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Mitral valve disease

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MITRAL VALVE DISEASE

Abstract. Mitral valve disease is a frequent cause of heart failure and death. Emerging evidence indicates that the mitral valve is not a passive structure, but even in adult life remains dynamic and accessible for treatment. This concept motivates efforts to reduce the clinical progression of mitral valve disease through early detection and modification of underlying mechanisms. Discoveries of genetic mutations causing mitral valve elongation and prolapse have revealed that growth factor signaling and cell migration pathways are regulated by structural molecules in ways that can be modified to limit progression from developmental defects to valve degeneration with clinical complications. Mitral valve enlargement can determine left ventricular outflow tract obstruction in hypertrophic cardiomyopathy, and might be stimulated by potentially modifiable biological valvular–ventricular interactions. Mitral valve plasticity also allows adaptive growth in response to ventricular remodeling. However, adverse cellular and mechanobiological processes create relative leaflet deficiency in the ischemic setting, leading to mitral regurgitation with increased heart failure and mortality. Our approach, which bridges clinicians and basic scientists, enables the correlation of observed disease with cellular and molecular mechanisms, leading to the discovery of new opportunities for improving the natural history of mitral valve disease.

Introduction

Mitral valve insufficiency is a major source of morbidity and death worldwide and a frequent cause of heart failure, with complications that include arrhythmia, endocarditis, and sudden cardiac death [1, 2]. Structural deficiencies in the mitral valve and secondary changes induced by abnormal ventricular size and deformation are implicated in the development of these valvular lesions [3]. However, the effect of mitral insufficiency on cardiac function is more than purely mechanical, whereby pump function is maintained at the expense of elevated filling pressures, but extends to impaired contractility and electrical instability.

Mitral valve diseases (MVDs) that lead to valve insufficiency have long been conceived as inexorable processes. The underlying genetic mutations and mechanisms of mitral valve dysfunction associated with myxomatous degeneration have remained elusive [4]. In mitral valve lesions associated with ventricular disease, the leaflets are considered biologically passive and fixed in size relative to the enlarged or narrowed ventricle and not an available target for therapy [5]. Valve plasticity—defined as the potential for change in cellular phenotype and behavior—and altered leaflet matrix and micromechanics, have been considered in studies of valve development. However, MVD had not been associated with modified adult valve biology until the 2014 discovery in a mouse model of Marfan syndrome, in which a mutation in an extra cellular matrix (ECM) protein alters the regulation of aorta and valve cell biology and thereby creates opportunities for modifying the course of the disease [6] Mitral valve insufficiency is a major source of morbid-ity and death worldwide and a frequent cause of heart failure, with complications that include arrhythmia, endocarditis, and sudden cardiac death. Structural deficiencies in the mitral valve and secondary changes induced by abnormal ventricular size and deformation are implicated in the development of these valvu-lar lesions. However, the effect of mitral insufficiency on cardiac function is more than purely mechanical, whereby pump function is maintained at the expense of elevated filling pressures, but extends to impaired contractility and electrical instability [7, 8].

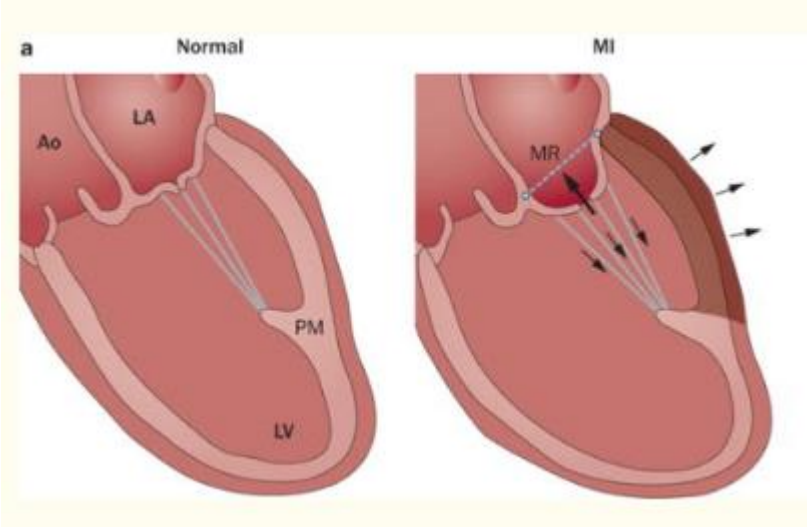
Mitral valve diseases (MVDs) that lead to valve insuf-f Mitral valve diseases (MVDs) that lead to valve insufficiency have long been conceived as inexorable processes (Figure (1) a) for which therapeutic options are limited to surgical valve repair or replacement, with the under-lying mechanisms inaccessible. The under lying genetic mutations and mechanisms of mitral valve dysfunction associated with myxomatous degeneration have remained elusive. Science have long been conceived as inexorable processes (Figure (1)a) for which therapeutic options are limited to surgical valve repair or replacement, with the under-lying mechanisms inaccessible. The under lying genetic mutations and mechanisms of mitral valve dysfunction associated with myxomatous degeneration have remained elusive [9].

Mitral valve disease, which is located between your left heart chambers (left atrium and left ventricle), does not work properly.Types of mitral valve disease include :Mitral valve stenosis and Mitral valve regurgitation. In Mitral valve stenosis condition, the flaps of the mitral valve become thick or stiff, and they may fuse together. This results in a narrowed valve opening and reduced blood flow from the left atrium to the left ventricle [10].

Ischemic mitral regurgitation

Definition and mechanisms

2D echocardiography has been used to demonstrate that apically restricted leaflet closure in the ventricle, owing to papillary muscle displacement causing tether-ing on the mitral leaflets, is the mechanism of ischemic mitral regurgitation (IMR) in patients with either localized inferior wall ischemia or global dilatation and failure. The use of 3D echocardiography in experimental studies has proved that global left ventricular dysfunction alone is insufficient to cause IMR. Displacement and abnormal contraction of the left ventricular wall underlying the papillary muscles, along with decreased shortening of the distance between the papillary muscles, causes mitral leaflet tethering and restricted closure that leads to IMR as described in figure (1) [10].



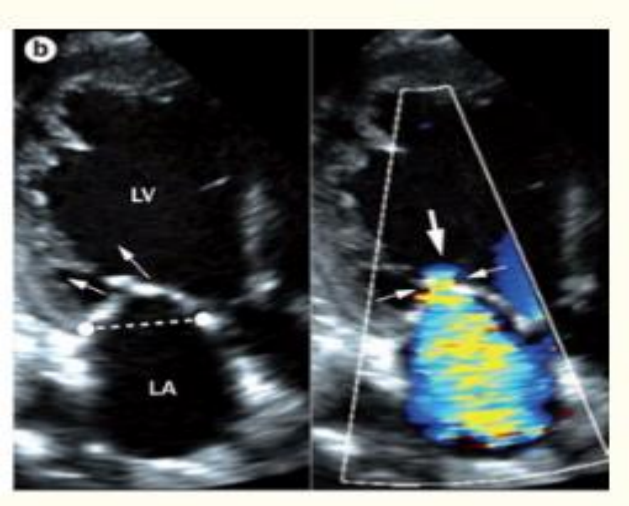


Figure (1): Mechanism of ischemic mitral regurgitation.

a| Mechanism of ischaemic mitral regurgitation caused by increased mitral leaflet tethering owing to left ventricular remodelling after MI (shaded wall) with outward bulging (three arrows on the outer surface of the heart). Leaflet closure is restricted by increased tethering forces on the leaflets exerted via the chordae (arrows within the heart, exceeding normal on the left).

b | Echocardiogram from a patient with inferior wall infarction and tethered mitral leaflets (arrows in left panel) with characteristic anterior leaflet bend and concavity towards the LA indicating chordal tethering, mitral regurgitation orifice (small arrows in right panel), and mitral regurgitant flow (large arrow in right panel). Abbreviations: Ao, aorta; LA, left atrium; LV, left ventricle; MI, myocardial infarction; MR, mitral regurgitation; PM, papillary muscle.

Aetiology:

- Rheumatic fever
- congenital
- carcinoid
- Sle
- Rheumatoid arthritis

Pathophysiology

• Normal mitral valve area = 4-6 cm2.

• A mitral valve area ≤ 1cm2 equates to severe mitral stenosis.

• Symptoms usually develop when mitral valve area ≤ 2.5cm2

• Symptoms in mild mitral stenosis usually precipitated by exercise, emotional stress, infection, pregnancy or fast atrial fibrillation.

Mitral Regurgitation

Mitral valve regurgitation — also called mitral regurgitation, mitral insufficiency or mitral incompetence — is a condition in which your heart's mitral valve doesn't close tightly, allowing blood to flow backward in your heart. If the mitral valve regurgitation is significant, blood can't move through your heart or to the rest of your body as efficiently, making you feel tired or out of breath .

Causes

Mitral valve prolapses. In this condition, the mitral valve's leaflets bulge back into the left atrium during the heart's contraction. This common heart defect can prevent the mitral valve from closing tightly and lead to regurgitation.

Damaged tissue cords. Over time, the tissue cords that anchor the flaps of the mitral valve to the heart wall may stretch or tear, especially in people with mitral valve prolapse. A tear can cause leakage through the mitral valve suddenly and may require repair by heart surgery. Trauma to the chest also can rupture the cords. Rheumatic fever. Rheumatic fever a complication of untreated strep throat can damage the mitral valve, leading to mitral valve regurgitation early or later in life. Rheumatic fever is now rare in the United States, but it's still common in developing countries. Endocarditis. The mitral valve may be damaged by an infection of the lining of the heart (endocarditis) that can involve heart valves. Heart attack. A heart attack can damage the area of the heart muscle that supports the mitral valve, affecting the function of the valve. If the damage is extensive enough, a heart attack can cause sudden and severe mitral valve regurgitation.

Abnormality of the heart muscle (cardiomyopathy). Over time, certain conditions, such as high blood pressure, can cause your heart to work harder, gradually enlarging your heart's left ventricle. This can stretch the tissue around

your mitral valve, which can lead to leakage. Trauma. Experiencing trauma, such as in a car accident, can lead to mitral valve regurgitation. Congenital heart defects. Some babies are born with defects in their hearts, including damaged heart valves. Certain drugs. Prolonged use of certain medications can cause mitral valve regurgitation, such as those containing ergotamine (Cafergot, Migergot) that are used to treat migraines and other conditions. Radiation therapy. In rare cases, radiation therapy for cancer that is focused on the chest area can lead to mitral valve regurgitation. Atrial fibrillation. Atrial fibrillation is a common heart rhythm problem that can be a potential cause of mitral valve regurgitation.

Clinical Features

• Breathlessness (pulmonary congestion)
• Fatigue (low cardiac output)
• Oedema, ascites (right heart failure)
• Palpitation (atrial fibrillation)
• Haemoptysis (pulmonary congestion, pulmonary embolism)
• Cough (pulmonary congestion)
• Chest pain (pulmonary hypertension)
• Thromboembolic complications (e.g. stroke, ischaemic limb)

Signs
• Atrial fibrillation
• Auscultation
Loud first heart sound, opening snap Mid-diastolic murmur
• Crepitations, pulmonary oedema, effusions (raised Pulmonary capillary pressure)

Investigations

ECG
• P mitral or atrial fibrillation
• Right ventricular hypertrophy: tall R waves in V1–V3
Chest X-ray

• Enlarged LA and appendage

• Signs of pulmonary venous congestion

Echo

• Thickened immobile cusps

• Reduced valve area

• Reduced rate of diastolic filling of LV

• Enlarged LA

Doppler

• Pressure gradient across mitral valve

• Pulmonary artery pressure

• Left ventricular function Cardiac catheterisation

• Coronary artery disease

• Mitral stenosis and regurgitation

• Pulmonary artery pressure

Management

Medical treatment

• The asymptomatic patient with mild mitral stenosis should be managed medically. Medical therapy includes:

- Avoidance of unusual physical stress.
- Salt restriction.
- Diuretics if needed.
- Control of heart rate – β-blocker or digoxin.
- Anticoagulation for AF or prior embolic event.
- Annual follow-up.
- Echocardiography if deterioration in clinical condition.

Management of symptomatic mitral stenosis

• Patients with symptoms should undergo clinical re-evaluation with echocardiography.

• NYHA class II symptoms and mild mitral stenosis may be managed medically.

• NYHA class II symptoms and at least moderate stenosis (MVA≤1.5cm2 or mean gradient ≥5mmHg) may be considered for balloon valvuloplasty.

• NYHA class III or IV symptoms and severe mitral stenosis should be considered for balloon valvuloplasty or surgery.

Surgical:

Mitral balloon valvuloplasty and valve replacement.

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