Clinical Cardiology Reserch and Reports

www.alcrut.com

Review Article | Clinical Cardiology Research and Reports | Open Access

A Retrospective Analysis of Mitral Valve Prolapse

Bonow N*, Zuppiroli N, Roman B, Galloway F, Biaggi H, Hortop G

Department of Cardiology, Tajik State Medical Universit, Tajikistan

***Corresponding Author**: Bonow N, Department of Cardiology, Tajik State Medical Universit, Tajikistan.

Received Date: March 08, 2022; **Accepted Date:** March 22, 2022; **Published Date:** March 28, 2022

Citation: Bonow N*, Zuppiroli N, Roman B, Galloway F, Biaggi H, Hortop G, A Retrospective Analysis of Mitral Valve Prolapse, J. Clinical Cardiology Research and Reports, V1(2).

Abstract

Mitral valve prolapses (MVP), also known as floppy mitral valve syndrome, systolic click-murmur syndrome, and billowing mitral leaflets, is a valvular heart disease disorder. It is a benign condition. In rare cases, it may present with sudden cardiac death, endocarditis, or a stroke. The condition affects nearly 3% of the United States population. The disorder produces symptoms as a result of a redundant and abnormally thickened mitral valve leaflet prolapsing into the left atrium during systole.

Keywords: Myxomatous degeneration; echocardiography; exertional dyspnea; electrocardiographic repolarization abnormalities

Introduction

MVP is a clinical entity that is not fully understood, despite being known for more than a century. A 'mid-systolic click' was first described in 1887 by Cuffer and Barbillon. In 1963 Barlow demonstrated the presence of MR by angiography in patients with the 'click-murmur' syndrome. Criley subsequently coined the term mitral valve prolapse.

MVP may be familial or sporadic. Despite being the most common cause of isolated MR requiring surgical repair little is known about the genetic mechanisms underlying the pathogenesis and progression of MVP. Studies on the heritable features of MVP have been limited by the analysis of relatively small pedigrees and by self-referral and selection biases, including a preponderance of data from hospital-based cohorts. Nonetheless, a majority of data favors an autosomal dominant pattern of inheritance in a large proportion of individuals with MVP. Despite the variability in the clinical features, familial MVP might be considered a prevalent Mendelian cardiac abnormality in humans. While filamin A has been identified as causing an X-linked form of MVP, the causative genes for the more common form of autosomal dominant MVP have yet to be defined. In this review we summarize our current knowledge regarding the diagnosis, epidemiology, prognosis, pathophysiology, and genetics of MVP, with a focus on potential future research directions.

In the early days of 2D echocardiography, the diagnosis of MVP occurred with prevalence ranging from 5 to 15%, and even as high as 35% of those undergoing imaging. In part, this over-diagnosis was due to the erroneous assumption that the mitral valve (MV) was planar; thus, any sonographic view that showed excursion of the leaflets superior to the mitral annulus was deemed pathological. Pivotal echocardiographic work in the late 1980s redefined normal mitral anatomy Using three-dimensional (3D) echo imaging, Levine and colleagues established that the mitral annulus was in fact saddle-shaped.

Clinical Characteristics of MVP

A diagnosis of MVP is not reliably made using clinical symptoms. In fact, patients with MVP appear to exhibit symptoms that have been previously attributed to MVP (dyspnea, chest pain, and electrocardiographic abnormalities) at an equivalent rate to patients without any identifiable prolapse. Even patients with quite severe regurgitation as a result of prolapse may be asymptomatic. As such, symptoms should not be used to diagnose MVP, although the presence of symptoms warrant further investigation. Physical exam findings that are associated with MVP include a lower body mass index and a mid-systolic "click" heard best at the apex on cardiac auscultation. A late systolic or holosystolic murmur may be appreciated as well, and suggests the presence of mitral regurgitation, Thoracic, or bony abnormalities, as well as other extra-cardiac findings may suggest the presence of MVP as part of a syndrome.

Mitral Aorta Skeleton and Skin Phenotype

The mitral, aorta, skeleton, and skin (MASS) phenotype is a marfanoid syndrome that consists of phenotypes involving MASS, but that doesn't meet the Ghent criteria for Marfan syndrome. As would be expected, MVP is seen at a high prevalence in patients with MASS phenotype.

Ebstein Anomaly

Ebstein anomaly is a developmental heart defect characterized by inferior displacement of the proximal attachments of the tricuspid valve leaflets from the atrioventricular valve ring, resulting in an "atrialized" portion of the right ventricle. Ebstein anomaly occurs in ≈1 in 14 000 live births, and has been associated with other cardiovascular defects, including MVP.

Familial Myxomatous Valvular Degeneration

Familial myxomatous valvular degeneration, also called familial cardiac valvular dystrophy, is a heterogeneously defined group of disorders characterized by myxomatous degeneration in multiple heart valves.

Common risk alleles for MVP: Genome wide association studies

Alternatively, the identification of frequent genetic variants showing higher allele frequencies in patients compared to controls allowed the identification of genes and biological pathways involved in MVP. Genome-wide association study analyses use frequent, genome-wide single-nucleotide polymorphisms (SNPs), and combine these results with information on expression data and epigenetic marks in biological tissues of interest, possibly involved in the disease.

Conclusion

MVP is a common clinical phenotype and remains the most common valvular pathology requiring surgery. Multiple loci for autosomal dominant non-syndromic MVP and a gene responsible for a rare, X-linked form of MVP have been discovered. Studies in a mouse Marfan model and in clinical specimens of excised myxomatous mitral valves have underlined the role of excessive TGF-β signaling in the development of degenerative MV disease and the potential of angiotensin I receptor blockade in limiting MVP progression.

References

1. Devereux RB, Jones EC, Roman MJ, Howard BV, Fabsitz RR, Liu JE, Palmieri V, Welty TK, Lee ET. Prevalence and correlates of mitral valve prolapse in a population-based sample of American Indians: the Strong Heart Study. Am J Med. 2001;111:679–685.
2. Chesler E, King RA, Edwards JE. The myxomatous mitral valve and sudden death. Circulation. 1983;67:632–639
3. Zdrojewski TR, Wyrzykowski B, Krupa-Wojciechowska B. Renin-

aldosterone regulation during upright posture in young men with mitral valve prolapse syndrome. J Heart Valve Dis. 1995;4:236–241

4. Levine RA, Stathogiannis E, Newell JB, Harrigan P, Weyman AE. Reconsideration of echocardiographic standards for mitral valve prolapse: lack of association between leaflet displacement isolated to the apical four chamber view and independent echocardiographic evidence of abnormality. J Am Coll Cardiol. 1988;11:1010–1019.
5. Waller BF, Morrow AG, Maron BJ, Del Negro AA, Kent KM, McGrath FJ, Wallace RB, McIntosh CL, Roberts WC. Etiology of clinically isolated, severe, chronic, pure mitral regurgitation: analysis of 97 patients over 30 years of age having mitral valve replacement. Am Heart J. 1982;104:276–288.