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REVIEW ARTICLE **OPEN ACCESS**

Mitral Valve Prolapse and Its Importance

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ABSTRACT

We have come to the conclusion that "MVP is a common cardiac issue that affects between 2% and 3% of the general population". Furthermore, studies revealed that the "majority of patients have a benign clinical course, and the majority of patients have MVP. There is evidence to indicate that a subset of patients with MVP have an elevated risk of sudden cardiac death (SCD)". Although there are a number of variables that have been related to AMVP, a definite causal relationship between MVP and SCD has not yet been totally shown. Thus, in our review, we have discussed and reviewed MVP in terms of etiology, epidemiological studies, classification, pathology, treatment, prognosis, and complications.
Key words: *MVP, etiology, epidemiological studies, classification, pathology, treatment, prognosis, complications.*

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INTRODUCTION

Researchers have also found that "mitral valve prolapse (MVP) is a disorder of valvular heart disease that may also be referred to as floppy MVS, systolic click-murmur syndrome, and billowing M leaflets". "This condition in no way poses a threat to one's health. In very rare cases, it may show up as a stroke, endocarditis, or sudden cardiac death". [1] [2] [3] Studies have shown that "it is estimated that around three percent of the population in the United States is afflicted with this condition. Studies have also concluded that people with this disorder have symptoms when an extra leaflet of the MV that is abnormally thickens and prolapses into the left atrium during the systole phase of the heart's beating cycle". "The diagnosis of MVP is made using cardiac auscultation as a component of the examination procedure in clinical practice. The test that serves to validate the diagnosis is called echocardiography. Furthermore,studies have concluded that the most prevalent cause of MR in the US that isn't due to an ischemia condition in the mitral valve is this disorder".[4] Patients who are having symptoms may need repair. Thus, in our review we have discussed and reviewed about MVP.

ETIOLOGY

Researchers have also found that "connective tissue diseases such as Pseudoxanthoma elasticum(PE), Marfan syndrome (MS), Loeys-Dietz syndrome(LDS), Ehlers-Danlos syndrome(EDS), osteogenesis imperfecta(OI), and aneurysms-osteoarthritis syndrome(AOS) are examples of conditions in which MVP often occurs as an isolated condition".[5]

EPIDEMIOLGICAL STUDIES

Additionally, researchers have discovered that MVP affects about 3% of the population, making it a common valvular disorder. Studies have also found that this disorder affects over 16 million people worldwide, with 7-8 million of them living in the United States. In addition to this, studies have also concluded that there are two different types of MVP: primary and secondary. Primary mitral valve prolapse is characterized by myxomatous D in the absence of any CT disease. Furthermore, studies have also concluded that conditions such as multiple sclerosis, polycystic kidney disease, Graves' disease, pectus excavatum, and Ehlers-Danlos syndrome are all linked to secondary MVP. Studies have shown that "several complications can occur as a result of MVP, such as severe MVR (4%), bacterial endocarditis(BEC), congestive(C) HF , and even sudden D ". [6] Therefore, studies have also concluded that "autosomal dominant genetic changes affecting chromosomes, including MMVP1 (Chromosome 16p11.2-p12), MMVP2 (Chromosome 11p15), and MMVP3 (Chromosome 13q31.3-q32), may be the cause". "Studies have further concluded that "there are certain mutations on chromosome Xq28, like P637Q, G288R, and V711D, or in-frame deletions of 1944 base pairs, that may have an impact, although they are not very common". [7 ,4]

CLASSIFICATION

Additionally, researchers have discovered that the Most Valuable Player award may be broken down into two categories: primary, which is also known as the nonsyndromic MVP at times, and secondary, which is also known as the syndromic MVP. Studies have also concluded that "this second case shows MVP that is caused by a group of CT diseases, including MFS, LDS, EDS,OI,PE and recently discovered AOAS. PE,OI, and other CT diseases are other examples of these conditions".[8,9,10,11,12,13] Studies have also concluded that "MVP has also been found in hypertrophic cardiomyopathy (HCM), which suggests that it may play a part in the pathophysiology of blockage that is typical of this myopathy".[14] Studies have also concluded that "this discovery was made possible by the fact that HCM also contains MVP. The heart muscle becomes thicker as a result of this myopathy, which may be identified by its symptoms".[14]

1. NONSYNDROMIC MVP

In a study, researchers concluded that " the occurrence of MVP and its clinical associations within the community using updated ECG criteria.[15] In addition to this, studies have concluded that a total of 3491 subjects underwent regular 2D echocardiograms, which were both accessible and sufficient for the assessment of the MV. This sample size was used for the evaluation. The estimated overall prevalence was 2.4%, with 47 individuals (1.3%) diagnosed with classic MVP and 37 individuals (1.1%) diagnosed with nonclassic MVP. MVP was found to be evenly distributed across different age groups, spanning from 30 to 80 years old. The MVP award was evenly distributed between men and women in terms of gender. These findings differed from previous research that relied on "M-mode diagnostic criteria (MMDC) wehich revealed that MVP preferentially affects women & older individuals".[16,17] In a study, researchers found that "MVP prevalence was found to be low among children and young people".[18,19] Despite the fact that studies told that "the genetic tendency to develop MVP may be present from birth".[18, 20] According to these findings, MVP is a progressive disease that mostly affects individuals between the ages of 40 and 50.[15] When compared to participants who did not have MVP in FHS, those who did have MVP had a lower body fat percentage.[15] A number of studies have come to the conclusion that the "fact that the FHS sample is mostly white is a significant drawback".[21, 22]

In a study, researchers found that although "these more recent studies are based on better ECG criteria, the prevalence of MVP in blacks was examined using older MMDC and NS-2D-ECG pictures. This is despite the fact that these "more recent studies are based on better ECG criteria".[23] There have been no previous studies that have looked at the "prevalence of MVP in Hispanic populations, as the findings of a comprehensive assessment of the relevant published literature have shown. Patients who have primary or non-syndromic MVP have a tricuspid valve prolapse between forty and fifty percent of the time, although isolated tricuspid prolapse has been found relatively seldom.[24]

2. SYNDROMIC MVP : MFS & OTHER CONNECTIVE TISSUE DISORDERS

In a study, researchers found that "it has been estimated that the prevalence of MV disease in MFS ranges anywhere from 75% to 80%".[25] Furthermore , studies have shown that "the prevalence of MVP in patients with EDS seems to be substantially lower when using traditional ECG criteria (6%)".[8] It would seem that patients who have Loeys-Dietz syndrome have a lower prevalence of MV disease (compared to patients who have MFS).[26] In one study reseachers compared "the prevalence of MVP in 71 people with TGF-β1-R2 mutations, which are typical of LDS, and 243 people with fibrillin-1 (FBN1) mutations, which are typical of MFS, as well as in 50 family members who did not have the condition.They discovered that mutations in FBN1 had a much higher prevalence for both MVP and MR than the group that had mutations in TGF-β1 R2 (45% and 56%, respectively, vs. 21% and 35%, respectively)".[26] Studies have also conclude that "individuals with the AOS were often affected by MV anomalies, and the degree of these abnormalities ranged from mild to severe. The number of people who had MVP was ten out of twenty-two (45%), while the number of people who had MR was six out of twenty-two (27%)".[13] In a study, researchers found that "although the presence of MVP has been shown in OI and PE, yet the true prevalence of the disease is unclear owing to the fact that MMDC were not utilized in the early imaging studies performed on these patients".[9,27] As a result, according to many studies , it is still unclear how prevalent is the disease".[9, 27]

3. HYPERTROPHIC CARDIOMYOPATHY (HCM)

In a study, researchers found that the largest study yet conducted to investigate the "prevalence of MVP in HCM reported that it was present in 3% of the 528 patients who were diagnosed with HCM. This study also suggest that 2 different disorders that, may coexist with one another".[28] On the other hand, studies have also shown that "higher prevalence of other MV abnormalities, such as leaflet elongation and increased thickness, was feature of HCM. According to the findings of one study, the prevalence of this

condition was determined to be 66%".[29] This suggested according to sudies that "MV abnormalities are essential for the development of HCM, either as a primary trait or as a secondary adaptive response to shear stress in a turbulent outflow tract or to paracrine actions happening in the surrounding hypertrophic ventricle".[30]

PATHOLOGY

Studies have also found that "myxomatous degeneration can cause problems with the end leaflets, weakening and stretching of the chordae tendineae, leading annular dilatation or thicker leaflet tissue, chordae that are longer, and Segmental MVP. Other pathologic abnormalities include fibroelastic deficiency, which is defined by thin, transparent, and smooth leaflets; a deficiency in elastin, proteoglycan, and collagen; and a deficiency in connective tissue. Fibroelastic deficiency is characterized by thin, transparent, and smooth leaflets".[31,4] Studies have also concluded that "complications such as IEC and thrombosis may result from a disruption in the endothelium. The majority of individuals who have been diagnosed with MVP only have moderate structural abnormalities of the MV, which are considered to be clinically irrelevant".[32] Thus, studies also concluded that "the leaflets of the mitral valve often have a significant redundancy, which hinders the correct coaptation of the leaflets during systole, leading to mitral insufficiency".[32] The patient will, at some point, suffer mitral yearly dilatation, which will ultimately result in further development of the mitral insufficiency over the course of time. To our delight, the great majority of patients have just modest abnormalities in their leaflets and do not present any symptoms.[33]

TREATMENT

Several studies have also found that patients who have MVP but have no symptoms often do not need to be treated for the condition.[34,35,36] Studies have also concluded that "patients with MVP who display symptoms of dysautonomia, including chest tightness and palpitations, should be treated with beta-blockers like propranolol, which are medications in the category of antiarrhythmic drugs". Studies have also concluded that MVP repair or replacement may be helpful for MVP patients who have severe mitral regurgitation. The ACC and AHA recommend mitral valve repair at the end of the mm before symptoms of congestive heart failure occur. In addition to above studies, researches have also concluded that "people who have MVP have a higher risk of acquiring bacterial endocarditis than those who do not have MVP. Before 2007, the American Heart Association (AHA) recommended that patients get antibiotics before invasive procedures, including dental surgery, in order to reduce the risk of infection. According to more recent AHA guidelines, only patients with other cardiac conditions who are most at risk of suffering severe consequences from infective endocarditis should receive prophylaxis for dental procedures".[33]
Several studies have also found that the "link between having a major vascular event (MVP) and having a cerebrovascular incident (CVI) is a tenuous one. The American Heart Association and the American College of Cardiology (AHA/ACC) in 2014 and the European Society of Cardiology in 2012 did not provide any opinion on the use of antiplatelet or antithrombotic therapy in MVP. Studies have also concluded that both of these organizations published their findings in the same year".[33] The ACC/AHA recommendations from 2006 recommend endorsing using aspirin for patients with unexplained transient ischemic episodes who are in sinus rhythm and do not show atrial thrombi. Additionally, studies have also concluded that "aspirin may be a therapy option to consider in patients with sinus rhythm who have echocardiographic evidence of high-risk MVP".[33] Studies have also concluded that even "when aspirin therapy is being delivered, it is recommended that anticoagulation be performed in instances of systemic embolism or recurrent transient ischemic episodes (TIA)".[33] Studies have also concluded that "in the absence of systemic embolism, an unexplained transient ischemic attack (TIA), an ischemic stroke, or atrial fibrillation, anticoagulation is not recommended as a course of treatment. In addition to this, studies have also concluded that in patients who are not experiencing any symptoms, a mitral valve prolapse may be managed with the conservative approach of observation and monitoring. Furthermore, studies have concluded that patients who do not have contemporaneous mitral regurgitation may only need to be monitored once every three to five years, but patients who do have mitral regurgitation should be monitored once every year".[33] It is very important to reassure the patient, as well as to provide them with advice for leading a healthy lifestyle and maintaining constant physical exercise. Studies have also concluded that if the patient is experiencing symptoms like heart palpitations, anxiety, or chest tightness, it is essential to rule out any other probable reasons first. Studies have also concluded that repairing the mitral valve is preferable to replacing it.[33] Transcatheter repair of the mitral valve may be an option for those who are experiencing symptoms but who are not good surgical candidates due to having too many other health conditions. Studies have also concluded that "those athletes who are experiencing mitral valve prolapse may get help from the AHA. Athletes who participate in high-intensity competitive sports

may be selected based on their clinical H/O". Studies have also concluded that "they can participate if they do not have a prior history of syncope, sustained or repetitive and non-sustained supraventricular tachycardia, SMR, a prior embolic event, LVSD with an ejection fraction < 50, or a family h/o MVP-related sudden cardiac death. Athletes may participate in low-intensity competitive sports owing to the aforementioned criteria".[33]

PROGNOSIS [33]

Studies have also concluded that, due to the lack of noticeable symptoms, the vast majority of spatients with MVP go undiagnosed and do not require therapy. Mitral valve prolapse (MVP), mitral regurgitation (MVR), atrial fibrillation (AFib), transient ischemic attack (TIA), and systemic embolism (SEM) are all consequences of MVP. The severity of mitral valve regurgitation and ejection fraction is the best predictor of death in MVP.[33]

COMPLICATION [33]

1. "Progression to SMR
2. IEC
3. AF
4. Stroke
5. Sudden Death"

CONCLUSION

We have come to the conclusion that the management of MVP should involve an interprofessional team consisting of a cardiologist, cardiac nurse, primary care provider, and cardiac surgeon. Furthermore, it is also important to inform patients that they have a harmless condition and that the likelihood of complications is minimal. Moreover, tell them about the symptoms of MR and endocarditis and advise them on when to seek medical attention and the benefits of adopting a healthy lifestyle, avoiding smoking, engaging in regular exercise, and refraining from alcohol and caffeinated beverages. Thus,follow-up is essential because certain patients may experience a progression of mitral insufficiency and encounter symptoms that are difficult to tolerate.

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