Valvular Heart Disease in Heart Failure

Giuseppe MC Rosano

Cardiovascular Clinical Academic Group, St George’s NHS Trust Medical School, Cranmer Terrace, London SW17 0RE, UK and Department of Medical Sciences, IRCCS San Raffaele, Roma, Italy

Corresponding author:
Giuseppe MC Rosano, Cardiovascular Clinical Academic Group, St George’s NHS Trust Medical School, Cranmer Terrace, London SW17 0RE, UK
Email: giuseppe.rosano@gmail.com

Abstract

Structural valvular heart disease may be the cause of heart failure or may worsen the clinical status of patients with heart failure. Heart failure may also develop in patients treated with valve surgery. Patients with heart failure with valvular heart disease are at increased risk of events including sudden cardiac death. Before considering intervention (surgical or percutaneous) all patients should receive appropriate medical and device therapy taking into account that vasodilators must be used with caution in patients with severe aortic stenosis. Numerous percutaneous and/or hybrid procedures have been introduced in the past few years and they are changing the management of valvular heart disease. In patients with heart failure and valvular heart disease, either primary or functional, the whole process of decision-making should be staged through a comprehensive evaluation of the risk–benefit ratio of different treatment strategies and should be made by a multidisciplinary ‘heart team’ with a particular expertise in valvular heart disease. The heart team should include heart failure cardiologists, cardiac surgeons/structural valve interventionists, imaging specialists, anaesthetists, geriatricians and intensive care specialists. This article will review recent developments and distill practical guidance in the management of this important heart failure co-morbidity.

Keywords: Heart Valve disease; Heart failure; Cardiovascular imaging; Echocardiography; Guidelines

Citation: Rosano GMC. Valvular Heart Disease in Heart Failure. International Cardiovascular Forum Journal 2017;10:70-72, DOI: 10.17987/icfj.v10i.453

Introduction

Structural valvular heart disease may be the cause of heart failure or may worsen the clinical status of patients with heart failure. Heart failure may also develop in patients treated with valve surgery.[1,2] Patients with heart failure with valvular heart disease are at increased risk of events including sudden cardiac death. Before considering intervention (surgical or percutaneous) all patients should receive appropriate medical and device therapy taking into account that vasodilators must be used with caution in patients with severe aortic stenosis. Numerous percutaneous and/or hybrid procedures have been introduced in the past few years and they are changing the management of valvular heart disease.

Aortic stenosis

Aortic stenosis occurs in nearly 2.8% of patients ≥75 years of age and can occur because of degenerative calcification and congenital valvular defects such as bicuspid aortic valves or as one of the sequelae of rheumatic disease.[3]

One of the major tasks in patients with HFrEf with aortic valve disease is the adequate assessment of the degree of stenosis since ‘low-flow, low-gradient’ aortic stenosis (ΔPmean <40 mm Hg) can be both a pseudostenosis due to a co-existent cardiomyopathy or be a manifestation of a significant understimation of the degree of aortic stenosis and its clinical significance because of the added effects of diseased left ventricle reducing the pressure head driving blood across the diseased valve.[4] Patients with pseudostenosis have moderately diseased aortic valves with impaired leaflet opening because of the reduced left ventricular function. The identification of pseudostenosis is of crucial importance since, unlike true aortic stenosis, it carries a high mortality risk (nearly 50%) with surgical aortic valve replacement while it benefits more from medical therapy for heart failure.[5] Dobutamine stress either during echocardiography or catheterization is the main method of differentiating pseudostenosis from true aortic valve stenosis. During the dobutamine test the contractility increases the stroke volume and will increase the trans-aortic pressure gradient and velocity while the effective orifice area will not change in the patient with true severe aortic stenosis. In patients with pseudostenosis, dobutamine infusion with the consequent increase in stroke volume will open the aortic valve causing no change in pressure gradient and velocity resulting in an increased calculated valve area confirming the moderate degree of aortic stenosis.[4,5]

In symptomatic patients with heart failure and a mean gradient >40 mmHg, left ventricular function is not a limitation for aortic valve replacement. In patients with heart failure and severe aortic stenosis who are not suitable for surgery as assessed by a “heart
team Transaortic valve implantation (TAVI) is recommended if a post-TAVI survival of at least one year is predicted. The heart team has a central role in deciding whether TAVI should be also considered in high-risk patients with heart failure and severe aortic stenosis who may be suitable for surgery, but in whom TAVI is favoured on the basis of individual risk profile and anatomic appropriateness.[6–9] The field of TAVI is quickly evolving and recent trials in patients with both moderate and severe aortic stenosis have shown that TAVI confers a significantly higher rate of event free survival at 1 year.[6–9]

Aortic regurgitation
Aortic regurgitation (AR) increases the load on the left ventricular in ventricles with already reduced function and therefore significantly aggravates heart failure. Aortic valve repair or replacement is recommended in all patients with heart failure who are symptomatic and have severe aortic regurgitation and in those who are asymptomatic with severe AR but otherwise fit for surgery.[10,11]

Mitral regurgitation
Patients with heart failure may present with mitral regurgitation (MR) because of a primary valve disease or rupture or may represent a functional consequence of a dilated hypokinetic left ventricle. In symptomatic patients with severe organic MR with no contra-indications mitral valve replacement is indicated.[12–13] Available surgical expertise is a major factor in deciding whether to replace or repair the mitral valve but the decision is individualised by the heart team and depends on factors such as mitral valve anatomy, co-existing coronary disease, other co-morbidities and the overall clinical condition of the patient. Although surgical repair may durably improve symptoms, its long term effect on survival in patients with HFpEF and left ventricular EF <30% is unknown. In this condition, the decision to operate should take into account the overall clinical status, the presence of co-morbidities and the response to medical therapy. In this case the choice to repair or replace the mitral valve lies with the surgical team.[12–13]

Functional mitral regurgitation
Functional mitral regurgitation (FMR) is the consequence of left ventricular enlargement and remodelling that leads to reduced mitral valve leaflet closure and coaptation. FMR is often present in patients with HFpEF and is a marker of adverse outcomes and poor long-term survival when present.[14,15] FMR may be variable and can also contribute to progressive left ventricular dilatation and dysfunction and worsened symptoms. Anatomical changes can lead to a progression of FMR with an average annual increase of 5–8 ml in regurgitant volume and 4–6 mm² in the effective regurgitant orifice area.[16]

In patients with FMR, the valvular apparatus (papillary muscles, chordae, and valve leaflets) is typically normal. Mitral leaflet mal-coaptation and mitral valve regurgitation may result from a combination of papillary muscle displacement due to left ventricular remodeling, decreased systolic closing force and annular enlargement secondary to left ventricular dilatation. Progression of left ventricular dilatation and remodeling, aggravated by the volume overload imposed by FMR, amplifies leaflet tethering increasing the inter-papillary muscle distance and a lateral redirection of the tethering forces, which are normally perpendicular to the mitral annular plane, resulting in a further progression of FMR.[17]

Effective medical therapy (including CRT in patients with clear indications) may reverse left ventricular remodelling and may reduce FMR. Therefore, every effort should be made first to optimize medical treatment in patients with heart failure presenting with FMR. In symptomatic patients with heart failure of ischaemic origin and left ventricular EF <30% combined valve and coronary surgery should be considered if there is evidence of myocardial viability and a coronary anatomy suitable for revascularization. Mitral valve surgery is recommended also in those patients with heart failure and left ventricular function <30% and severe mitral regurgitation undergoing CABG.[18] The addition of mitral valve repair to CABG, however, has not clearly proven to have a significant effect on left ventricular reverse remodeling in patients with moderate, secondary ischaemic mitral regurgitation as there is no evidence favouring mitral valve repair over replacement. In patients with heart failure and FMR and atrial fibrillation, atrial ablation and LA appendage closure may be considered at the time of mitral valve surgery.

In patients with severe FMR and severe left ventricular dysfunction (LVEF <30%) who have non-ischaemic cardiomyopathy or who are not suitable for myocardial revascularisation the indication for mitral valve surgery is debatable, and medical and device therapy is to be preferred, even though in selected patients mitral valve repair may be considered in order to slow the progression of the disease.[18] In patients with heart failure and moderate-severe FMR who are judged at high surgical risk, percutaneous edge-to-edge repair with the Mitra-Clip device or with newer devices may be considered in order to improve symptoms and quality of life. However there is no evidence that this approach leads to any long term prognostic improvement.[19,20]

Tricuspid regurgitation
Primary tricuspid regurgitation (TR) is an extremely rare cause of heart failure. In secondary tricuspid regurgitation the leaflets and chords themselves are not primarily affected but the tricuspid annulus stretches because of right ventricular failure or enlargement as a consequence of elevated pulmonary pressures. Functional tricuspid regurgitation frequently complicates the natural course of patients with heart failure. A “V” wave on the jugular venous pulse is a clear sign of tricuspid regurgitation in patients with heart failure. When tricuspid regurgitation becomes severe it may worsen symptoms of right heart failure.

In these patients adequate diuretic therapy is important also because hepatic congestion that is often present in these patients may contribute to hyperaldosteronism. Therefore, the addition of an MRA (at higher natriuretic doses) may improve decongestion. [21] There are presently no clear indications for surgical correction of functional TR in patients with heart failure.[18] The correction of TR, if appropriate, may be considered at the time of surgical correction of left-sided valve lesions but this is a decision to be taken on an individual patient level taking into account the fact that the additional procedure prolongs surgical procedure times and therefore risk. Although catheter-based interventions have been proposed for the correction of functional tricuspid regurgitation, their role and safety remains inadequately studied.[22]
Conclusions
In patients with heart failure and valvular heart disease, either primary or functional, the whole process of decision-making should be staged through a comprehensive evaluation of the risk–benefit ratio of different treatment strategies and should be made by a multidisciplinary ‘heart team’ with a particular expertise in valvular heart disease. The heart team should include heart failure cardiologists, cardiac surgeons/structural valve interventionists, imaging specialists, anaesthetists, geriatricians and intensive care specialists.

Declaration of Interest
The author has no conflicts of interest to declare.

Acknowledgements
The author states that he abides by the “Requirements for Ethical Publishing in Biomedical Journals” [23].

References