Study of Mitral Valve Insufficiency: A Systemic Review

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ABSTRACT:
The Rationale Behind the Evaluation Mitral valvular disease, which includes mitral stenosis and mitral regurgitation, is the focus of this review. The structure of the valve is our starting point for this discussion. According to recent studies, 3D imaging has made it possible to better understand valve anatomy. Percutaneous balloon mitral valvuloplasty is still the best treatment for mitral stenosis when necessary and possible based on the patient's anatomy. The most common cause of mitral stenosis is atherosclerosis. Changes in the left ventricle's shape can lead to mitral regurgitation (MR), which can be caused by a problem in the mitral apparatus or the aortic valve. Researchers are exploring percutaneous methods of mitral valve repair and replacement, but surgery is still the gold standard for treating severe primary MR. The disease of the mitral valve is a common medical issue. If you want to provide the best care for a patient, you need to have a thorough understanding of the mitral valve and the diseases that can damage it.

Keywords: Mitral valve disease, Mitral valve anatomy, Mitral stenosis, Mitral regurgitation

INTRODUCTION:
As previously discussed (Zoghbi et al. 2003)(1), the mitral valve (MV) apparatus includes an annulus, two leaflets, three types of chordae tendineae, and two dominant papillary muscles. There are multiple parts to this structure, including an annulus, two leaflets, and two powerful papillary muscles (Rodriguez et al. 1993)(2).
The mitral valve annulus (MV annulus) is joined to the aortic valve by the fibrous aortic-mitral curtain. Geometrically speaking, it has a larger diameter between the joints than it does along the middle, making it a non-planar structure in the shape of a saddle.
There are two sets of leaflets. The base leaflet is shorter and wider than the one closest to the stem, which is long and narrow. The flyers meet at two junctures that are referred to as commissures. Both the anterior and posterior medial axes intersect at these two points. Three scallops numbered 1 through 3, run from the edge to the center of each leaflet, which is designated as a "A" on the front and a "P" on the back (Vahanian et al. 2013)(3).
The papillary muscles are attached to the MV leaflets via chordata tendineae. They consist of various fibers. Depending on where they attach, chordae are classified as primary, secondary, (Mentias et al. 2018)(4), or tertiary. Only the edges of the leaflets support primary chordae, the rough surfaces of the leaflets support secondary chordae, and the base of the back leaflet supports tertiary chordae.

Four, the anterolateral and posteromedial sides of the face are home to the two most significant papillary muscles. Blood for the posteromedial papillary muscle typically comes only from the posterior descending artery. The anterolateral papillary

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muscle, on the other hand, receives blood supply from the left anterior descending artery and the left circumflex artery. When there is a problem with the MV system at any level or multiple levels, the MVs will become dysfunctional. MV stenosis or MV regurgitation, or both, are common culprits in this condition.

Comprehensive Review:
The Causes of Mitral Valve Stenosis: Anatomy
Rheumatic heart disease (RHD) is responsible for approximately one-third of all cases of mitral valve stenosis (MS) worldwide. The problem occurs more frequently and worsens more rapidly in developing nations than in developed nations. According to research by (Muresian 2009)(5), this condition is brought on by an overactive immune response to a streptococcal antigen present in valve tissue. Inflammation of the heart (carditis) caused by repeated episodes of acute rheumatic fever greatly increases the likelihood of RHD. While damage to the mitral valve is most common, damage can occur to any of the heart's valves. People with rheumatism often experience the following changes:

The cracks in the system began to heal: The leaflet becomes thicker, especially at the edges, and the chordae become shorter and joined together to form the "fish-mouth" shape of the MVortex. These two alterations to the leaflet's structure cause it to resemble a hockey stick in echocardiography images.

Other possible triggers for MS include: This condition is known as mitral annular calcification (MAC). Although MAC is more common in the elderly and those with advanced kidney disease, it is only rarely linked to clinically relevant MS. Radiation valvulitis, as studied by (Luxereau et al. 1991)(6), typically manifests 10–20 years after the completion of mediastinal radiation therapy. (Gorlin and Gorlin 1951)(7) list several extremely rare congenital causes, including cor triatriatum, a parachute mitral valve, a double-orifice valve, and a supra valvular mitral ring. Systemic inflammatory diseases, such as lupus erythematosus and rheumatoid arthritis, can lead to valvulitis. Multiple sclerosis (MS) is another possible cause. Blood flow obstructions, such as a large atrial myxoma or infected plants, have been linked to functional MS.

The Pathophysiology and Consequences of the Disease
An MV is usually between 4 and 6 cm2 in size. It has been shown (Schlosshan et al. 2011)(8) that a diastolic pressure gradient develops between the left atrium (LA) and the left ventricle (LV) when the affected area of the myocardium shrinks to less than 2 cm2. Some of the main things that make it hard for people with cardiac amyloidosis to work out are an abnormal coronary flow velocity reserve and a lower myocardial contractile reserve (2018). Because of this, the forward flow drops (B), and the pressures in the LA rise (A).

Multiple sclerosis is made worse by tachycardia, which shortens the time it takes for the diastolic chamber to fill and raises the transmural gradient. Some of the effects of more people in the LA area are: People with bigger left atriums are more likely to get atrial arrhythmias like atrial fibrillation and systemic thromboembolism. Pulmonary edema and pulmonary hypertension happen when pulmonary pressures are too high. The former can lead to the latter, which can show up as tricuspid regurgitation or failure of the right ventricle.

Reduced forward flow can cause low cardiac output because the LV isn't getting enough blood.

Mitral stenosis is measured in terms of its severity.
In this case, the best method is echocardiography. For a quantitative assessment of MS, the ASE/EAE joint guidelines (Ruckman and Van Praagh 1978)(9) say to look at the MV area, the mean diastolic transmitral pressure gradient, and secondary changes like the size of the left atrium and right side chambers and the pressure on the right side.

The mean diastolic pressure gradient, or MDP gradient, is Most of the time, this is measured with an apical four-chamber view continuous-wave Doppler through the MV. This value is found by using
Doppler data to track the diastolic transmitral flow and the simplified Bernoulli equation to turn speeds into pressures. According to the research, a mean gradient of more than 10 mmHg is a sign of severe MS, 5-10 mmHg is a sign of moderate MS, and 5 mmHg is a sign of mild MS. Remember that the heart rate and the forward flow of blood have a big impact on the transmitral gradient. Because of this, the 2014 AHA/ACC Guideline for the management of patients with valvular heart disease and the 2017 AHA/ACC Guideline update (Nishimura et al., 2014)(10) did not include it as a way to measure how bad multiple sclerosis was.

Diastolic MVarea can be measured right at the leaflet tips using either two-dimensional (2D) planimetry (Anwar et al. 2007)(11) or, ideally, three-dimensional (3D) planimetry(De Backer et al. 2014). Estimating MVarea (PISA) can be done directly or indirectly using tools like the pressure half time (PHT), the deceleration time (DT), the continuity equation, and the proximal iso velocity surface area. Even though it is not usually recommended for most patients, the mitral valve gradient can also be measured invasively in the cardiac catheterization lab, and the mitral valve area can be calculated using the Gorlin equation(Goldstein et al. 2016)(12).

The Function of Stress Testing
When there is a discrepancy between how severe MS symptoms are reported to be and how severe they are, stress testing, most commonly performed through exercise echocardiography, can be extremely beneficial. A person with severe MS may not show any symptoms, but an exercise test can confirm the diagnosis.

The Causes and Effects of Mitral Valve Regurgitation: Etiology
Primary and secondary mitral valve regurgitation (MR) are the two main categories of causes. These categories do not rule out one another. The primary causes are Mitral valve issues including those involving the leaflets, chordae tendineae, papillary muscles, or annulus. In developed countries, prolapse of the MV is the most common cause of MR requiring surgery, according to research by(Baird 2011)(13). Two distinct degenerative changes have been identified pathologically, but they share a lot of similarities. Myxomatous degeneration and an abnormal buildup of mucopolysaccharides cause Barlow disease; fibroelastic deficiency results from structural abnormalities in connective tissue and causes a loss of mechanical integrity(Alashi et al. 2016)(14). More than 2 millimeters of abnormal leaflet motion during systole, as seen in a long-axis view of the heart, is diagnostic of mitral valve prolapse. Primary MR can be brought on by a wide variety of factors, including infective endocarditis, mitral annular calcification (MAC), rheumatic heart disease, connective tissue disorders, congenital

Management
When the reported severity of MS symptoms differs from the actual severity, stress testing, typically performed through exercise echocardiography, can be extremely helpful. The diagnosis of MS can be made through an exercise test even if the patient shows no symptoms. has the strength to keep going without tiring People with moderate MS and severe symptoms can have hemodynamically significant MS detected by a stress test. This is consistent with findings in individuals exhibiting severe MS symptoms. Measuring MV gradients and approximating right-side pressures is standard practice during a stress test. When deciding whether or not to proceed with an intervention, these findings should be considered.
anomalies (like a cleft palate), and even certain medications.

Subsequent Reasons: These arise from shifts in left ventricular geometry that make it challenging for the MV apparatus to function normally. Tethering of mitral valve leaflets due to changes in the shape of the MV and its papillary muscles has been proposed as a cause, along with poor LV remodeling and dyssynchrony, mitral annular dilatation, and poor LV contractility with loss of the forces needed for MV closure during systole. Second, you can distinguish between ischemic and non-ischemic myocardial infarction (MR and MI, respectively).

Left ventricular dysfunction due to coronary artery disease is the cause of ischaemic MR. What this means is that the person's prognosis is poor, and they may develop heart failure.

All forms of non-ischemic cardiomyopathy, including dilated cardiomyopathy, restrictive cardiomyopathy, and hypertrophic cardiomyopathy, exhibit non-ischemic magnetic resonance imaging (MRI). Annular dilation from atrial fibrillation and dyssynchrony from right ventricular pacing are two additional causes of non-ischemic MR.

The Pathophysiology and Consequences of the Disease

Based on the motion of the leaflets, Carpentier divides mitral regurgitation into two types: functional and non-functional. This classification aids in surgical planning (Mentias, Naji, et al. 2016)(15): Movement of the leaflets is normal (annular dilatation and perforation of the leaflets) in Type I. Excessive flailing, prolapse, or rupture of the papillary muscles characterize Type II. Type IIIa: Systolic and diastolic leaflet movement is restricted (thickening and retraction), as seen in rheumatic disease. In ischemic and functional disease, the leaflets are tethered to the heart and can only move in response to cardiac contractions.

Too much blood collects in the left atrium and left ventricle when mitral regurgitation occurs. The severity of this condition is proportional to the duration of the regurgitation (acute vs. chronic) and the amount of blood loss (acute vs. chronic). Preload increases dramatically and rapidly during an acute MR. Since the ventricle hasn't had time to adapt to the new volume, LV filling pressures have increased significantly. This increase is relayed to the pulmonary circulation and leads to a greater volume of blood passing through the pulmonary capillaries. The majority of the blood flow is now entering the left atrium, which significantly reduces forward output. This may lead to cardiogenic shock. After a heart attack (myocardial infarction), this can occur if the papillary muscle or a chord is torn.

There are three distinct phases in the natural history of chronic MR: the asymptomatic early compensated stage, the intermediate stage when the LV begins to change, and the advanced decompensated stage. This timeline may be misleading.

This can be achieved by detecting potentially harmful changes in the LV before they cause irreparable damage and allowing doctors to recommend treatment.

Left atrial enlargement, brought on by chronic MR, has been linked to left atrial arrhythmia.

Mitril regurgitation is quantified in this study

Echocardiography is the primary imaging method used in magnetic resonance imaging (MRI). According to the ASE guidelines developed by (Perloff and Roberts 1972)(16), a thorough evaluation takes into account all relevant parameters. You shouldn't rely solely on color Doppler. They can be divided into two categories: those that use color Doppler, those that use pulsed wave (PW) Doppler, those that use continuous-wave (CW), Doppler, those that use PISA, volumetric methods, and those that use other supportive echo findings (Hugenholtz et al. 1962)(17).

Color Doppler Ultrasound: A color Doppler is an instrument that detects the presence of various hues. Color Doppler is utilized for both vena contracta and jet area measurements. It is also employed in calculating the jet area/LA area ratio. Signs of severe MR include a jet that occupies more than 40% of the LA area and a vena contracta wider than 7 mm.
Several technical, hemodynamic, and anatomical factors were found to influence the jet region (Eagle et al. 2011)(18). Therefore, this metric should not be used as the sole basis for assessing severity.

Second, PW Doppler: The ins and outs of blood flow through the mitral valve and pulmonary veins. PW Doppler can improve a project's odds of success. When MR is severe, the peak inflow velocity of the mitral valve typically exceeds 120 cm/s. During both the systole and diastole phases of a normal heartbeat, pulmonary venous blood flows forward. During systole, (Mentias, Patel, et al. 2016)(19) the atria experience a brief period of retrograde flow. The systolic component of the heartbeat weakens as cardiac muscle relaxation (MR) worsens. Extreme cases of MR are characterized by a reversal of systolic flow. Because of the CW Doppler effect, the CW jet profile can be used to assess MR scans. A dense jet is required to sustain high MR. The number of RBCs in circulation determines the jet's density. Triangular early peaking jets are common in acute MR. This is due to the rapid equalization of pressures in the left ventricular and left atrial chambers.

The Project for International Student Assessment (PISA) rubric We have established that when blood rushes through a hole, it divides into two distinct halves.

Management

(1) Mild TBI: (a) Surgery should be performed immediately because medical treatment is ineffective for acute MR. Long-term outcomes for people with chronic MR are not improved by any medication.

Mitral valve repair is preferable to replacement when possible due to reduced surgical risks and improved outcomes(Chen et al. 1991)(20).encapsulates the features of a fix that increase its chances of success. Reasons for surgery and the strength of those reasons are outlined in guidelines from the American College of Cardiology/American Heart Association (ACC/AHA) and the European Society of Cardiology (ESC). Transcatheter therapies include, among others: Mitral valve repair via transcatheter implantation: Several devices are currently undergoing (Wilkins et al. 1988)clinical trials, but only the MitraClip system from Abbott Laboratories in Illinois has received FDA approval. This gadget is designed to mend folded leaflets completely. The Alfieri repair is a surgical procedure that involves closing off the regurgitant orifices in the mitral valve leaflets. The mitral valve now has two openings or orifices. Long-term sufferers of New York Heart Association class III-IV symptoms may benefit from MR imaging. This treatment is rated as an IIB in the most recent AHA/ACC valve guideline update, despite being the best medical treatment for heart failure according to the guidelines, the patient has a good chance of living a long life, and mitral valve surgery is considered very dangerous. Similar to mitral valve repair, many technologies are currently being developed for transcatheter replacement of the mitral valve(Acker, Gelifns, and Kron 2014)(21), but the FDA has not yet approved any of them.

Two more MRs:

When necessary, cardiac resynchronization therapy should be considered in addition to standard medical treatment for the underlying left ventricular dysfunction (Class I).

In most cases, the severity of MR improves after electrophysiological therapy because LV function improves. Catheter-based procedures and surgical procedures: Because secondary MR is a ventricle disease and not a valve disease, surgical repair or transcatheter therapy to reduce (Ho 2002)(22)MR severity is not effective in patients with secondary MR. Because of this, valve surgery is extremely uncommon and is only recommended for patients with very severe secondary MR who have not improved with medication (Class IIB recommendation). Patients undergoing surgery to improve blood flow for ischaemic MR may require MV surgery if regurgitation is severe (Class IIA recommendation). Results from MV repair and replacement are comparable in this scenario, according to(Carapetis, McDonald, and Wilson
2005)(23); however, replacement is associated with a lower risk of MR recurrence.

**Patients with asymptomatic fibromyalgia have novel prognostic markers, MR was severe, but the ejection fraction was preserved.**

Patients with severe MR and preserved ejection fraction who are asymptomatic are of particular interest because they must undergo surgery(Nishimura et al. 2014) quickly to prevent irreversible damage to the left ventricle. The following markers in these patients are significant for predicting how they will do, according to the results of recent research:

The global longitudinal strain has been linked to an inhibitory neuropeptide. According to multiple studies, the Physical potentiality of Right ventricular systolic pressure (RVSP) was measured by (Carpentier 1983)(24). However, these considerations have not yet been integrated into society's norms and policies.

**Conclusion**

Some structures come together to form the mitral valve apparatus. These include the annulus, leaflets, chordae, and papillary muscles. Many diseases and conditions, both serious and mild, can lead to mitral valve dysfunction. Both the incidence of illness and mortality may be significantly impacted by mitral stenosis and regurgitation. A comprehensive evaluation that takes into account some parameters, including echocardiography, is recommended for determining whether or not a person has heart disease. Knowing the ins and outs of these illnesses is crucial for selecting the most appropriate treatment and for recognizing when treatment is needed. Percutaneous balloon valvuloplasty is the gold standard for treating mitral stenosis in patients who are candidates for the procedure. However, surgery remains the gold standard for mitral regurgitation treatment.

**References**


