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A REVIEW ON MITRAL REGURGITATION: PATHOPHYSIOLOGY, INVESTIGATIONS AND TREATMENT

OMAR ELSAKA [a*], MONEER AYMAN NOURELDEAN [a], MOHAMED ADEL GAMIL [a], MOSTAFA TAREK GHAZALI [a], ASHRAF HAMADA ABD AL-RAZIK [a] AND DALIA HISHAM [a]

[a] Department of Cardiology, Mansoura University, Faculty of Medicine, Mansoura Manchester Medical Program (MMMP), Mansoura, Egypt.

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Review Article

ABSTRACT

Background: The retrograde movement of blood from the left ventricle (LV) into the left atrium (LA) through the mitral valve (MV) causes a systolic murmur heard best at the apex of the heart with radiation to the left axilla. MR is the most common valvular anomaly in the globe, affecting around 2% of the population and increasing in incidence with age. This activity examines the diagnosis and treatment of mitral regurgitation, emphasizing the importance of the healthcare team in assessing and treating patients with this illness.
Conclusion: The goal of this review article is to identify the etiology and epidemiology of mitral regurgitation medical conditions and emergencies, review the proper history, physical, and evaluation of mitral regurgitation, outline the treatment and management options for mitral regurgitation, and describe interprofessional team strategies for improving care coordination and communication to advance mitral regurgitation and improve outcomes.

Keywords: *Echocardiography; heart failure; mitral regurgitation; Mechanism; mitral annulus; papillary muscles.*

1. INTRODUCTION

The most common valvular heart condition, mitral regurgitation (MR), is defined as an abnormal reversal of blood flow from the left ventricle to the left atrium. The mitral valve apparatus, which includes the mitral annulus, the leaflets (a large anterior [aortic] leaflet and a small posterior [mural] leaflet), the chordae tendineae, and the papillary muscles, is disrupted (anteromedial and posterolateral). Mitral valve prolapse (MVP), rheumatic heart disease, infective endocarditis, annular calcification, cardiomyopathy, and ischemic heart disease are the most common causes of MR. The biology, clinical symptoms, and therapy of MR vary according to the disease's chronicity and cause [1].

2. ETIOLOGY

2.1 Primary MR

Degenerative: The most prevalent pathophysiologic basis for degenerative mitral regurgitation is myxomatous mitral valve degeneration, which results in mitral valve prolapse (MVP). MVP can be a

**Corresponding author: Email: omarelsaka0808@gmail.com;*

primary, non-syndromic or secondary, syndromic condition. The advancement of age is the primary cause of disease progression in primary MVP. Secondary MVP causes MR in connective tissue illnesses to include Marfan syndrome, Ehlers-Danlos syndrome, MASS phenotype, systemic lupus erythematosus (SLE), osteogenesis imperfecta, and pseudoxanthoma elasticum. Isolated cleft of the mitral valve, double orifice mitral valve, and parachute mitral valve (PMV), which is a congenital valvular abnormality in which the chordae tendineae are linked to a single papillary muscle, are examples of congenital valvular anomalies, have been connected to the development of MR. These congenital disorders are clearly defined in the literature to induce primary MR, notwithstanding their rarity. Infectious/Rheumatic: Rheumatic heart disease (RHD) is highly widespread in developing countries due to a lack of medical resources and immunizations, with an estimated 15 million cases globally. Chronic RHD is linked to pancarditis and involves the mitral valve, resulting in regurgitation in nearly all instances due to scarring of the valve and valve mechanism [2].

2.2 Secondary MR

Secondary MR is caused by left ventricular dilatation caused by ischemic or nonischemic cardiomyopathy impairing leaflet coaptation of a structurally normal MV. Apical and lateral papillary muscle displacement can result in leaflet tethering, dilatation, and flattening of the mitral annulus, as well as reduced valve closing forces, due to dysfunction and remodeling. Reduced LV contractility, altered systolic annular contraction, diminished synchronicity between the two papillary muscles, and worldwide LV desynchrony, notably in the basal segments, are all examples of reduced closure forces. After a myocardial infarction (MI) or infective endocarditis, papillary muscle rupture is a very rare disease that affects 1% to 2% of patients. Because the papillary muscles are dysfunctional, it causes severe mitral regurgitation [3].

Prior MI coupled with normal mitral valve leaflets and chordae causes ischemic MR (IMR). Remodeling occurs when the segments underneath the papillary muscles are ischemic. This promotes papillary muscle displacement, which causes the leaflets to become more apical, resulting in the "seagull sign." The most prevalent form of IMR is type IIIb, which is caused by limited movements of the leaflet(s) in systole, according to Carpentier's classification. Changes in LV remodeling and valvular deformation, as well as changes in LV and papillary muscle synchrony, are related to the degree of exercise-induced increase or decrease in MR [4].

In a study of 558 individuals with severe congestive heart failure (EF less than or equal to 35%), MR was severe in 4.3 percent, moderate to severe in 12.5 percent, moderate in 21.9 percent, mild to moderate in 11.8 percent, mild in 39.1 percent, and absent or present in 10.4 percent. The link between severe CHF and MR, as well as their relationship, was discovered in this study. Atrial Fibrillation is linked to the following conditions: Atrial fibrillation (AF) causes increased atrial and valve annular size, leading to functional MR, according to a retrospective cohort study. Controlling AF and restoring sinus rhythm resulted in a greater reduction in functional MR in the patients tested. A randomized trial also discovered a relationship between AF and deteriorating valvular disease [5].

MR can potentially be caused by hypertrophic cardiomyopathy (HCM). Severe left ventricular hypertrophy creates increased papillary muscle mass, pushing them closer together, which is what defines HCM. The mitral valve leaflets grow extended and floppy as a result of this occurrence, which drags the leaflets closer to the left ventricular outflow path, generating regurgitant retrograde flow [6].

2.3 Carpentier's Classification

Type1: Regurgitation jet directed centrally, or normal leaflet motion caused by annular dilatation or leaflet perforation. Excessive leaflet motion is caused by papillary muscle rupture, chordal rupture, or superfluous chordae, or Eccentric jet directed away from the affected leaflet (Type 2). Type 3: Leaflet mobility is restricted: IIIa: Both systole and diastole leaflet mobility is restricted. Rheumatic heart disease is the most common cause. Normal papillary muscles, or Jet, can be guided either centrally or eccentrically. IIIb: In systole, leaflet motion is restricted due to papillary muscle dysfunction or left ventricular dilatation, abnormal papillary muscles, or a jet that is directed either centrally or eccentrically (Figs 1, 2) [7].

3. PATHOPHYSIOLOGY

Organic disease (e.g., rheumatic fever, ruptured chordae tendineae, myxomatous degeneration, leaflet perforation) or a functional defect can induce mitral regurgitation (MR) (ie, a normal valve may regurgitate [leak] because of mitral annular dilatation, focal myocardial dysfunction, or both). Although congenital MR is uncommon, it is frequently linked to myxomatous mitral valve disease. It can also be linked to a cleft of the mitral valve, which is common in people with Down syndrome, or an ostium primum atrial septal defect [10].

An increase in preload and a decrease in afterload cause an increase in end-diastolic volume (EDV) and a decrease in end-systolic volume (ESV) in acute mitral regurgitation (ESV). The total stroke volume (TSV) rises to supranormal levels as a result of this. However, because much of the TSV regurgitates as regurgitant stroke volume, the forward stroke volume (FSV) is reduced (RSV). As a result, there is an increase in left atrial pressure (LAP). The Laplace principle indicates that ventricular wall stress is proportional to both ventricular pressure and radius, hence LV wall stress is significantly lowered in the acute phase when both of these parameters are reduced (Fig. 3) [11].

Chronic compensated mitral regurgitation: The left atrium (LA) and ventricle have enough time to widen and accommodate the regurgitant volume in chronic compensated MR. As a result, LA pressure is frequently normal or only slightly increased. TSV and FSV are maintained as a result of left ventricular dilation caused by eccentric hypertrophy. As the radius of the LV cavity expands, wall stress may be normal to slightly elevated, but end-diastolic LV pressure stays normal. The mitral annulus may extend and hinder the mitral valve leaflets from coapting effectively during systole as the LV grows larger, exacerbating the MR and LV dilation [13].

Chronic decompensated mitral regurgitation: Cardiac dysfunction has evolved in the chronic decompensated phase, affecting both TSV and FSV (although ejection fraction still may be normal). This causes an increase in ESV and EDV, which causes an increase in LV and LA pressure, resulting in pulmonary edema and, if untreated, cardiogenic shock [14].

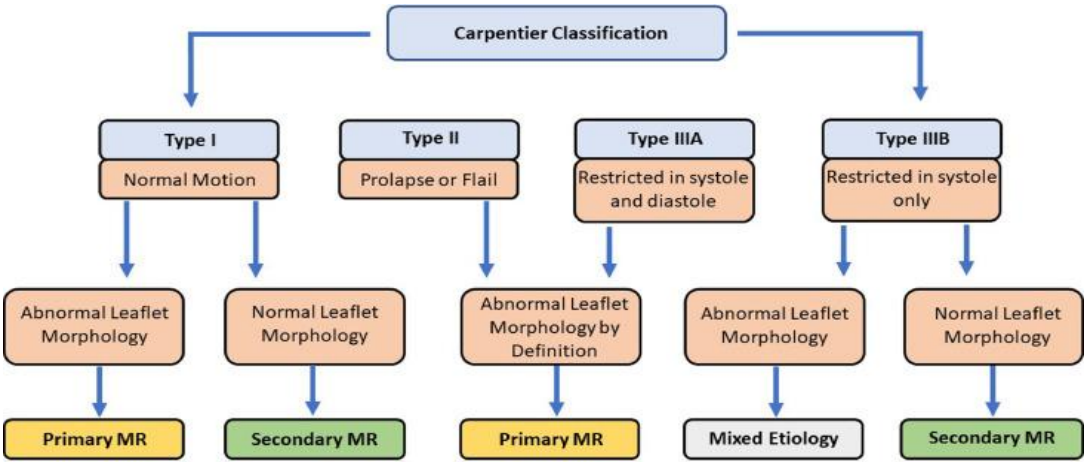

Fig. 1. Carpentier's classification for mitral regurgitation [8]

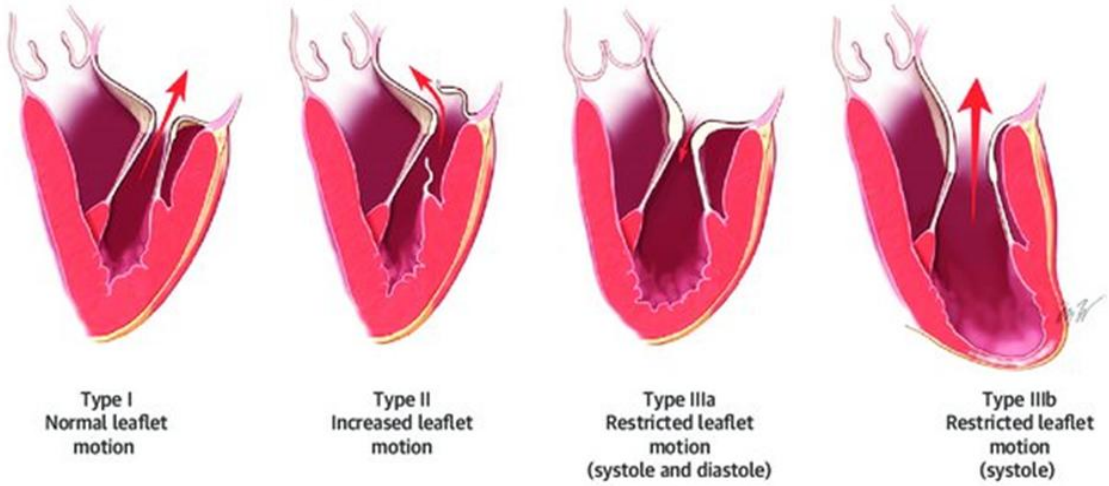

Fig. 2. Images illustrate Carpentier's classification for mitral regurgitation [9]

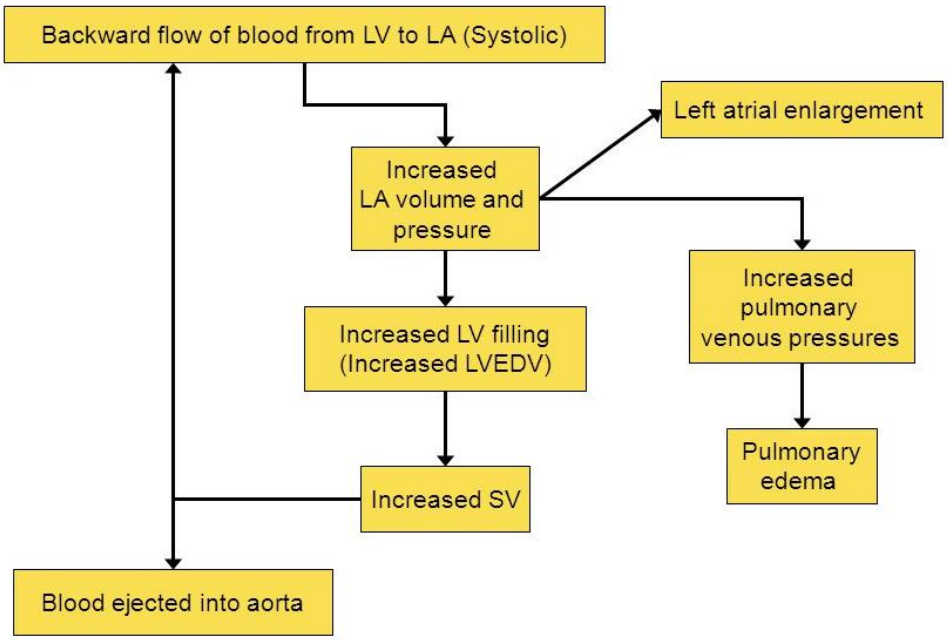

Fig. 3. Pathophysiology of mitral regurgitation [12]

4. HISTORY AND PHYSICAL PRESENTATION

A holosystolic murmur heard best at the cardiac apex with radiation to the left axilla is a sign of mitral valve regurgitation. However, it is critical to distinguish the mitral regurgitation murmur from other systolic murmurs: Early systolic murmur with a mid-systolic click heard best at the cardiac apex. Mitral valve prolapse (MVP) - early systolic murmur with a mid-systolic click heard best at the cardiac apex. TR is a holosystolic regurgitation that is best audible at the lower left sternal border with radiation to the right lower sternal border. When compared to MR, TR increases as you inhale, Holosystolic murmur caused by a ventral septal defect (VSD). The greater the VSD, the quieter the murmur, Aortic stenosis (AS) is mid-systolic stenosis with a crescendo-decrescendo pattern radiating to the neck. With Valsalva or standing, it's quieter. Pulmonic stenosis (PS) is characterized by a crescendo-decrescendo mid-systolic murmur that intensifies during inspiration. The S2 heart sound is widely divided into severe PS. Mid-systolic, S2 heart sound is fixed-split and does not change with inspiration. Atrial septal defect (ASD) - mid-systolic, S2 heart sound is fixed-split and does not change with inspiration. Mid-systolic hypertrophic cardiomyopathy (HCM) radiates and is best heard near the left sternal border, and it's vital to distinguish from MR. With Valsalva and standing, the HCM murmur becomes louder [15].

Clinical findings connected with MR can be divided into two groups: those related to the MR itself and those related to the underlying etiology. Maintaining a broad differential diagnosis is critical, but in general, a focused history and physical examination can determine if the MR is acute or chronic, narrowing the possible etiologies dramatically [16].

5. ACUTE MITRAL REGURGITATION

The clinical examination will reveal symptoms that point to a sudden drop in cardiac output and perhaps cardiogenic shock. The patient will typically experience severe dyspnea at rest, which is aggravated in the supine position, as well as a cough with clear or pink, frothy sputum. They may also mention symptoms like chest pain extending to the neck, jaw, shoulders, or upper extremities, nausea, and diaphoresis, which are all signs of myocardial ischemia. Physical examination may reveal altered mental status, tachycardia (or bradycardia if the conduction system is ischemic), hypotension, tachypnea, hypoxemia, and cyanosis, among other things. A precordial exam may also reveal jugular venous distension, widespread crackles on lung auscultation, and an apical holosystolic murmur with radiation to the axilla. Acute MR is usually caused by papillary muscle rupture due to an acute coronary syndrome or fulminant valvular apparatus destruction due to acute bacterial endocarditis. As a result, more clinical testing should be done to confirm these potentially fatal diseases [17].

There will be signs and symptoms of sepsis, including fevers and chills if you have acute bacterial endocarditis. Patients with a history of intravenous drug misuse are more likely to have comorbid illnesses that predispose them to immunocompromise, such as diabetes, HIV/AIDS, and alcohol use disorder. Depending on the eventual fate of the emboli, there may be several additional clinical symptoms, including focal neurologic impairments if the brain is involved, hematuria or oligoanuria if the kidneys are involved, and Janeway lesions or widespread petechiae if the skin is involved. Acute infections, unlike subacute bacterial endocarditis, often occur in patients with structurally sound heart valves, making rheumatic heart disease and prosthetic valves less likely in this group. Furthermore, because bacterial transport to the mitral valve occurs through the right side of the heart, tricuspid and pulmonic valve diseases are common, and they can easily be detected on physical examination [18].

6. CHRONIC MITRAL REGURGITATION

Patients are frequently asymptomatic until late in the course of the disease. Fatigue, dyspnea on exercise, orthopnea, paroxysmal nocturnal dyspnea, weight gain, widening of pulse pressure, apical holosystolic murmur with radiation to the axilla, dependent edema, misplaced apical impulse, and jugular venous distension are all common clinical signs across all etiologies. Syncope or near syncope, cyanosis, clubbing of digits, graphic anasarca, hepatomegaly, signs of ascites with a fluid wave, or shifting dullness, and evidence of pleural or pericardial effusions may also be present in more severe instances. The development of pulmonary hypertension and subsequent right ventricular systolic dysfunction as a result of chronic pressure overload is reflected in these latter findings. The differential diagnosis is also much broader, and clinical findings are highly dependent on the cause [19].

7. INVESTIGATIONS

7.1 Echocardiogram

The primary and most important diagnostic test for mitral regurgitation is echocardiography (American College of Cardiology/American Heart Association (ACC/AHA) [Class I recommendation]). Both qualitative and quantitative analyses are available with transthoracic echocardiography (TTE) and transesophageal echocardiogram (TEE). The breadth of the regurgitant jet as it exits the regurgitant orifice is known as the vena contracta. It depicts the area of the regurgitant orifice. Severe MR is associated with vena contracta greater than 7 mm. Quantification of MR can also be done using the Doppler volumetric approach. The difference between the mitral and aortic stroke volumes is used to calculate the regurgitant volume in this method. The product of the effective regurgitant orifice (ERO) and the MR velocity-time integral can be used to compute regurgitant volume (VTI). Severe MR is defined as an ERO more than or equal to 0.2 cm2, a regurgitant volume greater than or equal to 30 mL, and a regurgitant fraction of 50% or higher (Fig. 4) [20].

The maximal mid-systolic distance between the leaflet tips and the annular plane of the mitral valve is known as tenting height. At mid-systole, the tenting area is defined as the space between the mitral annular plane and the anterior and posterior leaflets. A tenting height of less than 0.5 cm, a tenting area of 0 cm2, and anterior and posterior mitral leaflet angles of less than 3 degrees are all considered normal. A tenting height of more than or equal to 1 cm, a tenting area of more than 2.5 to 3 cm2, complicated jets, or a posterolateral angle of more than 45 degrees is all signs of poor outcomes after mitral valve replacement. The sphericity index is the ratio of LV end-diastolic volume to the volume of an imaginary sphere whose diameter extends from the annular plane's midpoint to the apex. Unfavorable MV repair outcomes are predicted by end-diastolic diameters more than 65 millimeters (mm), end-systolic diameters greater than 51 millimeters (mm), and systolic sphericity index greater than 0.7 (Tables 1, 2) [22].

Poor prognosis after MV repair surgery is linked to an interpapillary distance of more than 20 mm, a posterior papillary fibrosa distance of more than 40 mm, and lateral wall motion abnormalities. Repeat echocardiogram is recommended for individuals with moderate or greater MR with or without symptoms every 6 to 12 months for severe MR, every 1 to 2 years for moderate MR, and every 3 to 5 years for mild MR after the initial evaluation. Patients with any degree of MR and a change in clinical status or physical examination findings should have a repeat echocardiogram [25].

7.2 Chest Radiography

Although pulmonary congestion (ie, enlarged pulmonary markings) may not be visible until heart failure has been established, evidence of left ventricular (LV) enlargement due to volume overload may be seen (especially in chronic MR). Due to the massive left atrial appendage, left atrial enlargement can also be seen in the anteroposterior (AP) view as a double shadow in the right cardiac silhouette and/or straightening of the left cardiac boundary (Fig. 5) [26].

7.3 Electrocardiogram (ECG)

The following are examples of electrocardiography findings: When papillary muscle rupture causes acute mitral regurgitation (MR), there is ischemia or infarction in the inferior or posterior pathways. Left ventricular (LV) dilatation and hypertrophy are seen in chronic mitral valve regurgitation, as well as increased QRS voltage and ST-T wave alterations in the lateral precordial leads. In chronic mitral valve regurgitation, left atrial enlargement causes a negative P wave in lead V1 and/or a wide notched P wave in leads II, III, or aVF. Atrial fibrillation can be detected in the late stages of the disease [28].

7.4 Exercise Stress Testing

Exercise treadmill testing may provide information on the patient's symptom status and exercise tolerance in patients with severe, asymptomatic primary MR. In symptomatic patients with non-severe MR, exercise echocardiography can be used to assess changes in MR severity and/or pulmonary artery pressure [29].

Table 1. Echocardiographic semi-quantitative used to define severe MR [23]

Semi-quantitative assessment	Definition
Vena contracta width (mm)	≥7 (>8 for biplane)
Upstream pulmonary venous flow	systolic reversal
MV inflow	E-wave velocity ≥1.5 m/s
Regurgitant index	TVI mitral/TVI aortic >1.4

Table 2. Echocardiographic quantitative parameters used to define severe MR [24]

Quantitative assessment	Primary	Secondary
EROA (mm3)	≥40	≥20
Regurgitant volume (mL/beat)	≥60	≥30

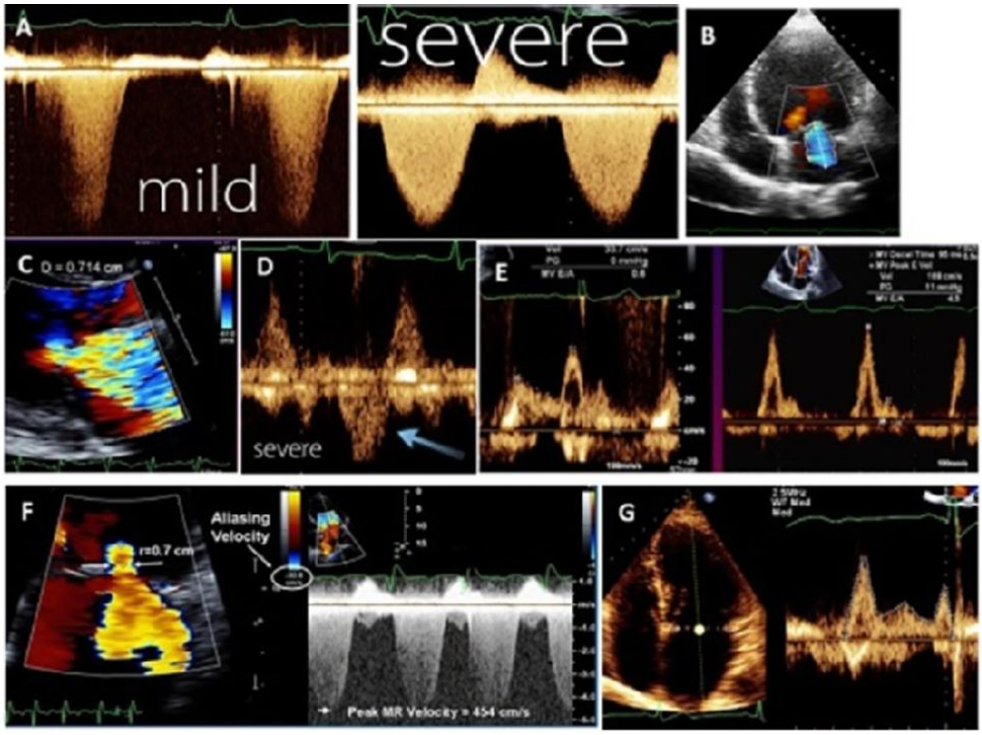

Fig. 4. Methods used for MR quantification. Continuous Doppler tracing showing dense signal with severe MR and faint with mild MR (A), tracing of MR jet area (B), width of vena contracta (C), reversal of pulmonary venous flow (D), dominant E-wave of mitral inflow in severe MR (E), calculation of MR using PISA method (F), and volumetric measurement of stroke volume across the MV (G) [21]

7.5 Cardiac Catheterization

Cardiac catheterization can be utilized to quantify MR volume with excellent accuracy and is useful in the assessment of MR when clinical symptoms do not match noninvasive test results [30].

7.6 Cardiac MRI

Cardiac MRI is a valuable and useful tool for determining the severity of MR. Quantitative data, such as regurgitant volume and regurgitant fraction, can be accurately assessed using cardiac MRI. When MRI is used to diagnose severe MR, it has a greater link with left ventricular remodeling (particularly, a smaller left ventricular end-diastolic volume after MR is removed) than when echocardiography is used. In clinical practice, cardiac MRI can help distinguish between severe and non-severe MR in patients whose echocardiographic examination is inconclusive, especially if surgery is being considered (Fig. 6) [31].

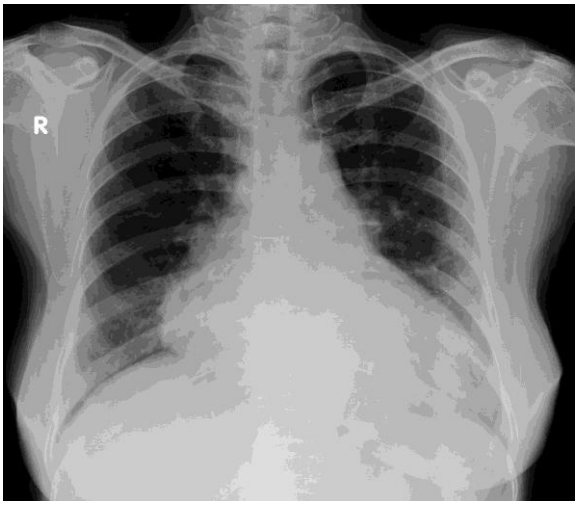

Fig. 5. Chest X-ray revealing cardiomegaly due to mitral regurgitation [27]

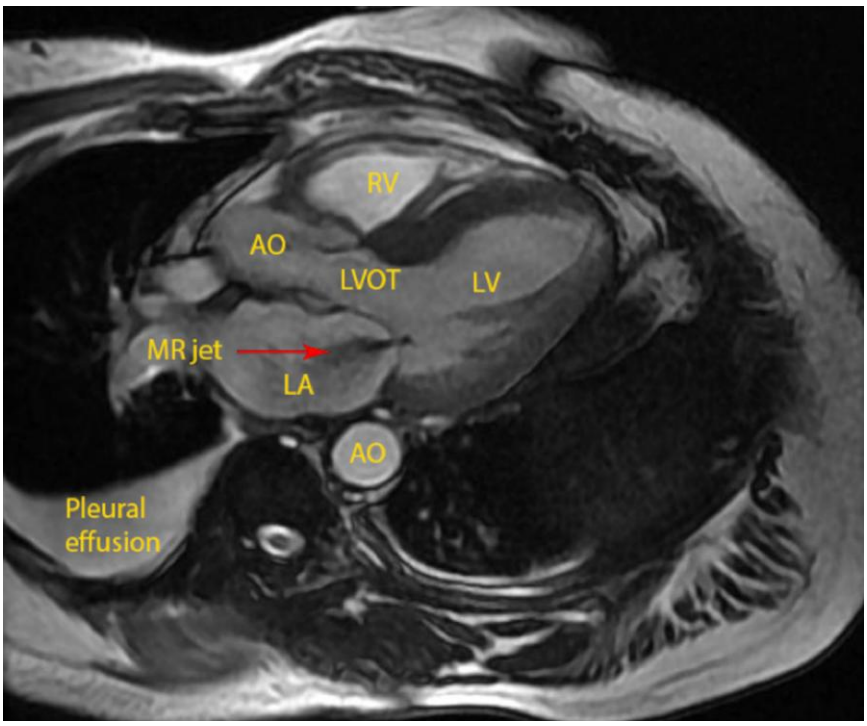

Fig. 6. Role of cardiac MRI in mitral valve regurgitation [32]

7.7 Biomarkers

In individuals with MR, B-type natriuretic peptide (BNP) is generated by ventricular myocytes in response to increased wall stress and correlates with the degree of symptoms as well as providing predictive information. In the absence of symptoms or deleterious hemodynamic effects, a BNP level may be normal in severe, compensated MR. During follow-up of patients with asymptomatic, severe MR, an increased BNP level is linked to the composite endpoint of New York Heart Association (NYHA) class III or IV HF symptoms, LV dysfunction (ejection fraction less than 60%), or mortality [33].

7.8 Treatment

The degree, chronicity, comorbidities, and cause of mitral regurgitation determine whether it should be treated medically or surgically. While some pharmacologic medications are utilized in MR, the evidence for their utility isn't good, and the American College of Cardiology (ACC) and the American Heart Association doesn't endorse them (AHA). Valve surgery is frequently used to treat primary severe MR and ischemic MR [34].

7.9 Medical

In asymptomatic patients, angiotensin-converting enzyme inhibitors (ACEI) and angiotensin II receptor blockers (ARB) have been used to slow the course of MR. In chronic primary MR patients, ACEI/ARBs are thought to reduce regurgitant volume and left-ventricular size. However, there are few studies to back up these claims, and their use in MR is not suggested. Some studies have found no improvement or survival advantage in people with MR who use ACEI/ARBs, and some patients' results have even worsened. Vasodilators have been shown to exacerbate the severity of MR in patients with hypertrophic cardiomyopathy or mitral valve prolapse [35].

Beta-blockers have also been examined in the context of MR treatment. In primary MR, beta-blocking medicines have shown little to no effect; however, in secondary MR, certain trials have shown an enhanced survival benefit with these agents. One study that looked at carvedilol found that it helped to preserve left ventricular function and remodeling while also lowering regurgitant volume. The American College of Cardiology and the American Heart Association have no specific recommendations for the use of beta-blockers in patients with MR [36].

Loop diuretics may be used in conjunction with other pharmacologic treatments to reduce afterload and regurgitant volume in medical management; however, further research is needed to thoroughly establish this association [37].

7.10 Surgical

The choice of whether or not to operate is based on the underlying cause of MR. Valvular injury caused by chordal or papillary muscle rupture, as well as infective endocarditis, necessitates MR surgery. Patients who have MR due to a functional reason, such as ischemia, usually need coronary artery bypass grafting (CABG). Surgical intervention is required in patients with acute, symptomatic MR or an effective regurgitant orifice of at least 40 mm2. Patients with deteriorated LV function or an end-systolic diameter of 4.5 cm may benefit from MR surgery. Patients with primary severe MR require surgery if their ejection fraction is greater than 30% and they are symptomatic or asymptomatic with an EF of 30% to 60%. Mitral valve repair has two aims: have an acceptable surface area of mitral valve leaflet coaptation, 5 to 8 mm being essential, and correct annular dilatation [38].

Because of the decreased recurrence of MR after repair, the American College of Cardiology (ACC) and the American Heart Association (AHA) generally advocate mitral valve repair over replacement. There is additional evidence that surgical repair reduces morbidity and mortality when compared to replacement. When there is substantial tissue loss, as in some cases of infective endocarditis, mitral valve replacement is preferable to repair. Mechanical prostheses are usually preferred over bioprosthetics in terms of replacement because of their enhanced longevity and ease of insertion; nonetheless, both require anticoagulation after implantation. In patients who are at high risk for repair or replacement, Mitraclip is another surgical method that has been demonstrated to be effective and has low morbidity and mortality. Mitraclip can decrease the mitral valve area leading to stenosis, and therefore, an area of under 4.0 cm^2 is a contraindication for this procedure [39].

7.11 Prognosis

There appears to be a link between recurrent mitral regurgitation (MR) after mitral valve (MV) surgery and higher mortality and unfavorable LV remodeling in patients with degenerative MR. MV repair conducted before 2000, prior atrial fibrillation, high LV end-diastolic dimension (LVEDD), prolapse of the isolated anterior leaflet or numerous segments, and lack of ring annuloplasty are all independent risks factors for recurrent MR after mitral valve repair. LVEDD and repair without the implantation of

prosthetic chordae appear to be predictors of MR development. The following are the outcomes for asymptomatic chronic severe degenerative MR: At five years, mortality ranged from 50 to 73 percent. Patients with intact LV function had a mortality rate of 27-45%. Patients with a flail leaflet may die suddenly as often as 1-8 percent of the time [40].

The presence of functional MR was linked to 2-fold higher risk of all-cause death and hospitalization at 1-5 years in a study of individuals with reduced ejection fraction (EF) (independent of ischemic or nonischemic etiology). The following are examples of operational mortality in mitral valve surgery: A 2% mortality rate is associated with isolated mitral valve replacement surgery. Patients under the age of 50 have a 4 percent mortality rate, while those over the age of 80 have a 17 percent mortality rate [41].

Tribouilloy et al discovered that left ventricular end-systolic diameter (LVESD) is independently linked with higher mortality in patients with organic MR due to flail leaflets. Under conservative care, LVESD 40 mm independently predicted overall death (hazard ratio [HR] 1.95; 95 percent confidence interval [CI], 1.01-3.83) and cardiac mortality (HR 3.09; 95 percent confidence interval [CI], 1.35-7.09) in 739 patients. With LVESD >40 mm, the chance of death increased linearly (HR 1.15; 95 percent CI, 1.04-1.27 per 1-mm increment). These data, according to Tribouilloy et al, support urgent surgical rescue in patients with LVESD 40 mm, but also show that operating on patients before their LVESD reaches 40 mm will improve survival. Magne et al found that exercise pulmonary hypertension can be predicted using resting comprehensive echocardiography in asymptomatic patients with degenerative MR [42].

7.12. Epidemiology

Data from the United States: Acute and chronic mitral regurgitation (MR) affects about 5 persons out of every 10,000. Aortic stenosis is the most prevalent valvular lesion, followed by mitral valve disease. Rheumatic heart disease has been supplanted as the primary cause of mitral valvular anomalies by myxomatous degeneration. Prolapse of the mitral valve is estimated to affect 4% of the general population. Mild MR can be found in as many as 20% of middle-aged and older persons using color Doppler echocardiography. Female sex, lower BMI, advanced age, renal dysfunction, prior myocardial infarction, prior mitral stenosis, and prior mitral valve prolapse are all independently linked to MR. It has nothing to do with hyperlipidemia or diabetes. International data: In areas other than the Western world, rheumatic heart disease is the leading cause of MR [43].

7.13 Guidelines

7.13.1 2021 ACC/AHA guidelines on valvular heart disease

In December 2020, the American College of Cardiology (ACC) and the American Heart Association (AHA) modified their recommendations for treating valvular heart disease. The main points are given below. Patients with valvular heart disease (VHD) should be divided into phases (A-D) based on symptoms, valve anatomy, severity of valve failure, and ventricular and pulmonary circulation response. Findings from the history and physical examination (PE) should be compared to those from noninvasive testing when evaluating patients with VHD (ie, electrocardiography [ECG], chest x-ray, transthoracic echocardiography [TTE]). Consider getting more noninvasive (computed tomography [CT], cardiac magnetic resonance imaging [CMRI], stress testing) or invasive (transesophageal echocardiography [TEE], coronary angiography [CAG], cardiac catherization) studies to decide the optimal treatment strategy (Figs 7, 8) [44].

The decision to use oral anticoagulation with either a vitamin K antagonist (VKA) or a non-VKA anticoagulant to prevent thromboembolic events in the setting of VHD and atrial fibrillation (AF) (except for patients with rheumatic mitral stenosis [MS] or a mechanical prosthesis) should be a shared decision-making process based on the CHA2DS2-VASc score (congestive heart failure [CHF], hyper Those with rheumatic MS or a mechanical prosthesis and AF should be given oral anticoagulation with a VKA. All patients with severe VHD who are considering valve surgery should be examined by a multidisciplinary team, either through a referral or in consultation with a primary or comprehensive valve clinic. Symptomatic alleviation and prevention of the irreversible long-term consequences of left ventricular volume overload are the two main reasons for valvular regurgitation intervention. More permanent therapeutic choices and lesser procedural risks have resulted in lower intervention thresholds than previously [47].

Patients with severely symptomatic primary mitral regurgitation (MR) who are at high or prohibitive surgical risk, as well as a select subset of patients with secondary MR who remain severely symptomatic despite guideline-directed management and heart failure therapy, benefit from a mitral transcatheter edge-to-edge repair. Bioprosthetic valve dysfunction can be caused by either valve leaflet degradation or

valve thrombosis. In the absence of active infection, catheter-based therapy for prosthetic valve malfunction seems feasible in selected patients with bioprosthetic leaflet degeneration or paravalvular leak [48].

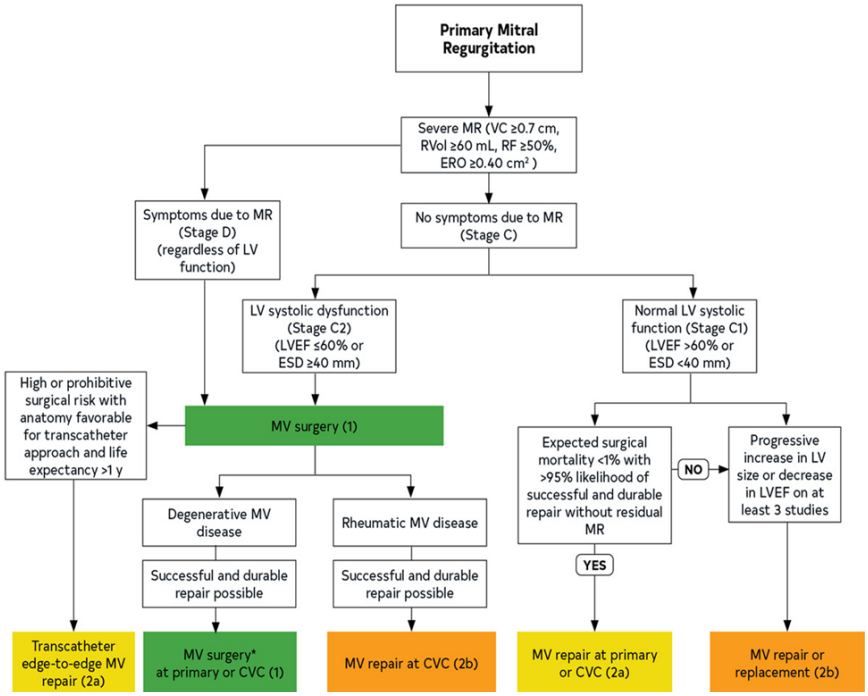

Fig. 7. Primary mitral regurgitation management [45]

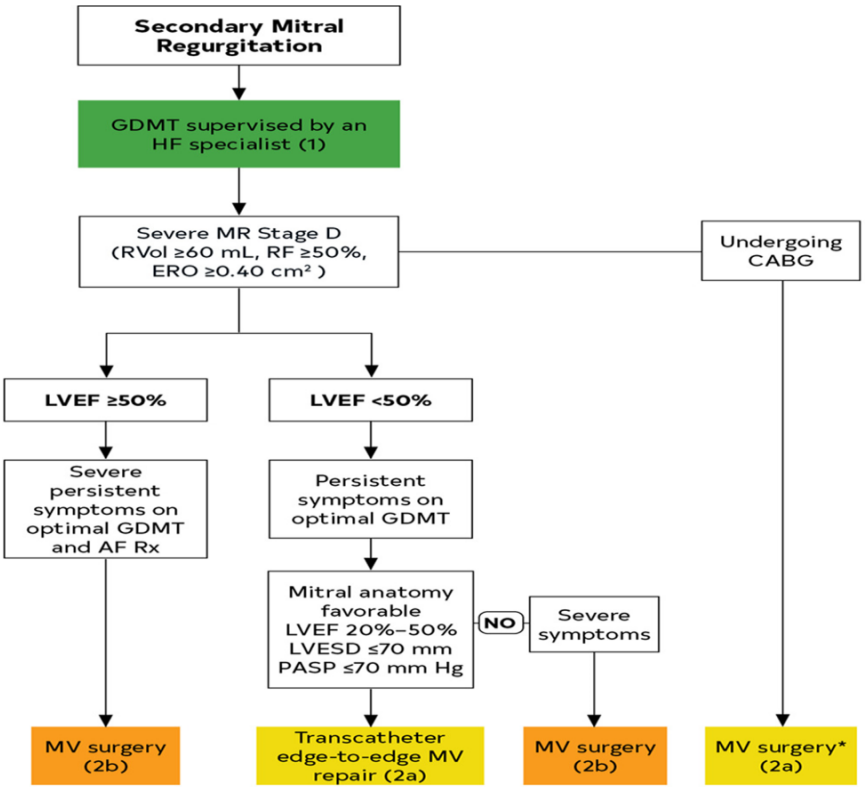

Fig. 8. Secondary mitral regurgitation management [46]

7.13.2 2021 ESC/EACTS management of valvular heart disease (VHD) clinical practice guidelines

The European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS) issued guidelines for the management of patients with valvular heart disease (VHD) in August 2021, which included the following recommendations on mitral valve disease: For symptomatic patients with severe primary mitral regurgitation who are operable and not high-risk, as well as asymptomatic individuals with LV dysfunction, surgery (mitral valve repair) is advised. Valve surgery/intervention is only suggested for patients with severe secondary mitral regurgitation who remain symptomatic despite medical treatment as directed by guidelines (GDMT; including cardiac resynchronization therapy [CRT] if indicated) [49].

8. DISCUSSION

During diastole, blood flows from the left atrium (LA) to the left ventricle (LV), and during systole, the LA is sealed off from the LV. The mitral annulus (MA), mitral leaflets, chordae tendineae, and papillary muscles (PM) are the primary components of the mitral apparatus (Fig. 1). The integrity and normal interplay of these components are required for normal valve function. Mitral regurgitation can be caused by abnormal function of any of the components or their interaction (MR). Understanding the anatomy and physiology of all of the mitral valve's components is critical for accurate diagnosis and planning of repair treatments. The typical anatomy and physiology of the various sections of the mitral valve will be covered first in this study (MA, leaflets, chordae tendineae, and PM). The clinical, anatomic, and physiologic derangements associated with various forms of MR will be discussed in the second half [50].

The most prevalent valvular heart condition is mitral valve regurgitation (MR). The mitral valve apparatus is affected in primary MR, whereas the left ventricle is affected in secondary MR. The diagnosis and management of MR are typically difficult, and it necessitates an organized strategy that incorporates data from the history, physical examination, and imaging. Treatment decisions are based on an understanding of the etiology, natural history, and outcome of therapies for these mitral valve disease patients. To assess the origin of mitral valve disease, the severity of regurgitation, and the effect of volume overload on the left ventricle, as well as determine if a durable valve repair can be performed, each patient must undergo a full 2-dimensional and Doppler echocardiography. Advances in surgical and catheter-based therapy have led to recommendations for lower operation thresholds and the expansion of interventional treatments to the older, sicker MR population. The pathophysiological rationale for current diagnostic and management procedures in MR is discussed in this study [51].

9. CONCLUSION

Mitral valve regurgitation is a valvular illness that is becoming more widespread, with a variety of causes and symptoms. MR has a high mortality rate due to comorbidities, especially in the older population, and the only definite treatment is surgery. MR is commonly misdiagnosed and undertreated, leading to a rise in its prevalence. Evidence-based guidelines in the diagnosis and therapy of valvular disorders have been developed by the American College of Cardiology (ACC) and the American Heart Association (AHA).

Early diagnosis and management have shown to be lifesaving, and advances in medicine and valvular studies have contributed to a dramatic decrease in the disease's morbidity and death. The development of a team of specialist healthcare providers, such as cardiologists and cardiac surgeons, has been found to improve patient care significantly due to efficiency in inpatient management and decision-making. Cardiology nurses keep track of patients, educate them, and help the team communicate by reporting any findings to the relevant clinical professionals. Pharmacists verify the dose, look for drug interactions, and educate patients and healthcare providers. Furthermore, patient care does not end in the operating room; postoperative care necessitates a team of health professionals who are critical for improved patient outcomes and morale. Patients with mitral valve regurgitation may benefit from these interprofessional team techniques.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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