The Management of Functional Mitral Regurgitation: The Role of Cardiac Resynchronization Therapy

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Abstract

Functional Mitral Regurgitation (FMR), which develops in Heart Failure (HF) settings as a result of changes in the geometry of the Left Ventricle (LV) and left atrium, is characterized by valve leaflets and chordae that are structurally normal. In this situation, FMR lowers life expectancy and raises mortality. There is still no universal cure for many patients with FMR, despite several medicinal and surgical treatments. FMR's pathophysiology is extremely intricate and involves numerous underlying pathways. Left ventricular dyssynchrony is a significant target for FMR therapy since it may contribute to the initiation and progression of FMR.

Keywords: Cardiac resynchronization therapy • Functional mitral regurgitation • Dyssynchrony heart failure • LV remodeling

Introduction

Over FMR is a pathological state in which the mitral valve is physically normal and the disease is brought on by valve distortion brought on by abnormal Left Ventricular (LV) remodeling and dysfunction. Both ischemia and non-ischemic cardiac disease can cause it. The prevalence of ischemic FMR ranges from 20% to 50%, and moderate or severe FMR is linked to higher rates of morbidity and mortality, including a 1.6-fold higher risk of death at the 5-year follow-up and a 3-fold higher risk of Heart Failure (HF). On the other hand, non-ischemic FMR is seen in individuals with LV dysfunction in 50% of instances (between 56% and 65%) and is linked to elevated cardiac mortality. HF episodes have 3 times and 2 times the risk of these events, respectively. Multiple variables, including diminished contractility, ventricular remodeling, impaired mitral annular function, and ventricular dyssynchrony, contribute to FMR. In addition, persistent volume overload contributes to the progression of FMR over time, as the adage "mitral regurgitation begets mitral regurgitation" states. Reverse LV or annular remodeling, decreased Mitral Regurgitation (MR) severity, or both may be able to break the vicious cycle between cardiac remodeling and FMR. Through the reduction of transmitral pressure gradients, preload, and afterload, pharmacotherapy can affect both goals. However, HF and MR have a dismal prognosis after they stop responding to medication, and there is currently no clear-cut treatment that can stop the progression of the disease. Resynchronization may help patients with HF and FMR who are eligible for Cardiac Resynchronization Therapy (CRT) by reducing the degree of FMR, which may increase survival and alleviate symptoms. LV dyssynchrony, however, is merely a part of a larger complicated illness with numerous diverse processes. Additionally, it has been noted that some patients have poor responses to CRT and no change in FMR. Additional device treatments (such percutaneous mitral regurgitation intervention) ought to be taken into consideration in such circumstances.

The MV apparatus modifications in HF that may be complex and heterogeneous are the primary factors of FMR. The MV leaflets typically overlap significantly when they are facing one another and close in a coaptation point within the annular plane (coaptation reserve). The coaptation reserve in FMR is gradually diminished until coaptation is no longer possible. The predominance of tethering forces causes Papillary Muscles (PMs) to dislocate away from the mitral annulus, which is the primary cause of the loss in coaptation reserve. As a result, PMs frequently pull MV leaflets via the chordae tendinae that are attached, increasing the tethering tensions. In addition, closing forces are reduced, primarily as a result of LV systolic dysfunction and, in particular, LV dilatation, which results in inadequate MV closure. This imbalance of the two opposing pressures causes what is known as the "loitering pattern," a typical phasic intra-beat variation in the time course of the regurgitant orifice area in FMR. In early and late systole, when the higher peak LV pressure makes it FMR severity may vary from beat to beat in addition to intra-beat variability. The phasic variations in the equilibrium between the tethering and closing forces, as well as the physiological and/or pharmacologic elements that have the ability to alter this equilibrium, are what determine this property. Exercise increases the cardiac load and may also result in LV and mitral valve apparatus dynamic geometric changes that exacerbate MR. Particularly, volume loading during isotonic exercise causes ventricular dilatation (end-diastolic volume and end-systolic volume both tend to rise) but does not affect ejection fraction easier to close the valve, the orifice area and regurgitation are greater. Additionally, exercise may cause the LV to contract dyssynchronically with a (rate-dependent) conduction delay. The exercise-induced rise in LV sphericity, which increases PMs distance and subsequently modifies mitral valve geometry, is what, however, primarily contributes to MR deterioration. The location and degree of myocardial scar play a significant effect in dynamic MR alterations in ischemic cardiomyopathy. Exercise causes PMs to be displaced apically in myocardial infarctions of the anterior wall that reach the apical portions of the anterior wall, worsening MR and deepening coaptation. Exercise worsens MR in a distinct way in individuals with inferior wall infarction: localized wall motion abnormalities bind the mitral valve more posteriorly, increasing the annular dimension. By revealing its dynamic properties, the echocardiographic measurement of MR during exercise may reveal increased severity and offer prognostic information. In fact, bigger increases in MR during exercise are linked to a higher risk of death and hospital admissions for HF that is getting worse. MR alterations can also be influenced by pharmacologic variables. In fact, by reducing preload, diuretics cause a decrease in ventricular size, which in turn causes a drop in tethering forces and, ultimately, in MR. The geometry (size and form) and mobility (sphincter function) of the annulus are compromised. The annulus tends to take on a flattened monoplane shape due to the alteration of the normal saddle morphology. Additionally, anterior myocardial infarction has a greater degree of this geometric deformation. Mitral valve tenting causes coaptation loss, which worsens FMR when combined with the imbalance of tethering and closing forces. Butamine infusion is an example of an inotropic drug that alters dP/dt and the closure forces, lowering MR. Mitral annular enlargement caused by left atrial dilatation in the context of atrial fibrillation may also aggravate FMR in individuals with HF. This process is sometimes referred to as atriogenic leaflet tethering, which means that left atrial dilatation stretches the posterior mitral leaflet across the LV wall, which is followed by the displacement or tethering of the mitral valve leaflets away from the PMs. Last but not least, some studies imply that functional mitral regurgitation has an organic component. It has been demonstrated that under loading conditions, HF patients' MV leaflets change structurally and biochemically, with a notable rise in glycosaminoglycans and collagen. It was also noted that these valves in HF have higher cell concentrations and lower water contents. As a result, the higher cell density suggests an elevation of mitogenic activity, leading to a rise in matrix synthesis. Reduced viscous relaxation is probably caused by

the loss of water, which is an important part of the extracellular matrix and enhances tissue viscoelasticity. As a result, these compositional changes could have an impact on how well the valves perform by making them stiffer, less extensible, and less viscous. LV mechanical dyssynchrony may cause FMR through a number of different methods. The displacement of PMs, which results in a lack of leaflet coaptation, and uneven regional mechanical activation caused by mechanical dyssynchrony both contribute to the deformed MV apparatus shape, which in turn raises the tethering force in dual modes. Additionally, LV dyssynchrony causes an increase in pressure between the left atrium and the LV as a result of a change in the timing of the atrioventricular contraction and relaxation cycles, which results in a diastolic FMR during incomplete mitral valve closure. Finally, LV dyssynchrony impairs mitral valve tenting by reducing LV contractility and, subsequently, closing forces.

The first step in addressing all patients with FMR should be appropriate medical therapy, according the recommendations for managing HF. According to ESC Guidelines, Cardiac Resynchronization Therapy (CRT) should be considered in patients with high blood pressure who meet ECG and LV function requirements or regardless of NYHA class or QRS width in patients who have a need for ventricular pacing due to high degree AV block. The lowering of FMR as a result of better, coordinated timing of mechanical activation of PMs insertion sites is the initial positive effect of CRT. On the other hand, LV reverse remodeling is what causes the long-term decline in FMR. Acutely, the improved contraction efficiency brought on by global LV resynchronization also significantly lowers FMR. This is particularly relevant when resynchronization affects basal and mid-LV parts as well as apical segments for a global effect.

The simultaneous recruitment of a larger section of the LV myocardium is associated with an initial electromechanical improvement in CRT patients receiving an LV lead with multiple selectable electrodes. The effects of CRT on FMR, however, may be restricted in ischemic patients with high scar load because the activation site may not be able to support LV contractility. Additionally, the LV lead cannot always be positioned by the implanting electro physiologist in the "optimal position" to increase myocardial survivability. An abrupt reduction in volume overload causes an instantaneous FMR reduction after CRT implantation, which is a manifestation of the tethering and closing forces being balanced. After CRT, the initial FMR drop has been shown to be a reliable prognostic indicator and also favors reverse remodeling. Patients with at least mild MR, but not severe MR, should derive the most benefit from CRT for FMR and LV remodeling since severe MR may indicate a level of LV dysfunction and dilatation that is too advanced and perhaps irreversible to benefit from electrical therapy. Dyssynchrony is merely one element of an illness that is quite complicated, hence CRT alone has some inherent limits for FMR resolution.

Conclusion

In the setting of HF, FMR has a major impact on survival rather than being a passive observer. As a result, FMR and HF should be viewed as a singular, complex disease rather than as two distinct disorders. A multilevel strategy that aims to treat all of the HF's underlying causes and difficulties should therefore be favored. In this regard, the multidisciplinary Heart Team, with its several subspecialties, will play a crucial part in determining the best course of action for such a complex and potentially fatal problem.

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