



POLISH HEART JOURNAL

Kardiologia Polska

The Official Peer-reviewed Journal
of the Polish Cardiac Society
since 1957

Online first

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ISSN 0022-9032

e-ISSN 1897-4279

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Article type: Editorial

Received: December 9, 2024

Accepted: December 13, 2024

Early publication date: December 18, 2024

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Ventricular displacement of the mitral annulus: An ignored anatomical variant or a potential cardiovascular risk factor?

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Related article

by Krawczyk-Ożóg et al.

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The mitral valve complex includes leaflets, chordae, mitral annulus, papillary muscles, and the left ventricular myocardium. Adequate mitral valve function requires structural and physiologic integrity of all of these components. The “mitral annulus” does not represent a complete, rigid ring but rather a complex, D-shaped structure consisting of fibrous, muscular, and adipose tissue [1, 2].

In 1986, Hutchins et al. [1] reviewed histologic sections from the mitral annulus and found that most mitral leaflet attachments were found at the connection between the left ventricular myocardium and the left atrium. In others, however, the attachment was found lower in the ventricle or somewhat separated from the atrial aspect of the left ventricular myocardium by a tendinous attachment. The latter, Hutchins et al. [1] called “disjunction” when they found a wide separation between the left ventricular myocardium and the atrium-valve junction. Unfortunately, “wide” disjunction was not further defined [1]. Since then, most literature focused on mitral annular disjunction (MAD) with atrial displacement [3–5] as initially described by Hutchins et al. [1].

Although the clinical implications of MAD have remained controversial for years, its relationship to ventricular arrhythmia is increasingly recognized in the literature, particularly in patients with a greater extent of MAD and late gadolinium enhancement [6]. Here, anatomical alterations of the mitral valve complex may increase the risk for associated myocardial inflammation, fibrosis and tissue conductance [7].

Recently, Krawczyk-Ożóg et al. [8] described the morphology and prevalence of ventricular displacement of the mitral leaflet attachment in specimens. In their work, published 2024 in *Rev Esp Cardiol*, they called this entity ventricular mitral annular disjunction (v-MAD) and defined it as a spatial displacement of the mitral leaflet hinge line by more than 2 mm towards the left ventricle [8]. In a cohort of 224 specimens without known cardiovascular disease, v-MAD was found in 24% of cases with the most frequent site being the P2 scallop (20% of all specimens) [8].

While echocardiography [9] and cardiac magnetic resonance imaging [4] are often used to non-invasively assess MAD, computed tomography may be even superior to assess MAD in comparison to both of these modalities due to the superior spatial resolution and the possibility to assess the mitral valve complex with multiplanar reformations in any arbitrary plane [2, 5]. As a drawback, cardiac computed tomography (CT) images have to be acquired in systole to assess MAD, which is not regularly performed when cardiac CT is done to assess coronary artery disease, where usually only diastolic images are acquired.

The present study by Krawczyk-Ożóg et al. [8] focused on the detection of "classical" MAD with atrial disjunction and v-MAD in 250 patients using cardiac CT. The vast majority of these patients (97%) underwent cardiac CT to evaluate aortic valve stenosis or coronary artery disease. The MAD with atrial disjunction was identified in 26% of patients and the v-MAD was identified in 28% of these patients. No patient showed the simultaneous presence of "classical" MAD and v-MAD. Both, MAD with atrial disjunction and v-MAD were most frequent at the attachment of the P2 scallop.

Krawczyk-Ożóg et al. [8] excellently showed that v-MAD may not only be detectable histologically [8] but also with cardiac CT [10], with similar results regarding the most frequent location. Whilst MAD has gained a lot of awareness in the scientific literature of recent years, the presence of v-MAD remained under the radar of almost all of these studies. Therefore, whether v-MAD is an anatomical variant or a pathological condition and whether v-MAD along with "classical" MAD also has the potential of an arrhythmogenic substrate needs future evaluation. Additional issues that should be evaluated are: 1) whether v-MAD can be reliably detected using echocardiography and cardiac magnetic resonance imaging; 2) whether patients

with v-MAD are more prone for myocardial fibrosis and/or mitral valve disease such as myxomatous mitral valve, and 3) whether the presence of v-MAD varies for different patient cohorts and patients of different age. In addition, it remains to be determined how reproducible the assessment of vMAD in cardiac CT might be, as the single slice illustrating the presence of v-MAD (Panel B) might not be recognized to the same extent by all cardiac imagers.

In conclusion, the study of Krawczyk-Ożóg et al. [8] raises some interesting questions on the implications of v-MAD. Further studies should evaluate the clinical significance of v-MAD and adding answers to the question whether v-MAD represents a benign anatomical variant or another potential cardiovascular risk factor.

Article information

Conflict of interest: None declared.

Funding: None.

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