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Commentary

Prognostic Significance of Mitral Valve Prolapse

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DESCRIPTION

A frequent valvular condition called Mitral Valve Prolapse (MVP) has been linked to heart failure, ventricular arrhythmias, sudden cardiac death, and Mitral Regurgitation (MR). It is crucial to assess Left Ventricular (LV) function and remodeling in addition to mitral valve shape and regurgitation given the prognostic significance of these disorders. Regarding the underlying processes of LV remodeling in the setting of non-syndromic MVP, a number of potential ideas have been put up too far, but the precise pathophysiological explanation is still difficult.

In general, volume overload associated with severe MR is thought to be the primary factor causing LV dilatation in MVP. But in patients with MVP and no/mild MR, notably in those with MVP or Barlow's disease, considerable LV remodeling has been seen, leading to the development of various novel ideas. A large volume load was recently added to the LV on top of the transvalvular MR volume with the idea of "prolapse volume." Given the connection between MVP and cardiac fibrosis, a concurrent cardiomyopathy is another possibility. This cardiomyopathy may have a hereditary basis, a secondary cause (such as an additional factor added to a genetic propensity), or recurrent ventricular ectopic beats as its cause.

This study include MVP assessment using various imaging modalities as technical aspects of imaging modalities affect the assessment of MR severity and LV remodeling, which may in turn influence clinical decision-making in these patients. Since 1887, the presence of a systolic murmur and midsystolic click has been documented.

The regurgitation was caused by excessive posterior leaflet motion into the left atrium during systole, and then first assumed that rheumatic illness was the underlying mechanism of MR and hence it is anomaly mitral valve prolapse. Histological analysis was used to explain the macroscopic features of the Barlow's valve, such as widespread thickening of the leaflets and extension of the chordae, and myxomatous degeneration was determined to represent the underlying mechanism rather than rheumatic illness.

However, another type of mitral valve prolapse known as FED, which is mostly caused by choroidal rupture and lacks excess leaflet tissue has been discovered. It will include MVP assessment using various imaging modalities as technical aspects of imaging modalities affect the assessment of MR severity and LV remodeling, which may in turn influence clinical decision-making in these patients.

A frequent valvular condition with a frequency of 2-3% in the general population is Mitral Valve Prolapse (MVP). In general, two primary MVP subtypes that correspond to opposite extremities of the illness spectrum may be identified. At one extreme, Barlow's Disease (BD) is defined by the dilatation of the mitral annulus as well as the lengthening, thickness, and prolapse of both leaflets, which are frequently related to mitral annular disjunction. Contrarily, Fibroelastic Deficit (FED), which affects individuals between the ages of 50 and 70, is marked by single leaflet or segment prolapse, chordal elongation or rupture, and thickening of the prolapsing leaflet segments.

MVP can be linked to Mitral Regurgitation (MR), LV dysfunction and remodeling with heart failure, ventricular arrhythmias, and sudden cardiac death, even though some individuals stay asymptomatic for a long time. In many cardiovascular disorders, including MVP, LV remodeling and dysfunction play a significant role in disease progression and poor prognosis. The precise pathophysiological mechanism causing LV remodeling in MVP is still unknown, and the underlying processes are only partially understood. Volume overload associated with MR is typically thought to be the primary mechanism causing LV remodeling in MVP. Therefore, in cases of severe MR, current recommendations advise surgical mitral intervention.

The discovery that LV dilatation and dysfunction, particularly in individuals with BD, might be disproportionate to the degree of MR has, however, cast doubt on this idea. When adjusted for MR volume, LV dilatation beyond the upper limit of normal for one's age and gender indicates disproportionate LV remodeling in MVP. The prolapse volume, concurrent cardiomyopathy caused by myocardial fibrosis and ventricular arrhythmias, hereditary susceptibility, and other recent theories have all been put forth to explain this abnormal LV remodeling in MVP.

An increasing amount of studies suggests that non-syndromic MVP is also a hereditary disorder with autosomal dominant or X-linked inheritance, in addition to syndromic types of MVP (like Marfan syndrome).

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