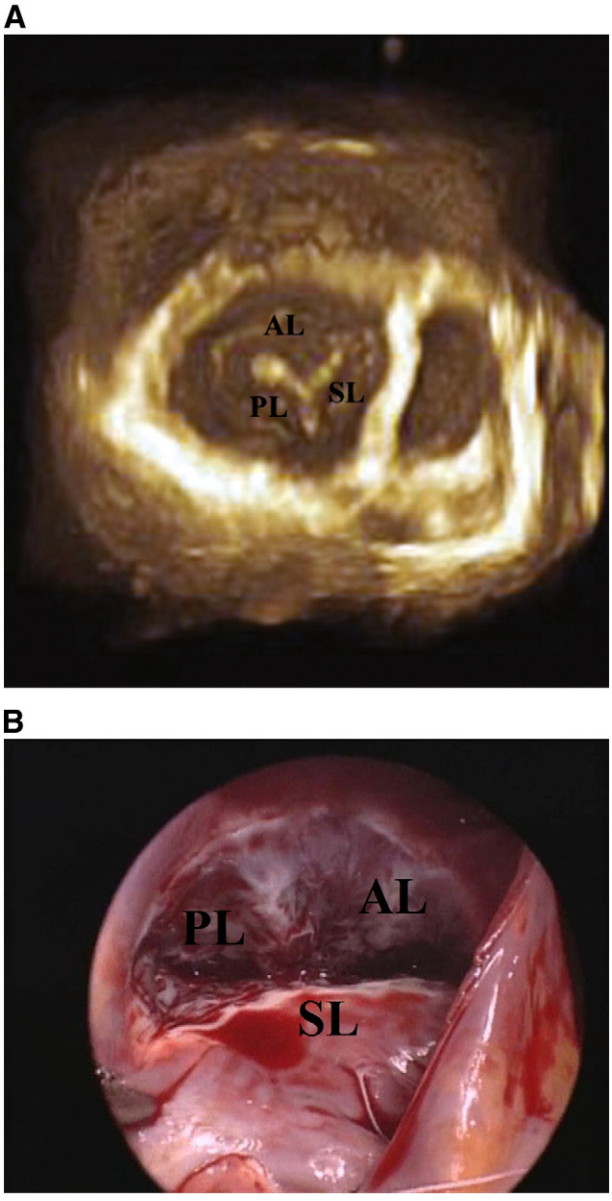

Fig. 1. Three-dimensional transthoracic echocardiography (A) and intraoperative echocardiographic view (B) of the tricuspid valve. SL = septal leaflet, AL = anterior leaflet, and PL = posterior leaflet.

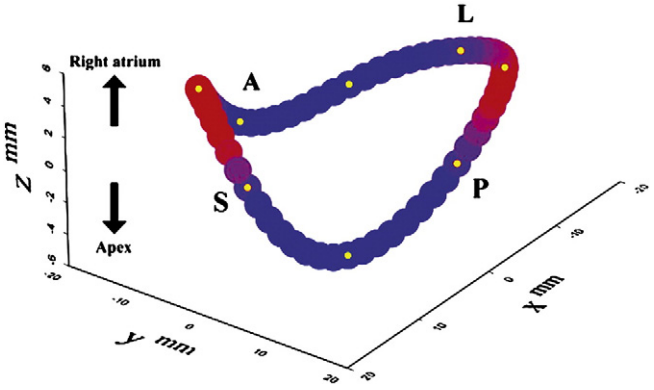

Fig. 2. The tricuspid annulus has a complex three-dimensional asymmetric shape with the postero-septal portion being the lowest (towards the right ventricular apex), and the antero-septal portion the highest (towards the right atrium).
With permission from [12].