Commentary

The evolution of mitral valve prolapse: insights from the Framingham Heart Study

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Abstract: The Framingham Heart Study group has described the non-diagnostic variants may evolve into mitral valve prolapse over time. These non-diagnostic variants include minimal systolic displacement, and abnormal anterior coaptation which is measured on surface echocardiography. Computed tomography and cardiac magnetic resonance imaging are evolving and can assess the degree of mitral regurgitation (MR); imaging techniques aside, genetic and proteomic detection of mitral prolapse is also evolving. However, the genetic basis for mitral prolapse is complex and likely involves multiple genetic loci. The same is also true for work determining possible biomarkers associated with mitral prolapse. The present study may be useful in counseling patients with a family history of mitral prolapse. Registry data is therefore of paramount importance in providing unbiased insight into this common disease.

Keywords: Epidemiology; echocardiography; valvular heart disease

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Mitral valve prolapse is common with a reported prevalence ranging from 2–3% (1,2). Many will go on to develop severe mitral regurgitation (MR) requiring intervention. However, the timeline for disease progression remains elusive. Data from the Framingham Heart Study (1,2) has been important in determining the natural history of degenerative mitral valve disease.

The Framingham Heart Study group has previously described several non-diagnostic mitral valve morphologies that can be viewed as pre-prolapse variants (1,2). Based on their data, including from the present study, these non-diagnostic variants may evolve into mitral valve prolapse over time. These non-diagnostic variants include minimal systolic displacement, and abnormal anterior coaptation which is measured on surface echocardiography. The primary advantage of this classification scheme is the wide spread availability of echocardiography.

In fact echocardiography remains the most relevant diagnostic modality for mitral prolapse. Computed tomography is limited by the need for ionizing radiation and the inability to accurately diagnose mitral prolapse in certain patient subsets (3,4). Cardiac magnetic resonance imaging is evolving and can accurately assess the degree of MR; however, leaflet thickening is challenging to assess given current limitations in spatial resolution (5). The role of delayed enhancement in the papillary muscles among mitral prolapse patients also remains unclear (6). Nevertheless, detecting abnormal anterior coaptation and minimal systolic displacement requires expertise as these changes are often subtle and requires familiarity with mitral prolapse variants (1).

Imaging techniques aside, genetic and proteomic detection of mitral prolapse is also evolving. Recent work from the Leducq MITRAL Network analyzing 1,412 mitral prolapse cases compared with 2,439 controls revealed a LMCD1 (LM and cysteine-rich domains 1), which encodes a transcription factor associated with atrioventricular valve regurgitation (7). Familial analysis has also implicated DCHS1 and beta-adrenergic receptor polymorphisms as playing a role in mitral prolapse (8,9). However, the role of genetic testing in risk-stratifying patient's remains years away from clinical application especially since the genetic basis for mitral prolapse is complex and likely involves multiple genetic loci. The same is also true for work

determining possible biomarkers associated with mitral prolapse. Proteomic analysis has implicated reduced levels of haptoglobin, platelet basic protein, and complement component C4b in patients with mitral prolapse and MR (10). However, this work is confined to small patient numbers and may not be applicable to the population of patients with prolapse.

One of the other important findings of the present study relates to the authors determination of patient risk associated with mitral prolapse. Previous work has suggested a benign clinical course for patients with mitral valve prolapse; however, in the present study, patients with prolapse trended towards worse adjusted mortality compared to patients without prolapse (hazard ratio 1.7, P=0.08). Although the severity of MR was not reported for patients in this study, this information appears to agree with work from the Mayo Clinic, which has reported a 33% cardiac event rate, 5-year after diagnosis of asymptomatic MR (11). Notwithstanding the limitations of the present study, this information may useful in counseling patients with a family history of mitral prolapse; although, the absolute clinical risk likely is small.

Overall, the data describing the natural history of mitral valve prolapse is scarce, especially considering the prevalence of the disease. Registry data is therefore of paramount importance in providing unbiased insight into this common disease.

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Footnote

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