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World Heart Day

Tech Pharma
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29 SEP

Theme:

"Use Heart for Action"

Issue-14 (December) 2024

Dr. D. Y. Patil College of Pharmacy, Akurdi, Pune-44

VISION

To impart quality education to the Students and mould them into proactive multifaceted Pharmacists.

MISSION

To establish a centre of Academic excellence and research in Pharmacy Education and thereby produce professionally competent and ethically sound Pharmacist to cater to the needs of the global society.

PROGRAM EDUCATIONAL OBJECTIVES (PEOs)

After graduation students will

1. Reflect critical thinking and problem solving skills through their Pharmaceutical knowledge, expertise and competency in industry, higher studies and research.
2. Practice ethics and values in their profession.
3. Contribute effectively in various fields of social healthcare system.
4. Inculcate leadership and entrepreneurship capabilities through effective communications, appropriate time management and self-upgradation.
5. Foster interdisciplinary engagement in evolving healthcare sector through lifelong learning.

Dr. R. G. Katedeshmukh

Dr. P. P. Wankhade

Mrs. K. S. Bhagat

Mrs. A. R. Marale

The World Heart Day 2024 is commemorated with a powerful call to "Use Heart for Action". This emphasizes the urgency of raising awareness on heart health and accelerating actions to prevent, detect early, and manage cardiovascular diseases (CVDs). Cardiovascular diseases remain a significant global health challenge responsible for over 18 million deaths each year. The primary causes of this high burden include modifiable lifestyle practices such as tobacco use, unhealthy diets, particularly those high in salt, physical inactivity, and alcohol consumption. In addition, drug treatment of hypertension, diabetes and high lipids are necessary to reduce acute events of CVDs.

Dr. Ramesh Katedeshmukh, Professor

Each year, World Heart Day focuses on a specific theme. The 2024 theme is 'Use Heart for Action.' It emphasises the importance of taking real, tangible steps to improve heart health. From encouraging individual people to take responsibility for their own heart health to advocating for stronger policies and initiatives at a national level, it's a holistic approach.

Dr. Pavankumar Wankhade, Assistant Professor

World Heart Day, celebrated annually on September 29th, is a global initiative dedicated to raising awareness about cardiovascular health. Spearheaded by the World Heart Federation, it aims to educate individuals, communities, and governments about the importance of heart health and the prevention of cardiovascular diseases (CVDs). The current issue of the journal highlights on awareness of Heart disorders. In this issue we published the articles author by students under the guidance of faculty. It contains detail information about introduction, epidemiology, pathophysiology, risk factor, diagnosis and recent treatments. Thus, this particular issue will be helpful, for all pharmacist to understand their role better.

Mrs. Kajal Bhagat, Assistant Professor

The global impact of World Heart Day is profound. It has united communities, healthcare providers, and governments in the fight against cardiovascular diseases. Many regions, particularly in low- and middle-income countries, where CVDs are on the rise, have adopted the day as a platform to educate people about the dangers of ignoring heart health. World Heart Day plays a critical role in promoting heart health awareness and preventing the leading cause of death worldwide.

Mrs. Aboli Marale, Assistant Professor

"We would like to express our gratitude and heartfelt thanks to our beloved Principal Dr. Niraj Vyawahare for constant support and motivation. We are also grateful to our Vice Principal Dr. (Mrs). Shilpa Chaudhari, all the teaching, non-teaching staff and our students."

Our organization feels special and privileged in presenting this issue.

Thank you once again to all....

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ANEURYSMS

Dr. Ashish Kulkarni, Ms. Pooja Deshmukh
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ABSTRACT:
Aneurysms are localized, abnormal dilations of blood vessels caused by a weakening of the vessel wall, which can occur in various locations throughout the body. They are classified into types such as abdominal aortic aneurysms (AAA), thoracic aortic aneurysms (TAA), cerebral aneurysms, and peripheral aneurysms based on their location. Pathophysiological, aneurysms develop due to structural changes in the vessel wall, including loss of elastin, collagen degradation, and inflammation, often influenced by genetic and environmental factors. Epidemiologically, aneurysms are more common in older individuals, with AAAs predominantly affecting men over 65 years of age, while cerebral aneurysms are often seen in younger adults, particularly women. Risk factors include hypertension, smoking, atherosclerosis, family history, connective tissue disorders, and infections. Diagnosis involves a combination of clinical evaluation and imaging methods like ultrasound, CT angiography, MR angiography, and cerebral angiography, which help determine aneurysm size, location, and possibility of rupture. Treatment strategies depend on aneurysm type, size, and symptoms.

KEYWORD: Abdominal aortic aneurysms (AAA), Thoracic aortic aneurysms (TAA), Cerebral aneurysms, Diagnosis, treatment

INTRODUCTION:

Aneurysms are localized dilations or bulges within a blood vessel's wall, resulting from a weakening of the vessel wall. They can occur in various locations throughout the body, with the most common types being aortic aneurysms (which can be thoracic or abdominal) and cerebral aneurysms. The formation of an aneurysm can lead to serious health complications, particularly if it ruptures, resulting in life-threatening internal bleeding [1] The development of an aneurysm is often associated with a combination of genetic predisposition and environmental factors. Conditions such as hypertension, atherosclerosis, and connective tissue disorders (e.g., Marfan syndrome) are significant contributors to the risk of aneurysm formation. The management of aneurysms typically depends on their size and location. Smaller aneurysms may be monitored through regular imaging studies, while larger or symptomatic ones often require surgical intervention. [2] Understanding aneurysms involves examining their types, causes, symptoms, treatment options, and associated risks.

EPIDEMIOLOGY:

Abdominal Aortic Aneurysms (AAA) [3]

Global Prevalence: Approximately 0.92% among individuals aged 30 to 79 years, translating to about 35.12 million cases worldwide as of 2019 23.

Demographics:

Higher prevalence in men (1.46%) in contrast to women (0.39%), with a ratio of 3.7:1

The highest prevalence is found in the Western Pacific region at 1.31%, while the African region has the lowest at 0.33% 23. Age Factor: Incidence increases significantly after age 60

Thoracic Aortic Aneurysms (TAA)[4]

Incidence: Estimated at approximately 5.3 per 100,000 individuals per year, with a prevalence of about 0.16% in the general population

Incidence: Estimated at approximately 5.3 per 100,000 individuals per year, with a prevalence of

about 0.16% in the general population.

Cerebral Aneurysms [5]

Prevalence in the U.S.: Approximately 6.7 million people, or about 1 in 50 individuals, have unruptured cerebral aneurysms.

Annual Rupture Rate: Estimated at about 8 to 10 per 100,000 people, leading to around 30,000 ruptures each year

PATHOPHYSIOLOGY:

The process begins with mechanical stress on the aortic wall, leading to endothelial injury and subsequent inflammation. Key pathological processes include proteolysis, where matrix metalloproteinase (MMPs) degrade elastin and collagen, resulting in loss of elasticity and structural integrity. This is compounded by oxidative stress and inflammatory responses, which recruit immune cells that further damage the vessel walls. The Vascular smooth muscle cell (VSMC) apoptosis reduces the wall's ability to withstand pressure, while compensatory collagen deposition can create additional structural weaknesses [6]

Thoracic aortic aneurysms share similar mechanisms but often involve different hemodynamic stresses due to their location. The pathogenesis includes extracellular matrix (ECM) remodeling, characterized by changes in collagen and elastin composition, which compromise the wall's strength. Inflammation plays a significant role, with immune cell infiltration leading to further degradation of the ECM and promoting aneurysm expansion [7,8]

Cerebral aneurysms, on the other hand, typically develop at arterial bifurcations where turbulent blood flow creates localized stress. Here, endothelial dysfunction initiates inflammatory cascades that contribute to vascular remodeling. The accumulation of inflammatory cells and the activation of proteolytic enzymes lead to weakening of the vessel wall, making it susceptible to rupture. [9]

Types of Aneurysms

Aneurysms can be classified based on their location and shape:

Aortic Aneurysm:

Thoracic Aortic Aneurysm (TAA): Thoracic aortic aneurysms (TAAs) are abnormal bulges in the wall of the thoracic aorta, which is the main artery that supplies blood to the body's other organs from the heart. These aneurysms can develop gradually and often remain asymptomatic until they reach a significant size or rupture, making them particularly dangerous. [10] TAAs account for approximately 25% of all aortic aneurysms and can occur in various sections of the thoracic aorta, which includes the descending thoracic aorta, ascending aorta, and aortic arch. [11]

Symptoms: Difficulty breathing, coughing, hoarseness, dysphagia

Abdominal Aortic Aneurysm (AAA):

Abdominal aortic aneurysms (AAAs) are localized dilations of the abdominal aorta, The body's biggest artery, which carries blood from the heart to the abdomen, pelvis, and legs. AAAs are most commonly found in men over the age of 65, with studies indicating that approximately 8% of this population may be affected. [12]

Symptoms: persistent abdominal or back pain, a pulsating sensation in the abdomen, and discomfort that can radiate to the legs or buttocks.

Cerebral Aneurysm:

Cerebral aneurysms, also known as brain aneurysms or intracranial Aneurysms are irregular protuberances that develop in the wall. of a blood vessel in the brain due to a weakness in the vessel wall. These aneurysms can vary in size and may remain asymptomatic until they rupture, leading to severe complications such as subarachnoid haemorrhage (SAH), which is a type of bleeding that occurs in the space surrounding the brain. The rupture of a cerebral aneurysm is a medical emergency that can result in devastating outcomes, including stroke, brain damage, or death. [13]

Symptoms: headaches, vision changes, eye pain,

numbness on one side of the face, stiff neck. [14]

Peripheral Aneurysm:

The vascular condition known as peripheral arterial disease (PAD) is typified by artery narrowing. outside the heart and brain, primarily affecting the blood vessels in the legs. This condition is often caused by atherosclerosis, where fatty deposits, or plaques, accumulate in the arterial walls, restricting blood flow. The classic symptom of PAD is intermittent claudication, which manifests as pain or cramping in the legs or buttocks during physical activities such as walking or climbing stairs; this pain typically resolves with rest [15]

Symptoms: weak or absent pulses in the legs or feet, coldness in the lower leg, changes in skin colour, slow-healing sores on the toes or feet, and hair loss on the legs [16]

Ventricular aneurysm

ventricular aneurysm is a serious cardiac condition characterized by an irregular dilatation or bulging in a ventricle, most commonly the left ventricle, which is The primary pumping chamber of the heart. [17] This condition typically arises as a complication following a myocardial infarction (heart attack), where damage to the heart muscle leads to the formation of scar tissue. As the heart muscle heals, the weakened area may stretch and bulge outward under the pressure of blood, resulting in a pouch-like structure filled with blood. [18]

Symptoms: chest pain, exhaustion, palpitations, ankle or leg edema, and shortness of breath due to fluid retention.

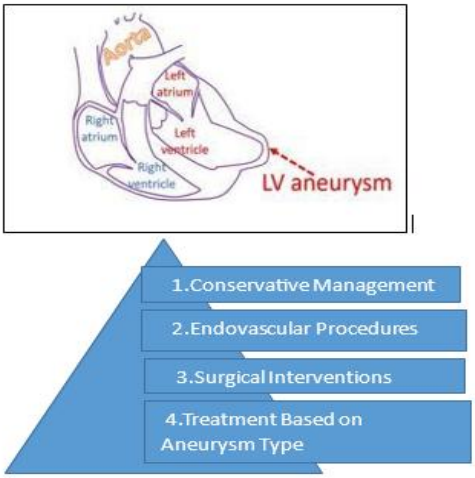

Fig 1: Ventricular aneurysm

RISK FACTOR:

Aneurysms are caused by a combination of non-modifiable and modifiable risk factors that contribute to the weakening of blood vessel walls. Among the non-modifiable factors, **age** plays a significant role, as the risk increases with advancing years, particularly in individuals over 50. **Sex** also influences susceptibility, with males being at higher risk for abdominal aortic aneurysms (AAA) and females for cerebral aneurysms. A family history of aneurysms and genetic disorders such as Marfan syndrome**,** Ehlers-Danlos syndrome, and polycystic kidney disease further elevate the risk due to inherited defects in connective tissue. [19]

DIAGNOSIS:

Ultrasound is the preferred first-line tool for screening abdominal aortic aneurysms, offering a safe and non-invasive method to measure aneurysm size. [20]

Computed Tomography (CT) Angiography is the gold standard for diagnosing aortic and cerebral aneurysms, providing detailed 3D images that accurately measure the size, shape, and location of the aneurysm. In patients with allergies to contrast agents or requiring long-term monitoring. [20]

Magnetic Resonance Angiography (MRA) is an excellent alternative, especially for cerebral aneurysms. [21]

Cerebral angiography remains the definitive diagnostic tool, offering precise visualization of blood vessels. Additionally, X-rays may incidentally detect aneurysms through calcifications, though their diagnostic role is limited. [22]

TREATMENT:

Conservative Management

Conservative treatment for aneurysms focuses on slowing their progression and reducing the risk of rupture, particularly for smaller, asymptomatic aneurysms. Blood pressure control is a priority, with drugs such as calcium channel blockers, ACE inhibitors, or beta-blockers used to minimize stress on the arterial walls. Lifestyle modifications are essential, including smoking cessation, maintaining a healthy weight, regular exercise, and adopting a heart-healthy diet to manage cholesterol levels and overall vascular health. Regular monitoring with imaging techniques, such as ultrasound for abdominal aortic aneurysms. [23]

Endovascular Procedures

In endovascular aneurysm repair (EVAR), commonly used for abdominal and thoracic aortic aneurysms, a stent-graft is inserted through the femoral artery using a catheter to reinforce the weakened blood vessel and restore normal blood flow. For cerebral aneurysms, endovascular coiling involves placing platinum coils into the aneurysm sac to induce clotting and seal it off, while flow diverters are used for large or complex aneurysms to redirect blood flow and promote healing. [24]

Surgical Interventions

In open surgical repair, commonly used for abdominal and thoracic aortic aneurysms, the weakened section of the artery is replaced with a synthetic graft, such as Dacron or PTFE, to restore normal blood flow and reinforce the vessel wall. For cerebral aneurysms, surgical clipping is performed through a craniotomy, where a small metal clip is placed at the neck of the aneurysm to stop blood flow and prevent rupture. Surgical intervention is highly effective and durable but requires general anesthesia, longer recovery times, and carries higher risks of complications such as bleeding or infection. [25]

Treatment Based on Aneurysm Type

Small AAAs (less than 5 cm) are managed conservatively with blood pressure control, lifestyle modifications, and regular monitoring through ultrasound. Smaller aneurysms are monitored closely with imaging and controlled through medications to lower blood pressure. For large, symptomatic, or rapidly growing TAA.For Thoracic Aortic Aneurysm: Small aneurysms: Monitoring and blood pressure management. Large aneurysms: Surgical repair with graft placement or thoracic endovascular aneurysm repair (TEVAR). Cerebral Aneurysm: Small, unruptured aneurysms: Conservative management and monitoring. Large or ruptured aneurysms: Endovascular coiling, flow diverter placement, or surgical clipping. [26]

CONCLUSION:

Aneurysms are serious vascular conditions that, if left untreated, can lead to life-threatening complications such as rupture or thrombosis. Early identification through risk factor assessment and advanced imaging techniques plays a crucial role in preventing catastrophic outcomes. Management strategies, including conservative treatment, minimally invasive endovascular procedures, and surgical interventions, are tailored based on the aneurysm's type, size, location, and risk of rupture. Lifestyle modifications, such as smoking cessation and blood pressure control, are essential in reducing the progression of aneurysms. Advances in imaging and treatment options have significantly improved patient outcomes, but ongoing monitoring and timely intervention remain

critical for long-term success. A multidisciplinary approach is necessary to ensure early diagnosis, effective treatment, and prevention of complications, ultimately improving patient prognosis and quality of life.

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ANGINA PECTORIS: A COMPREHENSIVE REVIEW OF ITS PATHOPHYSIOLOGY, DIAGNOSIS, TREATMENT, AND RECENT THERAPEUTIC TARGETS

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ABSTRACT:

Angina pectoris is a clinical manifestation of myocardial ischemia, commonly caused by coronary artery disease (CAD). The condition is characterized by chest pain due to an imbalance between oxygen supply and demand in the myocardium. This review explores the epidemiology, etiology, pathophysiology, and clinical presentation of angina pectoris. It also discusses various diagnostic tools and management strategies, including pharmacological, non-pharmacological, and Ayurvedic treatment modalities. Special emphasis is placed on recent advancements in drug therapies targeting novel pathways, offering a promising outlook for angina management. The review aims to provide an extensive overview of the condition and its treatment options.

INTRODUCTION:

Angina pectoris is a prevalent cardiovascular disorder associated with chest pain resulting from myocardial ischemia. The condition is most commonly caused by atherosclerotic coronary artery disease (CAD), but can also result from coronary artery spasm or other less common etiologies. Angina typically presents as discomfort or pain in the chest, which occurs when the heart muscle is deprived of sufficient oxygenated blood. The symptoms of angina can be transient, but in some cases, they may herald more serious complications like myocardial infarction (MI) or heart failure. Angina pectoris remains a major cause of morbidity and mortality worldwide, and its management is critical to improving patient outcomes.

EPIDEMIOLOGY:

Angina pectoris is one of the most prevalent cardiovascular conditions globally. According to the American Heart Association (2020), approximately 3 to 4 million adults in the United States alone suffer from some form of angina. The prevalence increases with age, with individuals over 45 years being at higher risk. In particular, men are more frequently affected at younger ages, while women develop angina at older ages, especially post-menopause. Risk factors such as hypertension, diabetes mellitus, dyslipidemia, and smoking are strongly associated with the development of angina.

Globally, the prevalence of angina is rising due to the increasing incidence of risk factors like sedentary lifestyles, poor dietary habits, and obesity. In high-income countries, angina remains a significant cause of hospital admissions and healthcare burden. However, in low- and middle-income nations, the lack of access to healthcare resources may delay the diagnosis and management of angina, leading to worse outcomes[1].

ETIOLOGY:

The most common cause of angina pectoris is coronary artery disease (CAD), a condition in which the coronary arteries become narrowed or blocked due to the buildup of fatty plaques, a process known as atherosclerosis. This leads to reduced blood flow to the myocardium, particularly during times of increased demand such as physical activity or stress. In addition to atherosclerosis, other less common causes of angina include coronary artery spasm, microvascular disease, and anemia, which may further compromise oxygen delivery to the heart muscle.

Fig. 1 Etiology of angina pectoris

Risk Factors: The risk factors for angina are the same as those for coronary artery disease and include:

Hypertension: Increases the workload on the heart and accelerates the process of atherosclerosis.

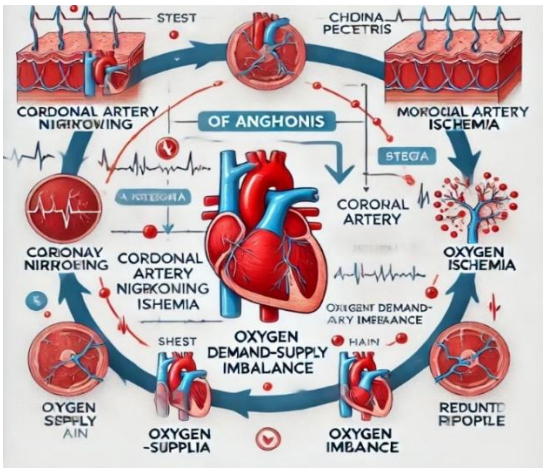
Hyperlipidemia: High cholesterol levels lead to the formation of plaques in the coronary arteries.

Smoking: Contributes to endothelial dysfunction and increases the risk of thrombosis.

Diabetes mellitus: Causes endothelial damage and accelerates the development of CAD.

Obesity: Increases the likelihood of developing hypertension, diabetes, and hyperlipidemia.

3 PATHOPHYSIOLOGY:



The pathophysiology of angina involves an imbalance between myocardial oxygen supply and demand. The heart muscle requires a constant supply of oxygenated blood to function efficiently. When this supply is reduced, ischemia occurs, leading to the clinical symptoms of angina. The mechanisms underlying this imbalance can be summarized as follows:

Coronary Artery Narrowing: Atherosclerotic plaques reduce the lumen of coronary arteries, impairing blood flow.

Increased Oxygen Demand: Situations such as physical exertion, emotional stress, or tachycardia increase the heart's demand for oxygen.

Ischemia: The narrowed coronary arteries cannot meet the heart's oxygen needs, leading to ischemia.

Metabolic Byproducts: Ischemic myocardium accumulates lactate and other metabolic byproducts, leading to chest pain.

Anginal Pain: The accumulation of metabolites triggers pain receptors in the heart muscle, resulting in the typical chest pain associated with angina.

In **unstable angina**, plaque rupture and the formation of a thrombus can lead to more severe and persistent ischemia, making this a medical emergency with a higher risk of myocardial infarction (MI)[2].

SIGNS AND SYMPTOMS:

The hallmark symptom of angina pectoris is chest pain, which is often described as a squeezing, pressure-like, or constricting sensation in the chest. The pain may radiate to the left arm, jaw, neck, or back, and is typically brought on by physical exertion or emotional stress. The pain is often relieved by rest or nitroglycerin. Other associated symptoms include:

Shortness of breath (dyspnea)

Fatigue

Sweating (diaphoresis)

Nausea or vomiting

Dizziness or lightheadedness

Instable angina, pain typically resolves with rest or medication, whereas in unstable angina, the pain may occur unpredictably, at rest, and last longer, and may require immediate medical attention[3].

DIAGNOSIS:

The diagnosis of angina pectoris is based on the clinical presentation and supported by a variety of diagnostic tests, including:

Electrocardiogram (ECG): Changes such as ST-segment depression or T-wave inversions may indicate ischemia.

Stress Testing: Exercise or pharmacological stress testing is used to provoke symptoms and evaluate myocardial ischemia under stress conditions.

Coronary Angiography: The gold standard for diagnosing coronary artery disease, it provides a detailed view of coronary artery blockages.

Cardiac Biomarkers: Elevated troponin levels can differentiate between angina and myocardial infarction.

Echocardiography: This imaging modality is used to assess cardiac function and structural abnormalities

TREATMENT:

The management of angina pectoris includes both pharmacological and non-pharmacological strategies aimed at reducing symptoms, improving prognosis, and preventing complications such as myocardial infarction.

1. Pharmacological Treatment:

Nitrates (e.g., nitroglycerin): These drugs dilate coronary arteries and reduce myocardial oxygen demand, providing relief from angina symptoms.

Beta-Blockers (e.g., metoprolol): Lower heart rate and blood pressure, which decreases oxygen consumption.

Calcium Channel Blockers (e.g., amlodipine): Help to relax and dilate the coronary arteries, improving blood flow.

Antiplatelet Agents (e.g., aspirin): Prevent clot formation and reduce the risk of heart attack.

Statins (e.g., atorvastatin): Lower cholesterol and stabilize plaques, preventing further atherosclerosis.

2. Non-Pharmacological Treatment:

Lifestyle Modifications: Smoking cessation, a healthy diet, regular exercise, and weight management are critical in managing angina.

Cardiac Rehabilitation: A multidisciplinary program that includes supervised exercise and education, aimed at improving heart health and reducing the risk of future cardiac events.

Surgical Interventions: In patients with significant coronary artery blockage, procedures such as coronary artery bypass grafting (CABG) or percutaneous coronary interventions (PCI) may be necessary.

3. Ayurvedic Treatment:

Ayurveda, an ancient system of medicine from India, offers a holistic approach to treating angina pectoris. Ayurvedic treatments focus on restoring balance among the body's three doshas—Vata, Pitta, and Kapha. Herbal remedies that are commonly used in the management of angina include:

Arjuna (Terminalia arjuna): Known for its cardioprotective effects, it strengthens the heart and improves blood circulation.

Ashwagandha (Withania somnifera): Reduces stress, enhances vitality, and supports heart health.

Guggul (Commiphora wightii): Used for its anti-inflammatory properties and its ability to lower cholesterol and improve circulation.

Although Ayurvedic remedies can offer supportive benefits, they should not replace conventional medical treatment, especially in severe cases[4].

RECENT TARGETS OF DRUG ACTION:

Advancements in the treatment of angina have focused on novel therapeutic targets, including:

PCSK9 Inhibitors: These drugs, such as evolocumab, reduce LDL cholesterol levels, preventing the formation of atherosclerotic plaques and improving heart health[5].

Ranolazine: This drug inhibits late sodium channels, reducing myocardial ischemia and offering an alternative for patients with chronic angina.

Inflammatory Pathways: New therapies targeting inflammation in the coronary arteries, such as interleukin inhibitors, are being explored as potential treatments for angina and CAD[6].

CONCLUSION:

Angina pectoris is a complex and common condition with a significant impact on global health. While established therapies remain essential in its management, recent advancements in drug therapy, including the targeting of new pathways and the use of personalized medicine, offer promising alternatives for improving patient outcomes. Furthermore, Ayurvedic treatments can provide adjunctive benefits, promoting heart health and overall well-being. A multidisciplinary approach, including lifestyle changes, pharmacotherapy, and surgical interventions, remains crucial for effective management of angina.

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ARRHYTHMIA (IRREGULAR HEARTBEAT)

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ABSTRACT:
Cardiac arrhythmia characterized by irregular heart rhythms can manifest as either excessively rapid (tachyarrhythmia) or slow (bradyarrhythmia) heart rates. These irregularities arising from disruptions in electrical impulses within the heart pose significant health risks ranging from benign conditions to life-threatening complications. The prevalence of arrhythmias such as atrial fibrillation affecting over 37 million individuals globally highlights the urgent need for effective diagnosis and management strategies. Risk factors include stress, lifestyle changes, and structural or electrical remodeling of cardiac tissues.
Electrocardiography (ECG) remains a pivotal non-invasive diagnostic tool for identifying arrhythmic patterns. Treatment options encompass pharmacological approaches, such as antiarrhythmic drugs and anticoagulants, and non-pharmacological interventions like pacemakers, catheter ablation, and lifestyle modifications. Emerging therapies, including ion channel modulation, gene therapy, and biological pacemakers, aim to address the underlying causes of arrhythmias with enhanced efficacy and fewer side effects. Complementary approaches such as traditional remedies and practices like yoga and acupuncture also contribute to arrhythmia management. Advancements in personalized medicine and innovative technologies including nanotechnology and targeted drug delivery hold promise for improving outcomes. Emphasizing early detection, preventive care, and multidisciplinary management is essential in reducing morbidity and mortality associated with arrhythmias.

INTRODUCTION:

A cardiac arrhythmia can occur at any age and is characterized by an irregular heartbeat that is either excessively rapid (>100 beats per minute) or too slow (<60 beats per minute).[1] People's lifestyles have gotten considerably more complex in the modern period as a result of stress, worry, and depression being a part of everyday life. Heart conditions are becoming more common in both young people and the elderly in such a situation. It is also noted that heart conditions are delicate and important with potentially fatal consequences. [2] Arrhythmia is detected in two categories as in first one single irregularity in heart beats also known as morphological arrhythmia and second class of arrhythmia is classified as multiple irregular heartbeats. [2] The electrocardiogram (ECG) which analyzes the heart's electromagnetic activity is a simple and non-invasive method of identifying cardiac problems. It is feasible to identify some of the heart's anomalies by examining the electrical signal of each heartbeat, which is the sum of the action impulse waveforms generated by the many expert cardiac tissues. [1,3] A competent doctor's treatment of a severe arrhythmia can often save a patient's life. Procaine amide (Pronestyl) hydrochloride, quinidine, carotid sinus stimulation, and the quickly acting digitalis preparations are the four primary treatment approaches that can be used effectively to achieve this. [4] A cardiac arrhythmia might have the sensation of a speeding, hammering, or fluttering heartbeat. There are certain benign cardiac arrhythmias. Others could produce symptoms that are fatal. A rapid or slow heartbeat is acceptable under some situations. For instance, the heart may beat more quickly when exercising or more slowly when you sleep. Treatment options for cardiac arrhythmias may involve medication, pacemaker devices, surgery, or other procedures. Treatment aims to reduce or eliminate rapid, sluggish, or otherwise irregular heartbeats. Living a heart-healthy lifestyle can

help avoid heart damage which can lead to some cardiac arrhythmias. [5,7] Certain arrhythmias are benign and don't need medical attention. You may be at risk for cardiac arrest from other people. Many people fall somewhere between these two extremes. The type of arrhythmia you have and the sort of therapy you require if any can be determined by a healthcare professional. [6]

TYPES OF ARRHYTHMIA:

Tachyarrhythmia: An irregular rhythm with a ventricular heart rate of 100 beats per minute or higher is called tachyarrhythmia. [6,8]

Supraventricular arrhythmias: Arrhythmia originating from above the AV node (from atrial origin or AV junction origin).

Atrial fibrillation (AFib)
Atrial flutter
Atrial tachycardia
Atrial premature complex (PAC)Atrioventricular nodal reentrant tachycardia (AVNRT)
Atrioventricular reentrant tachycardia (AVRT)
AV junctional extrasystoles

Ventricular arrhythmias

Ventricular fibrillation (V-fib)
Ventricular premature beats (PVC)
Ventricular tachycardia (sustained or non-sustained)
Bradyarrhythmias and junctional rhythms

EPIDEMIOLOGY:

Disturbances in the heart's regular beat known as arrhythmias can range from benign to fatal. The precise kind, age, sex, ethnicity, and risk factors that are linked to it all affect its epidemiology.

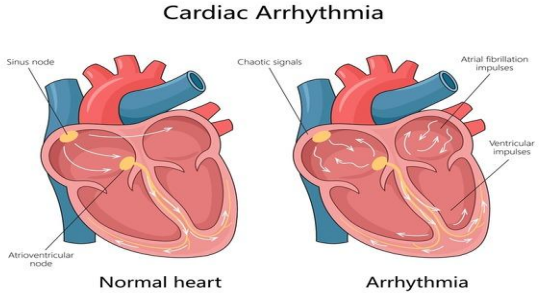
Cardiac arrhythmias another name for heart rhythm problems are among the most common heart illnesses occurring in an estimated 2% of the general population. They are linked to high rates of morbidity, death, and medical expenses. In instance atrial fibrillation is a serious public health issue that affects over 37 million people worldwide and is becoming more common. Significant morbidity is brought on by it such as heart failure and strokes. [8] It is anticipated that between 1.5% and 5% of the general population would have arrhythmias with atrial fibrillation being the most prevalent. Since arrhythmias can be paroxysmal and may or may not cause symptoms it is challenging to determine their actual prevalence. Higher morbidity and death are linked to the overall presence of arrhythmia. [7]

ETIOLOGY AND RISK FACTORS OF ARRHYTHMIA [6,7]

Figure 1. Causes of Arrhythmia and Risk factors of Arrhythmia

PATHOPHYSIOLOGY:

Arrhythmias originate from disturbances in impulse initiation conduction or a combination of both. These disturbances are linked to alterations in ionic currents across cardiac cell membranes affecting the heart's normal electrical activity. Abnormal Impulse Generation.



Automaticity: Normal automaticity involves spontaneous action potentials from the sinoatrial (SA) node, atria, AV junction or His-Purkinje system governed by phase 4 depolarization. Abnormal automaticity arises when working myocardial cells or Purkinje fibers develop spontaneous depolarizations due to reduced resting membrane potential (e.g., after ischemic injury). [10,11]

Triggered Activity: Early afterdepolarizations (EADs): Occur during repolarization (phases 2 or 3) and are associated with prolonged action potentials (e.g., long QT syndrome). Delayed afterdepolarizations (DADs): Occur after repolarization, often due to calcium overload (e.g., digitalis toxicity). [10,11]

Abnormal Impulse Conduction: Reentry is a common conduction abnormality where an impulse travel through a circuit reactivating previously excited tissue. This requires a unidirectional block and slow conduction, often due to reduced resting potential or fibrotic changes. Reentry underlies many arrhythmias including atrial fibrillation and ventricular tachycardia. Contributing Factors Structural remodeling (e.g., fibrosis, hypertrophy) and electrical remodeling (ion channel dysfunction) create substrates for arrhythmias. Autonomic influences such as sympathetic overdrive or parasympathetic withdrawal further modulate arrhythmic risks. This interplay between substrate triggers and modulating factors underpins the complex mechanisms of arrhythmias. [10]

SIGN AND SYMPTOMS: [6,12,13,14,15]

Arrhythmias often present with a range of symptoms which vary depending on the type severity and underlying cause. Common signs and symptoms include:

Symptoms

Palpitations: A sensation of a fast heartbeat. Common in tachyarrhythmias like atrial fibrillation or supraventricular tachycardia.

Dizziness or Lightheadedness: Often experienced prior to syncope (fainting) results from decrease cardiac output and cerebral perfusion.

Syncope (Fainting): Sudden loss of consciousness due to a transient decrease in blood flow to the brain. Frequently associated with ventricular tachyarrhythmias or bradyarrhythmias.

Fatigue: Generalized weakness or tiredness caused by insufficient cardiac output.

Shortness of Breath: May occur during exertion or rest especially in heart failure or atrial fibrillation.

Chest Pain or Discomfort: Due to myocardial ischemia or underlying coronary artery disease. Seen in arrhythmias with rapid heart rates.

Seizure-like Activity: Jerking movements may accompany syncope complicating differentiation from epilepsy.

Signs

Irregular Pulse: Can be fast, slow or erratic depending on the arrhythmia type.

Skin Color Changes: Cyanosis (bluish tint) or pallor may be noted during severe arrhythmias.

Hypotension: Low blood pressure due to poor cardiac output.

Urinary Incontinence: Occasionally reported during syncopal episodes.

These symptoms are variable and may overlap with non-cardiac conditions emphasizing the importance of a thorough clinical history and diagnostic evaluation.

DIAGNOSIS [6,16,17]

•Stress test.
•Ambulatory monitors.
•Blood tests to check your electrolyte levels or look for a genetic issue.
•Electrocardiogram (ECG or EKG).
•Heart MRI (magnetic resonance imaging).
•Computed tomography (CT).
•Tilt table test.
•Cardiac catheterization.
•Echocardiogram.

Table 1. Diagnosis test for Arrhythmia

TREATMENT:

Treatments for arrhythmias often involve medications, surgery to implant heart-controlling devices and other procedures to address issues with electrical impulses in the heart. Making healthy lifestyle adjustments may also be necessary to help reduce your chance of developing diseases like high blood pressure and various forms of heart disease that can exacerbate your arrhythmia.

Pharmacological treatment: [6,18,19,21]

1. Antiarrhythmic drugs

Class I Sodium channel blockers: These medications stop sodium from passing through cell membranes. In the heart muscle this may delay electrical impulses. Mexiletine, quinidine, propafenone, disopyramide, and flecainide are a few examples.

Class II beta blockers: These medications lower heart rate, frequently by inhibiting chemicals like adrenaline. Metoprolol, nadolol, propranolol, atenolol, bisoprolol, and acebutolol are a few examples.

Class III Potassium channel blockers: They stop potassium from passing through cell membranes. All of the heart's cells' electrical impulses may be slowed down by this. Ibutilide, sotalol, amiodarone, bretylium, dofetilide, and dronedarone are a few examples.

Class IV Calcium channel blockers: Heart muscle calcium channels are blocked by these medications. Contractions and heart rate may drop as a result. Examples are verapamil and diltiazem.

Adenosine: This drug slows or stops electrical impulses between the heart's upper and lower chambers at the atrioventricular node.

Digoxin: This medication can raise cardiac contractility and lower heart rate. Other drugs that are not antiarrhythmic, including anticoagulants (blood thinners) may also be used to treat arrhythmias.

The anti-arrhythmic properties of several medications that do not fall within the Vaughan Williams categorization scheme are noteworthy. For instance ranolazine which is mainly used as an antianginal drug due to its late sodium channel (INa-L)-blocking action also works similarly to amiodarone in preventing arrhythmias by blocking inward depolarizing and outward repolarizing currents that affect sodium, potassium and calcium currents to lengthen action potential duration. Ranolazine's main antiarrhythmic effects include blocking peak Ina which lowers excitability and lengthens the end-refractory period (resulting in less atria activation).

Non-Pharmacologic Treatment: [6]

Changes in lifestyle

Controlling blood sugar and blood pressure.
Staying away from tobacco products.
Reducing alcohol consumption.
Avoiding stimulants and coffee.
Attempting to achieve a healthy weight.

Implanted Technology [18,19]

Pacemakers: Used to treat certain conduction system abnormalities and bradycardia symptoms. ICDs or implanted cardioverter defibrillators: Use pacing or shocks to treat potentially fatal ventricular arrhythmias.

Ablation of the catheter: Useful for arrhythmias that don't respond to medicine such as atrial fibrillation. Disrupts aberrant electrical circuits by focusing on certain regions such as the ganglionated plexi or pulmonary veins

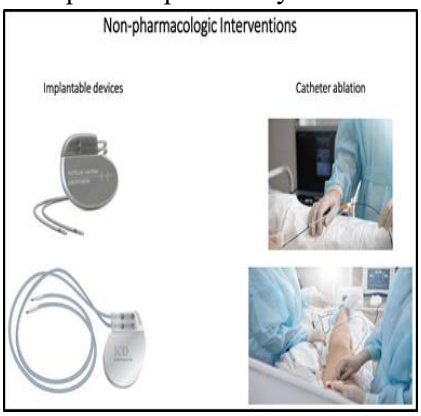


Figure 3: Implantable devices and catheter ablation. [18]

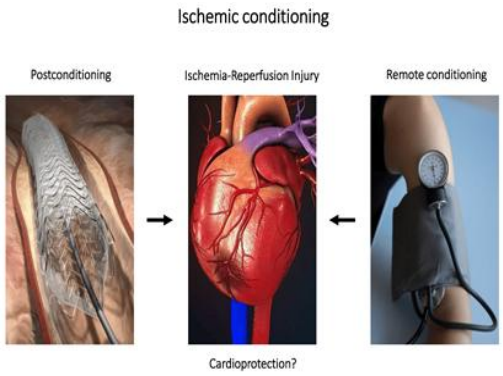


Figure 4: Ischemic Conditioning. [18]

Ischemic Conditioning: Includes both preconditioning and post conditioning caused by ischemia uses regulated ischemia-reperfusion cycles to guard against myocardial damage and reperfusion arrhythmias.

New Interventions: For the creation or repair of pacemaker tissues there are stem-cell therapies, gene therapies, and biological pacemakers.

Ayurveda Treatment

Motherwort (Leonurus cardiaca): Reduces sinus rate by modulating If, ICa.L, and IK.r currents; functions similar to class III antiarrhythmic drugs. [24,25]

Wenxin Keli (Traditional Chinese Medicine): Atrial-selective reduction of INa channels prolongs the atrial effective refractory period. [26]

Cinchona: Contains quinine and quinidine acting as class IA antiarrhythmic agents by prolonging phase 0 and the action potential. [27]

Hawthorne (Crataegus oxycantha): Inhibits potassium channels (IKs and IKr) mimicking class III antiarrhythmic effects.

Khella (Ammi majus): Similar to amiodarone acting on potassium channels.

Barberry (Berberis vulgaris): Prolongs action potential via Ito inhibition resembling class IA and III antiarrhythmic effects.

Omega-3 Fatty Acids: Reduce proarrhythmic fatty acids possibly stabilizing ion channel activity.

Acupuncture: Likely modulates the autonomic nervous system reducing sympathetic tone. [23]

Yoga: Reduces sympathetic tone and enhances parasympathetic activity potentially reducing arrhythmic events. [22,23]

CONCLUSION:

Cardiac arrhythmias encompass a wide spectrum of conditions ranging from benign to life-threatening significantly impacting public health. Early detection and appropriate management whether pharmacological, non-pharmacological or through emerging technologies are crucial in mitigating risks associated with these disorders. Advancements in diagnostics, treatment modalities and personalized approaches have improved patient outcomes. Additionally, lifestyle modifications and complementary therapies play an important role in arrhythmia prevention and management. A holistic and multidisciplinary approach is essential to reduce the burden of morbidity and mortality associated with arrhythmias while enhancing quality of life for affected individuals.

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CARDIAC ARRREST

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INTRODUCTION:

Sudden cardiac arrest (SCA) is the sudden cessation of all heart action due to an abnormal heart rhythm. Breathing pauses. The individual goes unconscious. Sudden cardiac arrest, if not treated immediately, can be fatal. Cardiopulmonary resuscitation (CPR) and heart shocks using an automated external defibrillator (AED) are among the emergency treatments for sudden cardiac arrest. Survival is achievable with prompt, competent medical attention. Sudden cardiac arrest differs from a heart attack. A heart attack occurs when blood flow to a portion of the heart is obstructed. A blockage does not cause sudden cardiac arrest. However, a heart attack can produce a shift in the heart's electrical activity, resulting in sudden cardiac arrest.

REASONS FOR CARDIAC ARREST:

A family history of coronary artery disease.
Smoking.
High blood pressure.
High blood cholesterol.
Obesity.
Diabetes.
An inactive lifestyle.

PREVENTIVE MEASURES FOR CARDIAC ARREST:

Eat healthy.
Stay active and get regular exercise.
Do not smoke or use tobacco.
Have regular checkups.
Get screened for heart disease.
Control blood pressure and cholesterol.

CARDIAC ARREST VS HEART ATTACK

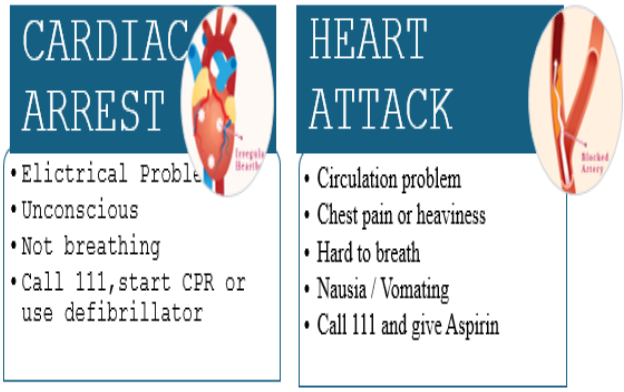

Fig 1: cardiac arrest vs heart attack

CAUSES:

Coronary Artery Disease: Blockages can lead to heart attacks, which may trigger cardiac arrest.
Cardiomyopathy: Diseases of the heart muscle can lead to heart dysfunction.
Arrhythmias: Abnormal heart rhythms can result in sudden cardiac arrest.
Congenital Heart Conditions: Some people are born with structural heart issues.
Electrocution or Drowning: Trauma that affects the heart's function can lead to cardiac arrest.
Drug Overdose: Certain drugs can significantly affect heart rhythm and function.
Severe Blood Loss or Lack of Oxygen: Conditions which compromise blood flow or oxygen supply can trigger arrest.

SYMPTOMS:

Chest discomfort.
Shortness of breath.
Weakness.
Sudden collapse.
No pulse.
No breathing.
Loss of consciousness.
Sudden Collapse: The most immediate sign is loss of consciousness or collapse.

No Pulse: The heart stops beating, resulting in the absence of a detectable pulse.
No Breathing: The individual will stop breathing or have abnormal breathing patterns.
Chest Pain: Some may experience chest discomfort prior to arrest.
Heart Palpitations: Some may feel a racing heart or irregular heartbeat before the event.

TREATMENT:
CPR (Cardiopulmonary Resuscitation): Immediate chest compressions can help circulate blood until help arrives.
Defibrillation: Using an automated external defibrillator (AED) can restore a normal heart rhythm.
Emergency Medical Services (EMS): Calling for immediate emergency assistance is critical.
Advanced Cardiac Life Support (ACLS): Once in a medical facility, advanced life support protocols will be followed.
Post-Resuscitation Care: Monitoring and care in a hospital setting is essential to prevent recurrence.

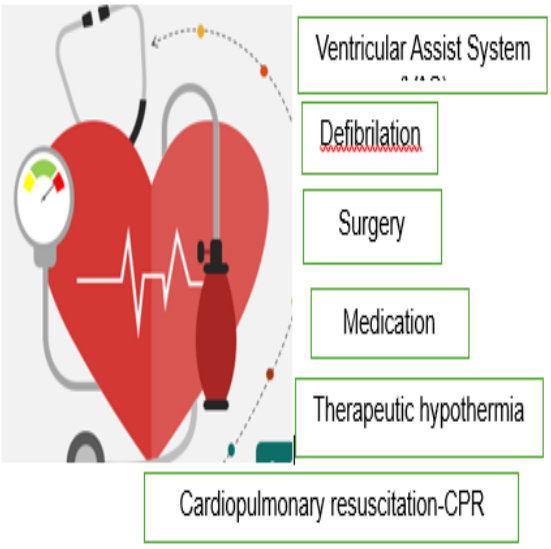

Fig 2 : Treatment for Cardiac Arrest:
Always remember that immediate action is crucial in case of cardiac arrest. Knowing how to perform CPR and use an AED can save lives.

The American Heart Association recommends doing CPR with hard and fast chest compressions if Cardiac arrest is observed-

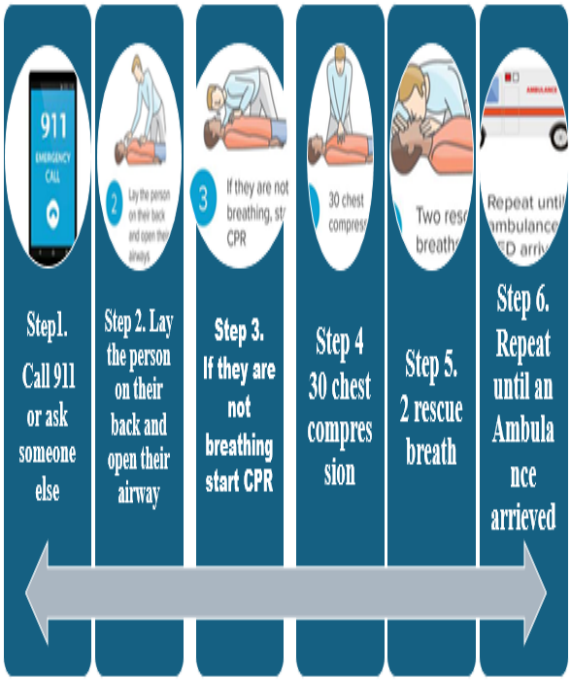

Fig 3 : Treatment for Cardiac Arrest:
Yoga and Cardiac Health:
While yoga cannot prevent or treat cardiac arrest directly, it can play a supportive role in overall heart health and cardiovascular wellness. Here's how yoga can benefit heart health and promote a balanced lifestyle:
Benefits of Yoga for Cardiovascular Health:
Stress Reduction: Yoga techniques, especially those focusing on breath control (pranayama), can help reduce stress and anxiety, which are known risk factors for heart disease.
Improved Circulation: Yoga can enhance blood flow and circulation throughout the body, supporting cardiovascular health.
Lower Blood Pressure: Regular practice of yoga may contribute to lowering high blood pressure, helping to mitigate one of the risk factors for heart disease.
Heart Rate Regulation: Gentle yoga practices can help regulate heart rate and improve overall heart function.

Enhanced Flexibility and Strength: The physical postures in yoga can improve overall body strength, flexibility, and endurance, promoting an active lifestyle

Weight Management: Regular yoga practice can aid in weight management, reducing obesity-related risks for cardiovascular diseases.

Mindfulness and Relaxation: Yoga encourages mindfulness, helping individuals become more attuned to their bodies and emotions, which can lead to healthier lifestyle choices.

Specific Yoga Practices:

Gentle Asanas: Poses such as Supta Baddha Konasana (Reclined Bound Angle Pose) and Viparita Karani (Legs-Up-the-Wall Pose) can promote relaxation and improve blood flow.

Breathing Exercises: Practices like Anulom Vilom (Alternate Nostril Breathing) and Ujjayi Breathing (Victorious Breath) aid in calming the nervous system and reducing stress.

Meditation and Mindfulness: Engaging in meditation can foster emotional well-being, contributing to lower stress levels and improved heart health.

Restorative Yoga: These gentle, supported poses are designed to promote deep relaxation and can be very beneficial for those recovering from stress or cardiovascular concerns.

Important Considerations: -

Consult a Healthcare Provider

Before starting any yoga program, especially for individuals with a history of heart issues, it's important to consult with a healthcare provider. – Listen to Your Body: Practitioners are encouraged to pay attention to their bodies and avoid pushing themselves too hard, especially if they experience discomfort or fatigue. –Choose Appropriate Classes: opt for classes designed for beginners, seniors, or those with health concerns, focusing on gentle movement and relaxation. In conclusion, while yoga is not a substitute for medical treatment or lifestyle changes, incorporating yoga can be a beneficial component of a comprehensive approach to maintaining heart health and well-being.

Recent Research on Cardiac Arrest

Recent studies and advancements in the understanding of cardiac arrest focus on prevention, treatment, and recovery, helping to enhance outcomes for patients. Here are some key areas of research:

1. Predictive Models and Risk Assessment:

Genetic and Biomarker Research: New studies are exploring genetic markers and blood biomarkers to predict individual risk for cardiac arrest. These advances may allow for better screening and preventative measures in high-risk populations. Machine Learning Models: Some research utilizes machine learning algorithms to analyze medical data, leading to better prediction of cardiac events based on existing patient conditions.

2. Compression-Only CPR:

Recent guidelines emphasize the effectiveness of hands-only CPR performed by bystanders. Research continues to demonstrate that this method can lead to better survival rates. Video Training: Studies show that video-based training can improve bystander CPR rates, especially among individuals who are less confident in performing regular CPR.

CPR Techniques and Training:

3. Use of Defibrillators:

AED Accessibility: Research highlights the importance of widespread access to Automated External Defibrillators (AEDs) in public spaces. Studies indicate that increased availability significantly improves survival rates when cardiac arrest occurs outside medical facilities.

Smartphone Apps: New technologies, including smartphone apps, are being developed to alert nearby trained individuals when someone suffers a cardiac arrest, facilitating quicker response times.

4. Post-Cardiac Arrest Care:

Targeted Temperature Management (TTM): Ongoing studies investigate the timing and effectiveness of induced hypothermia following cardiac arrest, aiming to protect the brain and improve neurological outcomes. Recovery Protocols: Research is focused on optimizing post-resuscitation care, including monitoring and therapies for organ support, to improve overall patient outcomes.

5. Long-Term Outcomes:

Psychological Impact: Studies are increasingly examining the long-term psychological effects on survivors, including anxiety and PTSD. This research aims to develop supportive strategies and treatments for mental health post-arrest.

Quality of Life Assessments: Research is being conducted to evaluate the long-term quality of life for survivors of cardiac arrest, focusing on how to enhance recovery processes and lifestyle modifications after the event.

6. Public Awareness and Education:

Community Programs: Research supports the implementation of community awareness programs on cardiac arrest signs, CPR, and AED use, particularly in schools and workplaces, to improve public readiness in emergencies. These areas of research represent a growing interest in not only improving immediate responses to cardiac arrest but also enhancing long-term outcomes for survivors. Ongoing studies are essential to develop more effective treatments, optimize care protocols, and understand the broader implications of cardiac arrest on health and recovery.

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CPR: WHEN, WHY, HOW TO DO IT

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INTRODUCTION:

Cardiac arrest or sudden cardiac death remains the single most common cause of death worldwide. Roughly around 300,000 patients have cardiac arrest every year in US alone. In India, it is estimated that around 7 lakh deaths occur due to cardiac arrest annually with 85% of them occurring outside hospital settings.1,2 Only one out of every ten such patients reach the hospital, while the others die at the scene or before reaching the hospital. With proper first aid, on-site resuscitation, and timely referral with continued care, the mortality rate due to sudden cardiac arrest can be reduced by up to 60%. Two protocols have been established for CPR: Basic life support (BLS) and Advanced cardiac life support (ACLS)The goal of treatment for cardiac arrest is to rapidly achieve return of spontaneous circulation using a variety of interventions including CPR, defibrillation, and/or cardiac pacing. Two protocols have been established for CPR: basic life support (BLS) and advanced cardiac life support (ACLS).

CARDIOPULMONARY RESUSCITATION:

Cardiopulmonary resuscitation (CPR) is a series of life-saving actions that improve the chances of survival, following cardiac arrest. Successful resuscitation, following cardiac arrest, requires an integrated set of coordinated actions represented by the links in the Chain of Survival. The links include the following: immediate recognition of cardiac arrest and activation of the emergency response system, early CPR with an emphasis on chest compressions, rapid defibrillation, effective advanced life support, and integrated post-cardiac arrest care.

The American Heart Association uses the letters C-A-B to help people remember the order to perform the steps of CPR.

C: Compressions.
A: Airway.
B: Breathing

Fig 1: CPR Steps

Perform the following basic CPR steps:
Call 911 or ask someone else to.
Lay the person on their back and open their airway.
Check for breathing. If they are not breathing, start CPR.
Perform 30 chest compressions.
Perform two rescue breaths.
Repeat until an ambulance or automated external defibrillator (AED) arrives.

CPR step-by-step: There are two main stages to CPR:
The preparation stage
The CPR stage.

Preparation steps:
Step 1. Call 911
First, check the scene for factors that could put you in danger, such as traffic, fire, or falling masonry. Next, check the person. Do they need help? Tap their shoulder and shout, “Are you OK?” If they are not responding, call 911 or ask a bystander to call 911 before performing CPR. If possible, ask a bystander to go and search for an

AED machine. People can find these in offices and many other public buildings.

Step 2. Place the person on their back and open their airway Place the person carefully on their back and kneel beside their chest. Tilt their head back slightly by lifting their chin. Open their mouth and check for any obstruction, such as food or vomit. Remove any obstruction if it is loose. If it is not loose, trying to grasp it may push it farther into the airway.

Step 3. Check for breathing Place your ear next the person's mouth and listen for no more than 10 seconds. If you do not hear breathing, or you only hear occasional gasps, begin CPR. If someone is unconscious but still breathing, do not perform CPR. Instead, if they do not seem to have a spinal injury, place them in the recovery position. Keep monitoring their breathing and perform CPR if they stop breathing.

CPR steps- Use the following steps to perform

Step 4. Perform 30 chest compressions

Place one of your hands on top of the other and clasp them together. With the heel of the hands and straight elbows, push hard and fast in the center of the chest, slightly below the nipples.

Push at least 2 inches deep. Compress their chest at a rate of least 100 times per minute. Let the chest rise fully between compressions.

Step 5. Perform two rescue breaths

Making sure their mouth is clear, tilt their head back slightly and lift their chin. Pinch their nose shut, place your mouth fully over theirs, and blow to make their chest rise. If their chest does not rise with the first breath, retile their head. If their chest still does not rise with a second breath, the person might be choking.

Step 6. Repeat

Repeat the cycle of 30 chest compressions and two rescue breaths until the person starts breathing or help arrives. If an AED arrives, carry on performing CPR until the machine is set up and ready to use.

SUMMARY:

Cardiopulmonary resuscitation (CPR) is a primordial skill necessary for all medical, nursing, and allied Health care professionals alike. As medical science is constantly and rapidly evolving, continuous updating of the knowledge and skills of people working in this field is the need of the hour. This review is an effort to outline the physiological basis of resuscitation and present the updated guidelines with a focus on both In-hospital and out-of-hospital cardiac arrests and the challenges faced by the medical rescuers in our country.

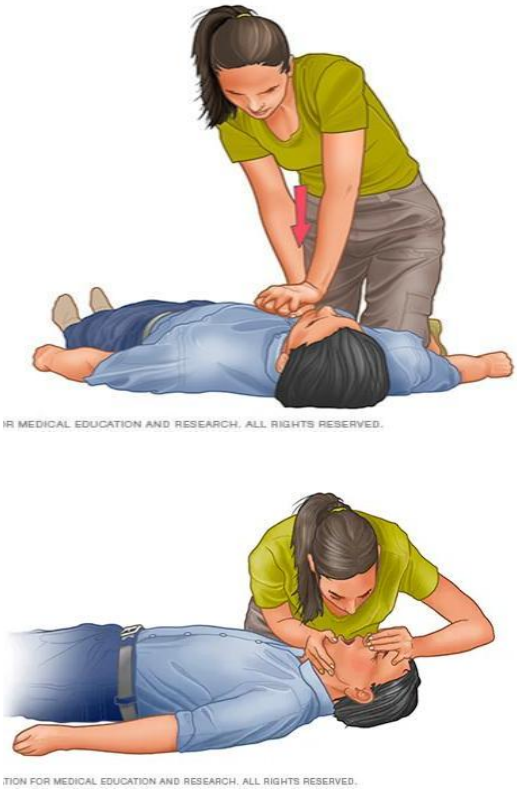

Fig 2: CPR Steps

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HYPERTENSION

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INTRODUCTION:

Hypertension, also referred to as high blood pressure or arterial hypertension, is a chronic medical condition characterized by persistently elevated blood pressure in the arteries. Blood pressure is typically measured by two values: systolic and diastolic pressures, representing the maximum and minimum pressures in the arterial system, respectively. Systolic blood pressure indicates the highest level reached when the heart contracts, pushing blood through the arteries.

Diastolic blood pressure signifies the lowest level reached when the heart relaxes between beats. Normal blood pressure at rest falls within the range of 100–140 mmHg systolic and 60–90 mmHg diastolic. World Hypertension Day is observed annually on May 17th to increase public awareness about hypertension. Statistics reveal that approximately three out of ten individuals worldwide suffer from hypertension.

There are two primary types of hypertensions and four less common types. The two primary types are:

Primary Hypertension: Individuals with essential hypertension typically do not exhibit symptoms, although they may occasionally experience headaches, fatigue, dizziness, or nosebleeds. Causes: Excessive physical activity

Secondary Hypertension: Around 5–10% of hypertension cases are secondary, stemming from identifiable causes such as:

Narrowing of the aorta or kidney arteries
Kidney damage or dysfunction
Adrenal gland tumors or hyperactivity
Thyroid disorders
Pregnancy-related conditions
Sleep apnea syndrome
Certain medications, recreational drugs, beverages, or foods

The other types of hypertensions include:

Malignant Hypertension: This type affects approximately 1% of people with hypertension, occurring more frequently in younger adults, African American men, and women with pregnancy-induced hypertension. Malignant hypertension occurs when blood pressure spikes rapidly.

Isolated Systolic Hypertension: In this condition, systolic blood pressure consistently exceeds 160 mm Hg while diastolic remains below 90 mm Hg. This may occur more commonly in older individuals. Causes: Reduced elasticity in the arteries, influenced by Western lifestyle and diet.

Resistant Hypertension: When blood pressure remains above 140/90 mmHg despite treatment with multiple medications, it's classified as resistant hypertension. This is more prevalent in older individuals, those who are overweight, women, African Americans, or those with underlying health issues such as diabetes or kidney disease.

White Coat Hypertension: Also known as anxiety-induced hypertension, this condition involves elevated blood pressure readings only when measured by healthcare professionals. If confirmed through repeat measurements outside clinical settings or via 24-hour monitoring, treatment may not be necessary. However, regular monitoring is advised to detect any potential development of persistent hypertension.

SYMPTOMS: Prolonged high blood pressure can lead to various complications, including aneurysms, kidney failure, eye damage, heart attack, heart failure, stroke, and peripheral arterial disease. Common symptoms may include severe headaches, fatigue, confusion, and blood in the urine.

How is hypertension diagnosed?

Your healthcare provider will take your blood pressure on several visits. You may also need to check your blood pressure at home. The provider will examine you and ask about medicines you take. He or she will also ask if you have a family history of high blood pressure and about any health conditions you have. He or she will also check your blood pressure and weight and examine your heart, lungs, and eyes. You may need any of the following tests:

An ambulatory blood pressure monitor (ABPM) is a device that you wear. ABPM measures your blood pressure while you do your regular daily activities. It records your blood pressure every 15 to 30 minutes during the day. It also records your blood pressure every 15 minutes to 1 hour at night. The recorded blood pressures help your healthcare provider know if you have hypertension not seen at your appointment.

Blood tests may help healthcare providers find the cause of your hypertension. Blood tests can also help find other health problems caused by hypertension.

Urine tests will be done to check your kidney function. Kidney problems can increase your risk for hypertension.

TREATMENT:

High blood pressure can be treated with various medications, each belonging to different classes:

β Blockers: These medications help reduce blood pressure by blocking the effects of adrenaline on the heart and blood vessels. By doing so, they decrease the heart rate and the force with which the heart pumps blood, leading to lower blood pressure.

Examples: Propranolol (Inderal®), Metoprolol (Betaloc®), Labetalol (Trandate®)

Dosage: Varies depending on the drug, typically taken one to two times daily.

ACE Inhibitors:

ACE inhibitors work by blocking the action of an enzyme called angiotensin-converting enzyme (ACE). This enzyme plays a role in narrowing blood vessels, so by blocking it, ACE inhibitors help relax blood vessels, making it easier for blood to flow through them, thus lowering blood pressure.

Examples: Ramipril (Altace®), Enalapril (Vasotec®)

Dosage: Ranges from 1.25 mg to 50 mg daily.

Calcium Antagonists: Calcium antagonists, also known as calcium channel blockers, prevent calcium from entering the muscle cells of the heart and blood vessels. This action relaxes and widens the blood vessels, reducing the pressure within them and lowering blood pressure.

Examples: Diltiazem (Cardizem®), Amlodipine (Norvasc®)

Dosage: Typically taken once or twice daily, dosage varies.

Thiazide diuretics: Thiazide diuretics help lower blood pressure by increasing the removal of sodium and water from the body through urine. By reducing the volume of fluid in the bloodstream, they decrease the pressure on the blood vessel walls, resulting in lower blood pressure.

Examples: Hydrochlorothiazide (Aquazide H), Chlorthalidone (Hygroton)

Dosage: Typically, 25-50 mg daily.

Direct Vasodilators: Direct vasodilators work by directly relaxing the muscles in the walls of blood vessels, causing them to widen. This dilation reduces the resistance to blood flow and lowers blood pressure.

Examples: Hydralazine (Apresoline®), Minoxidil (Loniten®)

Dosage: Dosage varies, usually taken once daily.

Central and Peripheral Sympatholytic: These drugs work by blocking or reducing the activity of the sympathetic nervous system, which is responsible for regulating various bodily functions, including blood pressure. By inhibiting sympathetic activity, these medications help

decrease heart rate and relax blood vessels, leading to lower blood pressure.

Examples: Reserpine (Serpasil®), Methyldopa (Aldomet®)

Dosage: Varies, taken once daily.

Alpha Blockers: Alpha blockers work by blocking receptors in the body called alpha-adrenergic receptors. By doing so, they prevent certain hormones from constricting blood vessels, allowing them to relax and widen, which ultimately lowers blood pressure.

Examples: Prazosin (Minipress®), Terazosin (Hytrin®)

Dosage: Usually taken once daily, dosage varies.

These medications work in different ways to help lower blood pressure and are often prescribed based on individual patient needs and response to treatment. It's essential to take them as prescribed by your healthcare provider and follow up regularly to monitor their effectiveness and any potential side effects.

SIDE EFFECTS:

Common side effects of antihypertensive drugs can vary depending on the specific medication, but here are some general side effects associated with these drugs:

Fatigue or tiredness
Dizziness or lightheadedness, especially when standing up quickly
Headache
Nausea or vomiting
Dry cough (common with ACE inhibitors)
Erectile dysfunction (in men)
Increased urination or frequent urination
Muscle weakness or cramps
Rash or skin reactions
Changes in taste perception
Swelling in the ankles or legs
Changes in electrolyte levels (such as potassium or sodium)
Increased blood sugar levels
Constipation or diarrhea
Difficulty sleeping or insomnia

It's important to note that not everyone will experience these side effects, and some individuals may experience different side effects depending on their individual response to the medication. Always consult a healthcare professional if you experience any concerning side effects while taking antihypertensive medication.

CONTRAINDICATIONS:

Antihypertensive drugs are **contraindicated** in certain conditions or situations. Some common contraindications include:

1. Allergy or hypersensitivity to the specific medication or its components.
2. Pregnancy, especially for certain classes of antihypertensive drugs like ACE inhibitors and ARBs, which can harm the fetus.
3. Severe kidney disease, as some antihypertensive drugs may further impair kidney function.
4. Severe liver disease, as these medications may be metabolized by the liver and could worsen liver function.
5. Hypotension (low blood pressure), as antihypertensive drugs can further lower blood pressure and lead to complications such as dizziness, fainting, or organ damage due to inadequate blood flow.
6. Heart conditions such as bradycardia (slow heartbeat), heart block, or certain types of heart failure, where the use of certain antihypertensive drugs may exacerbate these conditions.
7. Hyperkalemia (high levels of potassium in the blood), as some antihypertensive medications like ACE inhibitors and ARBs can increase potassium levels further.
8. Certain drug interactions, where antihypertensive drugs may interact with other medications a person is taking, leading to adverse effects or reduced efficacy.

It's essential for individuals to discuss their medical history and any current medications with

their healthcare provider before starting antihypertensive treatment to ensure it is safe and appropriate for them.

MANAGEMENT:

What can I do to manage hypertension?

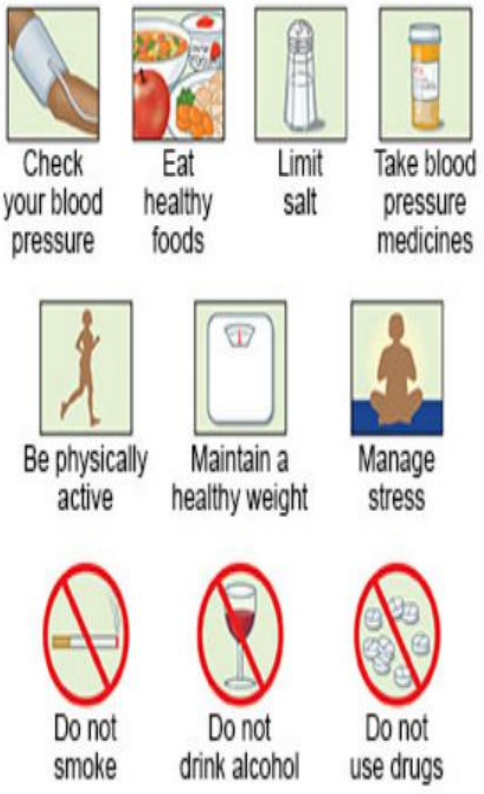
Check your blood pressure at home. Do not smoke, have caffeine, or exercise for at least 30 minutes before you check your blood pressure.

Manage any other health conditions you have. Health conditions such as diabetes can increase your risk for hypertension. Follow your healthcare provider's instructions and take all your medicines as directed.

Ask about all medicines. Certain medicines can increase your blood pressure. Examples include oral birth control pills, decongestants, herbal supplements, and NSAIDs, such as ibuprofen. Your healthcare provider can tell you which medicines are safe for you to take. This includes prescription and over-the-counter medicines.

What lifestyle changes can I make to manage hypertension?

1. Limit sodium (salt) as directed. Too much sodium can affect your fluid balance. Check labels to find low-sodium or no-salt-added foods. Some low-sodium foods use potassium salts for flavor. Too much potassium can also cause health problems. Your healthcare provider will tell you how much sodium and potassium are safe for you to have in a day. He or she may recommend that you limit sodium to 2,300 mg a day.
2. Follow the meal plan recommended by your healthcare provider. A dietitian or your provider can give you more information on low-sodium plans or the DASH (Dietary Approaches to Stop Hypertension) eating plan. The DASH plan is low in sodium, processed sugar, unhealthy fats, and total fat. It is high in potassium, calcium, and fiber. These can be found in vegetables, fruit, and whole-grain foods.
3. Be physically active throughout the day. Physical activity, such as exercise, can help control your blood pressure and your weight. Be physically active for at least 30 minutes per day, on most days of the week. Include aerobic activity, such as walking or riding a bicycle. Also include strength training at least 2 times each week. Your healthcare providers can help you create a physical activity plan.
4. Decrease stress. This may help lower your blood pressure. Learn ways to relax, such as deep breathing or listening to music.
5. Limit alcohol as directed. Alcohol can increase your blood pressure.
6. Do not smoke. Nicotine and other chemicals in cigarettes and cigars can increase your blood pressure and also cause lung damage. Ask your healthcare provider for information if you currently smoke and need help to quit.



When should I seek immediate care or call my doctor?

1. You have a severe headache or vision loss.
2. You have weakness in your arm or leg.
3. You feel faint, dizzy, confused, or drowsy.
4. You have been taking your blood pressure medicine, but your pressure is higher than your provider says it should be.
5. You have questions or concerns about your condition or care.

You can only take control of your life if your BP is in control."

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DECODING THE COMPLEXITIES OF HEART FAILURE: BRIDGING PATHOPHYSIOLOGY, RISK, AND COMPREHENSIVE CARE

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ABSTRACT:
Heart failure (HF) remains a major global health challenge with significant morbidity and mortality. This article delves into the pathophysiology, etiology, risk factors, management, and non-pharmacological treatment strategies for HF. It emphasizes the distinction between heart failure with reduced ejection fraction (HFrEF) and preserved ejection fraction (HFpEF) by elucidating their respective pathophysiological mechanisms. Etiologies such as ischemic heart disease, hypertension, cardiomyopathies, and valvular abnormalities are discussed alongside modifiable and non-modifiable risk factors. Management approaches encompass pharmacological therapies, device-based interventions, and lifestyle modifications, highlighting recent advancements in treatment modalities. The integration of patient education, cardiac rehabilitation, and psychosocial support underscores the importance of a holistic approach in HF care. This comprehensive review provides insights into current evidence-based practices to improve patient outcomes.

INTRODUCTION:
Heart failure (HF) is a complex syndrome where the heart fails to pump blood adequately, impacting the body's metabolic needs. It is the end-stage manifestation of various cardiovascular diseases and a leading cause of morbidity and mortality worldwide, affecting approximately 64 million people. The prevalence of HF is rising due to aging populations and improved survival rates from other cardiovascular conditions. In the U.S., HF contributes to over 1 million hospitalizations annually, placing a significant burden on healthcare. Despite advances in treatment, HF prognosis remains poor, with a five-year mortality rate of around 50% for both HFrEF and HFpEF patients. Understanding HF's pathophysiology, classification, and management is key to improving outcomes. Recent developments, including SGLT2 inhibitors and device-based therapies, offer new hope, but challenges such as high rehospitalization rates and limited treatments for HFpEF underscore the need for ongoing research and innovation. This review aims to provide an updated overview of HF, including its pathophysiology, classification, clinical presentation, and management strategies.

EPIDEMIOLOGY:
Heart failure (HF) affects approximately 64 million people globally, with prevalence increasing due to aging populations and rising rates of risk factors like hypertension, coronary artery disease, obesity, and diabetes (Crespo-Leiro et al., 2018). The incidence varies by region, with developed countries experiencing higher rates due to better diagnosis and longer life expectancies, while developing nations see different causes, such as rheumatic heart disease (Sliwa et al., 2016). HF is categorized into two main types: heart failure with reduced ejection fraction (HFrEF) and heart failure with preserved ejection fraction (HFpEF), the latter being more common in older adults and women (Yancy et al., 2017). Despite advances in treatment, heart failure remains a leading cause of hospitalization and mortality, with 5-year survival rates ranging from 35% to 60% (Lloyd-Jones et al., 2010). The growing global burden of HF is expected to continue, highlighting the need for improved prevention and management strategies (Ambrosy et al., 2014).

ETIOLOGY:

Heart failure (HF) can result from a variety of causes and is often associated with specific risk factors that predispose individuals to the development of the condition. Understanding these etiologies and risk factors is essential for prevention and management.

Ischemic Heart Disease: The most common cause of HF, resulting from reduced blood supply to the myocardium due to coronary artery disease (Ponikowski et al., 2016).

Hypertension: Chronic high blood pressure increases left ventricular afterload, leading to hypertrophy and eventual dysfunction

Cardiomyopathies: Dilated cardiomyopathy: Idiopathic or secondary to toxins, infections, or genetic factors

Hypertrophic cardiomyopathy: Often genetic and associated with left ventricular hypertrophy

Restrictive cardiomyopathy: Often due to infiltrative diseases like amyloidosis

Valvular Heart Disease Arrhythmias: Both tachyarrhythmia and Bradyarrhythmia impair cardiac output

Congenital Heart Disease

Endocrine Disorders

PATHOPHYSIOLOGY:

Heart failure (HF) arises from a complex interplay of structural, functional, and molecular mechanisms that impair the heart's ability to pump blood effectively. It is broadly categorized into two primary types based on ejection fraction: heart failure with reduced ejection fraction (HFrEF) and heart failure with preserved ejection fraction (HFpEF). While these categories differ in underlying mechanisms, both lead to systemic congestion, reduced perfusion, and progressive cardiac remodeling.

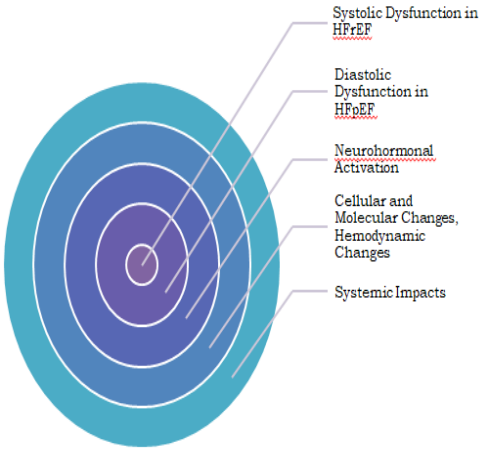

Fig 1 Pathophysiology of Heart Failure

1. Systolic Dysfunction in HFrEF

HFrEF is primarily characterized by impaired myocardial contractility, resulting in a reduced ejection fraction (<40%). The key mechanisms include:

Myocardial injury: Conditions such as myocardial infarction lead to loss of functional myocardium, reducing contractile force (Ponikowski et al., 2016).

Ventricular remodeling: Neurohormonal activation drives hypertrophy and fibrosis, altering the structure and function of the left ventricle.

Energy deficits: Mitochondrial dysfunction reduces ATP availability, impairing contraction (Zhou et al., 2020).

2. Diastolic Dysfunction in HFpEF

HFpEF is characterized by impaired ventricular relaxation and increased stiffness, with a preserved ejection fraction (≥50%). Pathophysiological contributors include:

Endothelial dysfunction: Impaired nitric oxide signaling reduces myocardial compliance (Paulus et al., 2007).

Fibrosis: Excessive collagen deposition in the myocardium increases stiffness.

Systemic inflammation: Chronic inflammatory states contribute to microvascular dysfunction and myocardial injury.

3. Neurohormonal Activation

Neurohormonal systems play a central role in the progression of HF:

Renin-Angiotensin-Aldosterone System (RAAS): Increased activity promotes sodium retention, vasoconstriction, and myocardial fibrosis (McDonagh et al., 2021).

Sympathetic Nervous System (SNS): Chronic SNS activation leads to tachycardia, increased afterload, and adverse ventricular remodeling.

Natriuretic Peptides: Although compensatory, chronic elevation of natriuretic peptides may fail to counterbalance the detrimental effects of RAAS and SNS.

4. Cellular and Molecular Changes

Hypertrophy and apoptosis.

Oxidative stress

Calcium handling abnormalities

5. Hemodynamic Changes

Increased preload and afterload.

Elevated filling pressures

6. Systemic Impacts

HF affects multiple organ systems, resulting in:

Kidney dysfunction: Reduced renal perfusion leads to fluid retention and worsens congestion.

Skeletal muscle wasting: Reduced perfusion and systemic inflammation contribute to muscle atrophy.

Cognitive impairment: Chronic hypo perfusion of the brain affects cognitive function.

Understanding these pathophysiological processes is critical for developing targeted therapies to address the underlying causes and mitigate the progression of HF.

SIGN AND SYMPTOMS:

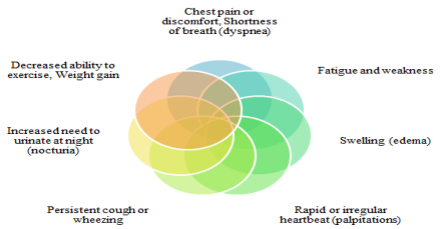

Fig 2:Sign and Symptoms of Heart Failure

DIAGNOSIS:

The diagnosis of heart failure typically involves a combination of clinical evaluation, laboratory tests, and imaging studies:

Medical History and Physical Examination

Blood Tests:B-type natriuretic peptide (BNP) or N-terminal pro BNP (NT-proBNP)

Electrocardiogram (ECG):

Chest X-ray:

Echocardiogram (Ultrasound of the heart):

Cardiac MRI:

Electrocardiogram (ECG):

Stress Testing:

Cardiac Catheterization or Coronary Angiography

TREATMENT:

1. Pharmacological Management

HFrEF-Specific Therapies:

ACE inhibitors or ARBs: Reduce afterload and prevent remodeling

ARNIs (e.g., sacubitril/valsartan): Improve outcomes compared to ACE inhibitors

Beta-blockers: Reduce myocardial oxygen demand and improve survival

Aldosterone antagonists: Prevent fibrosis and sodium retention

SGLT2 inhibitors: Recent evidence shows efficacy in reducing HF hospitalizations

Diuretics: Provide symptom relief by reducing fluid overload (Ponikowski et al., 2016).

HFpEF Management:

Limited targeted therapies, focusing on controlling comorbidities like hypertension and diabetes (Paulus & Tschöpe, 2007).

2. Device-Based Management

Implantable cardioverter-defibrillators (ICDs): Prevent sudden cardiac death in HFrEF

Cardiac resynchronization therapy (CRT): Improves cardiac efficiency in patients with ventricular dyssynchrony

Ventricular assist devices (VADs): Support patients awaiting heart transplantation or as destination therapy (Ponikowski et al., 2016).

3 Non-Pharmacological Treatment of Heart Failure

Non-pharmacological strategies are crucial for comprehensive HF management and focus on lifestyle modifications, education, and self-care practices.

Lifestyle Modifications

Dietary Adjustments: Sodium restriction: Helps manage fluid retention (Ponikowski et al., 2016).

Balanced diet: Focus on heart-healthy foods rich in fruits, vegetables, and whole grains

Physical Activity: Tailored exercise programs improve functional capacity and quality of life (McDonagh et al., 2021).

Avoid strenuous activities that exacerbate symptoms.

Smoking Cessation: Reduces cardiovascular risk (Ponikowski et al., 2016).

Weight Management

Education and Self-Monitoring

Psychosocial Support

Cardiac Rehabilitation

Herbal remedies

Arjuna (Terminalia arjuna): Traditionally used for heart health, it may support heart muscle function and reduce symptoms of heart failure.

Ashwagandha (Withania somnifera): Known for its adaptogenic properties, it can help reduce stress and improve energy levels.

Guggulu (Commiphora wightii): A resin used to reduce cholesterol and improve circulation, potentially benefiting those with heart conditions.

Brahmi (Bacopa monnieri): Supports cognitive function and may improve circulation and reduce anxiety, contributing to better overall cardiovascular health.

Recent target of drug action

Recent advancements in heart failure (HF) treatment have introduced several novel drug targets aimed at improving patient outcomes. Key areas of focus include:

Myeloperoxidase (MPO) Inhibition: MPO is an enzyme linked to inflammation and oxidative stress in the heart. Inhibiting MPO has shown promise in reducing biomarkers associated with HF and improving clinical outcomes.

Sodium-Glucose Cotransporter Inhibitors (SGLTis): SGLTis, such as sotagliflozin, target both SGLT1 and SGLT2 to reduce glucose absorption and improve heart function. Sotagliflozin has been approved for reducing cardiovascular death and hospitalization in HF patients.

Gene Silencing Therapies: Innovative treatments like vutrisiran utilize RNA interference to silence genes producing harmful proteins, offering potential benefits for conditions like transthyretin amyloid cardiomyopathy, a cause of HF.

GLP-1 Receptor Agonists: Drugs such as semaglutide (Wegovy) have been endorsed for treating HF with preserved ejection fraction (HFpEF) in obese individuals, demonstrating significant weight loss and improvement in heart failure-related health criteria.

Adrenomedullin Agonists: Targeting adrenomedullin, a peptide involved in vasodilation and fluid balance, has been identified as a potential strategy to protect against new-onset HF.

CONCLUSION:

In conclusion, heart failure remains a major global health challenge with increasing prevalence and significant morbidity and mortality. The pathophysiology of heart failure is complex, involving a combination of hemodynamic, molecular, and cellular factors, which contribute to the progression of the disease. Advances in the understanding of these mechanisms have led to the development of novel therapeutic strategies aimed at improving outcomes for patients. While current treatments such as ACE inhibitors, beta-blockers, and diuretics have significantly improved survival and quality of life, emerging therapies targeting

specific molecular pathways, including SGLT inhibitors, gene silencing, and GLP-1 receptor agonists, hold great promise in providing more effective and personalized treatments. However, challenges remain in the early diagnosis, management of comorbidities, and addressing disparities in access to care. Continued research into the molecular and genetic underpinnings of heart failure, coupled with innovations in pharmacotherapy and personalized medicine, will be essential to improving patient outcomes and reducing the burden of this disease in the future. **"Together, We Beat the Odds: Conquering Heart Failure One Step at a Time"**

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ISCHEMIC CARDIOMYOPATHY

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INTRODUCTION:

Ischemic cardiomyopathy is a condition in which the heart muscle is injured and is unable to effectively pump blood. This damage is typically brought on by coronary artery disease, which prevents enough oxygen-rich blood from reaching your heart muscle. Surgery and medication are two forms of treatment. The degree to which oxygen deprivation damages your cardiac muscles determines your prospects. [1].

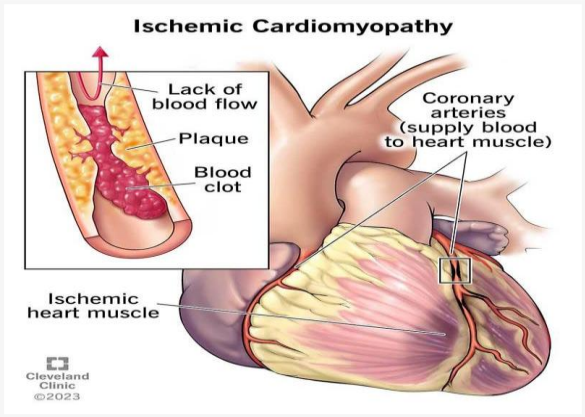
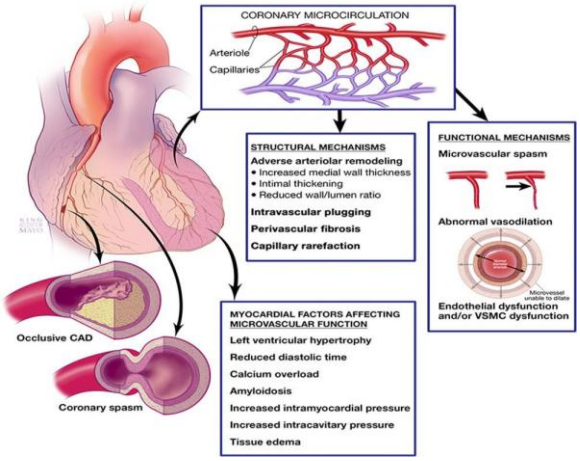


Fig 1: Lack of blood supply to heart muscle causes damage or Ischemic Cardiomyopathy) [1]

What is ischemic cardiomyopathy?

Ischemic cardiomyopathy is a condition in which the heart muscle is damaged by a lack of blood flow, making it unable to pump blood efficiently. This loss of blood flow (ischemia) is brought on by heart attacks and coronary artery disease.In patients with ischemic cardiomyopathy, this blood shortage causes the left ventricle (one of the chambers of the heart) to weaken and expand. A weak left ventricle reduces the heart's capacity to pump blood because it is the primary pumping chamber. [2][3].



Role of coronary microvascular dysfunction in the pathogenesis of ischemic heart disease. The leading cause of systolic heart failure worldwide and the most common form of dilated cardiomyopathy is ischemic cardiomyopathy. It is the cause of 60% of cases.

SYMPTOMS AND CAUSES:

Ischemic cardiomyopathy symptoms may include:
Breathlessness.
Fatigue that prevents you from exercising or performing daily tasks.
Angina (a symptom that is less common).
Coughing, congestion, and weight gain associated with fluid retention.
Arrhythmias, or irregular heartbeats, can produce palpitations or fluttering in the chest.
Feeling light headed or dizziness.
Fainting
Ischemic cardiomyopathy symptoms may not be present in certain individuals. [4] [5].

CAUSES:

Ischemic cardiomyopathy can be caused by a number of disorders that impair heart function by reducing blood flow, including:
Coronary artery disease. (This is the most common

cause.)
Fibromuscular dysplasia.
Coronary microvascular disease.
Coronary artery dissection.
Coronary vasculitis.
Heart attack.

RISK FACTORS:

Significant heart disease risk factors may boost your chance of ischemic cardiomyopathy and cardiovascular (heart and blood vessel) disease. These risk factors consist of: [5] [6].
Family history of heart disease.
Experiencing hypertension.
Use of tobacco products.
Being diabetic.
Having elevated cholesterol.
Being over 30 on the body mass index (BMI) scale.
Previously suffered a heart attack.
Lack of physical activity.

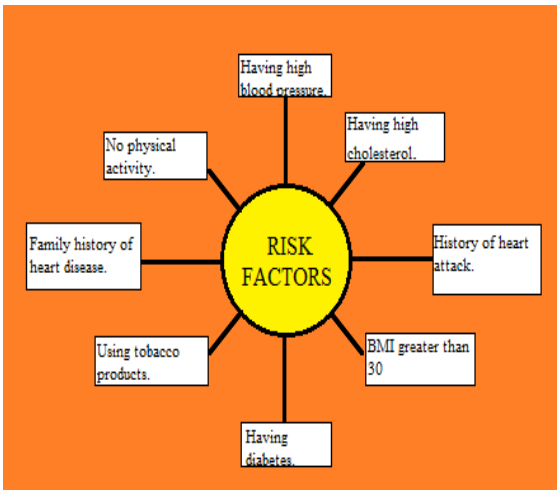


Fig 2: Risk factor

COMPLICATIONS:

Complications of ischemic cardiomyopathy are as follows: [7].
Arrhythmias.
Congestive heart failure.
Pulmonary edema.
Mitral valve regurgitation.
Blood clots.
Cardiac arrest.
Sudden cardiac death.

DIAGNOSIS AND TESTS:

The diagnosis of ischemic cardiomyopathy is based on:
Your medical history.
Your symptoms.
Family's medical history.
A physical exam. [4] [8].
Tests done to diagnose ischemic cardiomyopathy are:
Blood tests.
Electrocardiogram (ECG).
Chest X-ray.
Transthoracic echocardiogram (TTE).
Exercise stress test.
Cardiac catheterization.
CT scan.
MRI scan.
Nuclear medicine imaging.
To identify the cause of cardiomyopathy, a doctor may occasionally conduct a cardiac biopsy. [8] [9]

MANAGEMENT AND TREATMENT:

For ischemic cardiomyopathy treatment, Physician focus on:
Treating coronary artery disease if any.
Improving your cardiac function.
Reducing heart failure symptoms.
The following are possible therapies to lessen symptoms and enhance quality of life:
Medications.
Lifestyle changes.
Implantable devices.
Interventional procedures and surgeries

MEDICATIONS:

Medications that you may take includes:
Beta-blockers.
ACE inhibitors.
ARBs.
ARNI.
Diuretics.
Aldosterone antagonists.

antiarrhythmic.
Anticoagulants (blood thinners).
Statins. [10] [11] [12]

This medication can help you with:

Improving your cardiac function.
Treat symptoms of heart failure.
Prevent further complications.
Prevent coronary artery disease progression.
Prevent heart attacks.

LIFESTYLE CHANGES:

You can alter your everyday schedule to enhance your well-being. Some of the changes are:
Limiting your **salt** intake to 2,000 to 3,000 mg/day.
Exercising.
Avoiding intake of tobacco products.
Following the Mediterranean Diet (plant-based foods, healthy fats, and whole grains).
Implantable Devices: Certain individuals require a device that can improve the function of their hearts.
Types of devices include:
Cardiac re-synchronization therapy.
Implantable cardioverter defibrillators (ICD). [13].
Sometimes you may receive both devices combined in a single unit based on the severity.

Interventional procedures and surgeries

Interventional procedures and surgeries include:
Angioplasty.
Stents.
Coronary artery bypass surgery.
Left ventricular volume reduction surgery.
Left ventricular assist devices.
Heart transplant.
Other heart failure surgical options.

COMPLICATIONS/SIDE EFFECTS OF THE TREATMENT:

Side effects and complications vary by treatment:

Medications related:

Dizziness.
Upset stomach.
Fatigue.
Dry cough.
Headache.
Low blood pressure.
Hair loss.
Bleeding. [10] [12]

Implantable devices related:

Bleeding.
Infection.
Device malfunction.
Collapsed lung.
Cardiac tamponade.

Procedures and surgeries related:

Chest pain.
Bleeding.
Infection.
Heart attack.
Blood clots.
Irregular heart rhythms.
Stroke.
Rejection of a transplanted heart.

What is the duration of recovery after this treatment?

The full effects of a medicine may not be felt for a few weeks. Other treatments can take anywhere from a few days to weeks or months to recover from.

PREVENTION:

The prevention of coronary artery disease can help you avoid ischemic cardiomyopathy. This is ischemic cardiomyopathy's most frequent cause. [14]
You can lower your risk of ischemic cardiomyopathy by:
Avoiding consumption of tobacco products.
Exercising at least for 30 minutes five times a week.
Getting sufficient amount of sleep.
maintaining healthy weight.
Managing your daily stress.
Managing diabetes and high blood pressure.
Eating healthy foods, such as whole grains, green vegetables and fruits.
Limiting consumption of alcohol.
Avoiding smoking.

OUTLOOK / PROGNOSIS:

If I have ischemic cardiomyopathy, what can I expect?

The degree of damage to your heart muscle may vary depending on your circumstances. Medications helps to reduce symptoms and improve the function of heart. However, your medical professional will suggest procedures or devices if these are insufficient.

Can we reverse ischemic cardiomyopathy?

If the loss of blood supply is just temporary, you might be able to reverse ischemic cardiomyopathy. However, you cannot undo the damage if your heart muscle is deprived of blood and oxygen for an extended period of time. This can occur when you suffer a heart attack.

Prognosis for ischemic cardiomyopathy

Your prognosis is significantly influenced by the degree of damage to your heart muscle. But, therapy can help you. When discussing your outlook, a doctor will take into account a number of factors, including:

How much blood left ventricle can pump i.e. ejection fraction on.
How severe is your coronary artery disease is?
Whether you've had a heart attack. [14]

How do I take care of myself while suffering with ischemic cardiomyopathy?

You can make changes in your everyday life that improve your health. Such as: [15]
maintain healthy weight.
Limit the consumption of alcohol.
Get enough sleep every night.
Exercise regularly.
limit the salt intake and have a healthy diet.
Avoid tobacco products.
Manage your stress level.

When should I consult my doctor?

To keep an eye on your condition, you will need to see your doctor a few times a year. Regular appointments help them see:
How your medicines are working?
How your device is working, if you have one?
How you're recovering from an operation.

What questions should I ask my doctor?

How much heart muscle damage has been done?
What's the best treatment for me?
Is there a support group that could help me?

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MYOCARDIAL INFARCTION

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ABSTRACT:
One of the world's major causes of illness and mortality is still myocardial infarction (MI). The epidemiology and etiology of MI are covered first in this review, which then delves further into its pathophysiology to reveal its complex character. Effective treatment requires an understanding of the intricate mechanisms behind ischemia, reperfusion injury, and cellular damage. Current diagnostic techniques, such as imaging and biomarkers, are covered together with the clinical signs and symptoms of MI. Traditional pharmacological treatments including thrombolytics, β-blockers, and antiplatelet therapy are highlighted, while emerging therapeutic targets like inflammatory pathways and Na+/H+ exchanger inhibitors are investigated for their potential to improve patient outcomes. Non-pharmacological methods such as rehabilitation, lifestyle changes, and new Ayurvedic therapies are also taken into account. The review also explores new pharmacological targets, focusing on genetic and regenerative treatments that lessen ischemia damage and encourage tissue healing. With an emphasis on both present and future directions for enhancing patient care and survival, this thorough review seeks to present a comprehensive picture of myocardial infarction.

INTRODUCTION:

A decrease in or disruption of the blood flow to a portion of the myocardium causes heart attacks, also known as myocardial infarctions (MI). There are two types of myocardial infarctions: "silent" and undetected, and catastrophic, leading to hemodynamic deterioration and untimely death (1). The majority of myocardial infarctions and the leading cause of death in the US are caused by coronary artery disease. The myocardium cannot receive oxygen when the coronary arteries are clogged. Long-term disruption of the heart's oxygen supply can cause myocardial cell death and necrosis. It is possible for patients to arrive with chest pressure or discomfort that radiates to the arm, shoulder, jaw, or neck. The disorder known as coronary heart disease, or myocardial infarction (MI), commonly called a heart attack, damages the blood vessels that nourish the heart muscle (myocardium). The entire process is called a myocardial infarction, and the part of the heart muscle that is either infracted is the part that has so little or no flow that it is unable to sustain cardiac muscle action. MI symptoms include discomfort in the chest radiating from the left arm to the neck, dyspnea, sweating, nausea, vomiting, irregular heartbeat, anxiety, fatigue, weakness, stress, and depression (2).

EPIDEMIOLOGY:

Conventional studies of the epidemiology of myocardial infarction have focused on infarction and have seldom addressed the physiological entity of acute coronary syndromes, with or without biomarker increase. Several factors contribute to this, including the need for a standardized epidemiologic definition and the relative ease of standardizing the definition of myocardial infarction compared to the more challenging

task of defining acute coronary syndromes from an epidemiologic point of view, particularly in forms with transient or absent electrocardiographic changes and without biomarker elevation (3). Over the years, epidemiologic research has failed to consider a substantial amount of the burden of nonfatal cardiac disease, namely acute coronary syndromes, that do not meet recognized infarction criteria. The definition of myocardial infarction has highlighted this important problem, making it difficult for epidemiologists to incorporate acute coronary syndromes into the surveillance of cardiovascular disease. Depending on one or more biomarkers alters the classification of various acute coronary syndromes, according to research evaluating the impact of redefining myocardial infarctions (4).

ETIOLOGY:

As previously mentioned, coronary artery disease and myocardial infarction are intimately related. The global multi-center case-control study INTERHEART revealed the following modifiable risk factors for coronary artery disease:

The act of smoking.

Blood apolipoprotein and abnormal lipid profile (higher Apolipoprotein B (ApoB)/Apolipoprotein A-I (ApoA1).

Elevated blood pressure.

Diabetic retinopathy.

Abdominal obesity, or the waist-to-hip ratio (more than 0.85 for women and higher than 0.90 for males).

Desperation, a lack of control, external stress, financial stress, and life events such as divorce, losing one's job, and family conflicts are examples of psychosocial components.

Eating insufficient amounts of fruits and vegetables every day.

Insufficient exercise.

Drinking alcohol (protective, weaker connection).

The interheart research showed that all of the risk factors stated above were substantially associated with acute myocardial infarction, with the exception of alcohol usage, which showed a weaker association (5). Acute myocardial infarction was most strongly associated with smoking and aberrant a lipoprotein ratio. Women were found to have a higher risk of diabetes and hypertension, as well as a stronger protective impact from alcohol and exercise. Among the other risk variables is a markedly high level of plasma homocysteine, which is an independent risk factor for MI. Vitamins B6, B12, and folic acid are available as treatments for increased plasma homocysteine (6). Non-modifiable risk factors for myocardial infarction (MI) include advanced age, heredity, and male gender (males are more likely to experience myocardial infarction earlier in life). The risk of MI is increased if a first-degree relative has a history of cardiovascular events before to the age of fifty. The role of genetic loci that increase the risk of MI is still being investigated (7).

PATHOPHYSIOLOGY:

Acute blockage of one or more large epicardial coronary arteries for more than 20 to 40 minutes might cause an acute myocardial infarction. Usually thrombotic, the blockage results from the rupture of a plaque that has developed in the coronary arteries. The blockage causes the myocardium to become oxygen-depleted, which causes myofibril relaxation and sarcolemmal disruption. These alterations are among of the first ultrastructural alterations that take place after MI, and changes in the mitochondria follow. Ultimately, the prolonged ischemia results in the

necrotization and liquefaction of heart tissue. The sub-endocardium to the sub-epicardium is affected by the necrosis. It is thought that the sub epicardium has a higher collateral circulation, which postpones death. In The area of the heart that is impacted by the infarction determines how impaired the heart is. Heart remodeling, including dilatation, segmental hypertrophy of the remaining surviving tissue, and cardiac failure, is common because of the myocardium's extremely restricted capacity to repair, resulting in scarring in the infarcted area (8).

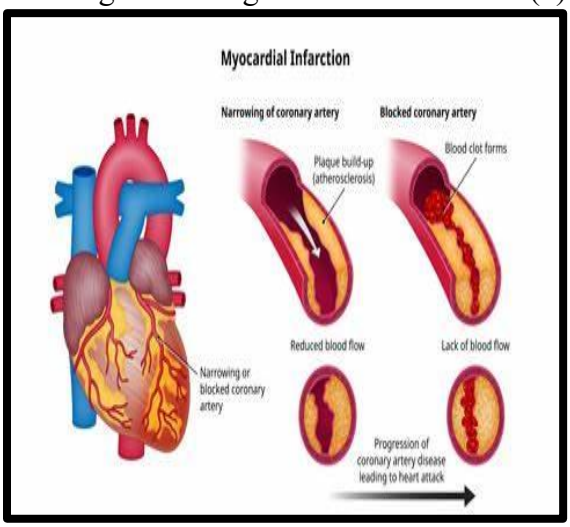

Fig1. Pathophysiology of MI

SIGNS & SYMPTOMS OF MYOCARDIAL INFARCTION:

Angina without any warning indications and chest discomfort are the typical markers of a heart attack. A "silent heart attack" is a minor heart attack that occasionally manifests no symptoms.

1. Chest pain or discomfort that is triggered by exercise and goes away with rest.

A little chest ache that persists even after resting.

Abrupt, intense, smothering chest pain that might spread to other bodily regions.

A sensation of fullness, tightness, burning, squeezing, pressure, or other discomfort in the jaw, arm, neck, shoulder, or chest.

A strange ache that travels down one or both arms.

Indigestion, nausea, and vomiting.

Weakness, pallor, sweating, shortness of breath, or unusual exhaustion.

Difficulty performing tasks that were formerly simple.

Extreme dread, denial, or worry (9).

DIAGNOSTICS OF MYOCARDIAL INFARCTION:

Patients with myocardial infarction or heart attacks are frequently treated in hospital emergency rooms under the Coronary Care Unit and Management. The patient's medical history, physical examination, and blood pressure are determined by the hospital. To find any irregularities in the heart's blood flow, an ECG or EKG is used to track the heart's beats or rhythms. A blood test is also performed to measure the quantity of lipids and proteins in the blood that might damage the heart muscles. Coronary angiography, also referred to as an X-ray of the heart and blood vessels, is used to monitor the narrowing of the coronary arteries. A tiny catheter is inserted into the arm or leg's coronary arteries. After a contrast agent is injected into the coronary arteries from the catheter's end, X-rays are taken to check for cardiac issues (10).

TREATMENT:

Patients with prolonged ST-segment elevation and ischemic symptoms lasting less than 12 hours should get reperfusion treatment. If Percutaneous Coronary Intervention (PCI) can be done within 120 minutes of diagnosis, it’s preferred over fibrinolysis. If PCI isn’t possible within 120 minutes, fibrinolysis should start within 10

minutes. If reperfusion is successful, PCI can be scheduled 60-90 minutes after fibrinolysis. Fibrin-specific drugs like tenecteplase, alteplase, or reteplase should be used for fibrinolysis.

Pain, Dyspnea, and Anxiety Relief: Chest pain from myocardial infarction causes stress on the heart. Morphine (Class IIa) is often used for pain relief, but the study suggests it may increase risks, further study found no significant issues with morphine in anterior ST-segment elevation Myocardial Infarction (MI). Benzodiazepines can help anxious patients.

Nitrates: When it comes to alleviating symptoms and lowering ST depression, intravenous nitrates work better than sublingual nitrates. Until symptoms subside or adverse effects like headaches or low blood pressure appear, the dosage is raised.

Beta-blockers: These medications lower blood pressure, heart rate, and the oxygen requirement of the heart.They block the effects of stress hormones on the heart. They shouldn't be used in cases of suspected coronary vasospasm.

Platelet Suppression: For both STEMI and NSTEMI, 150–300 mg of aspirin should be taken initially, then 75–100 mg per day.It helps prevent blood clotting by blocking thromboxane A2.

Statins: Statins help lower death rates in acute coronary syndrome survivors (12).

NON PHARMACOLOGICAL TREATMENT:

The goal of non-pharmacological therapy for myocardial infarction is to improve cardiovascular health and support recovery through lifestyle changes and complementary therapies. These strategies are vital due to the limitations and potential side effects of medications. Key approaches include:

Lifestyle Modifications: Adopting healthy habits, such as a proper diet, regular exercise, and quitting smoking, is essential for primary and secondary prevention of heart disease. Non-compliance with these recommendations significantly increases the risk of adverse cardiac events, as shown in the OASIS-5 study.

Smoking Cessation: Quitting smoking is critical, as it significantly reduces the risk of recurrent cardiac events. Patients who quit smoking after a heart attack see their risk approach that of non-smokers within three years.

Exercise and Physical Activity: Regular exercise improves heart function, increases oxygen supply, and reduces cardiovascular mortality. It also helps manage risk factors by enhancing coronary vasodilation and strengthening heart health.

Nutrition and Weight Management: A Mediterranean-style diet reduces the risk of recurrent heart attacks for up to four years, and weight control through calorie restriction lowers cardiovascular risk, especially in individuals with metabolic syndrome.

Psychological Support: Counseling and therapy can help manage anxiety and depression, which often occur after a myocardial infarction and can hinder recovery.

Stress Management: Practices like mindfulness, meditation, and relaxation techniques reduce stress and support emotional well-being.

Alternative Therapies:

Traditional Chinese Medicine: Techniques such as acupuncture, massage, and qigong may improve quality of life and reduce anxiety post-heart attack.

Cardiac Rehabilitation Programs: These comprehensive programs, involving experts like social workers, psychologists, and nutritionists, offer personalized plans to support recovery and long-term health

.

AYURVEDIC APPROACHES TO MYOCARDIAL INFARCTION TREATMENT:

Herbal treatments, lifestyle changes, and detoxification therapies are all part of Ayurveda's holistic approach to treating myocardial infarction. One of the most important herbs for heart health is Terminalia arjuna, which is believed to increase circulation and strengthen heart muscles. Additionally, important are anti-inflammatory herbs like Curcuma longa (turmeric) and stress-relieving adaptogens such Withania somnifera (ashwagandha). Yoga techniques like Pranayama and panchakarma treatments like Hridaya Basti aid in heart rehabilitation by increasing oxygenation and encouraging calm. These integrative methods are consistent with contemporary preventative measures and could be used in addition to traditional medical care as supplemental therapies.

Hridaya Nava Rasa: First For the treatment of angina, chest pain, and breathing issues, it is the finest option. It works well for Kaphaja Hridroga and is beneficial for atherosclerosis, coronary artery disease, and hyperlipidemia because it is Vata-Kaphahara in nature.

Arjuna Ksheera Paaka: Arjuna (Terminalia arjuna) possesses Ruksha Guna, Laghu, Kashaya Rasa, Srothoshodhana, and Kapprashamana Karma (PV. 2001). It works well for ischemia, angina, and atherosclerosis and has hypolipidemic, cardioprotective, and antioxidant properties.

Parthadyarishta: This Ayurvedic formulation is a potent cardioprotective with anti-ischemic, hypolipidemic, antiatherogenic, antianginal, antihyperglycemic, and antioxidant properties. It balances all three doshas (Tridoshahara) and supports the heart, lungs, and overall vitality (Ojovardhaka).

Prabhakara Vati: Known for its strong cardioprotective and antianginal effects, it enhances cardiac function and addresses various heart conditions, including coronary artery disease and congestive heart failure. It works irrespective of dosha dominance and possesses anti-arrhythmic and inotropic (force-enhancing) properties (17).

EMERGING THERAPIES FOR MYOCARDIAL INFARCTION: It focus on innovative approaches to minimize heart damage, enhance repair, and improve long-term outcomes.

Genetic Therapy: This approach involves introducing recombinant DNA into cardiac tissue to treat or prevent myocardial infarction. Both DNA and viral vectors have demonstrated effective delivery to healthy and ischemic heart tissues.

Heat Shock Protein Genes: Heat shock proteins, such as HSP70, are activated during stress or increased temperature, offering myocardial protection. They mitigate ischemia-induced cellular changes like proton and sodium ion accumulation, free radical generation, and calcium overload during reperfusion.

Antiapoptotic Therapies: Various pathways contribute to cell death during ischemia-reperfusion injury, including TNF-α receptor activation, Fas signaling, and p53 and c-Jun kinase pathways. Significant roles are also played by immune cell infiltration, elevated pro-apoptotic Bax, and reduced anti-apoptotic Bcl-2.

Mitochondrial-mediated apoptosis is prominent during reperfusion, highlighting the need for targeted antiapoptotic interventions.

Na+/H+ Exchanger Inhibitors: The Na+/H+ exchanger regulates intracellular pH but excessive activation leads to sodium and calcium overload, causing ischemia-reperfusion injury. Inhibiting Na+/H+ exchangers have shown significant

myocardial protection in preclinical models, reducing energy loss and tissue damage.

Potassium ATP Channels: Potassium ATP (KATP) channels link cardiac metabolism to membrane electrical activity and serve as a natural defense against ischemic damage. KATP channel openers have demonstrated myocardial protection in various models, although their effect is blocked by antagonists like 5-hydroxy decanoate and glibenclamide.

Oxidative Stress and Antioxidants: Nutrients such as N-acetyl cysteine and pantothenic acid enhance glutathione levels, reducing oxidative stress. Antioxidants like curcumin, α-lipoic acid, α-tocopherol, and ascorbic acid have shown protective effects in animal models of myocardial infarction.

CONCLUSION:

In recent decades, myocardial infarction has emerged as a major global cause of death and morbidity. MI is a multi-faceted illness since several pathways have been investigated and found to be involved in its onset and progression. It is ideal to use an interprofessional team to diagnose and treat patients with ischemic heart disease. The majority of hospitals have cardiology teams specifically tasked with managing these patients. Myocardial infarction has been a leading cause of death and morbidity worldwide in recent decades. Since multiple pathways have been studied and found to be involved in the onset and progression of MI, it is a complex illness. When diagnosing and treating patients with ischemic heart disease, an interprofessional team is the best option. These patients are managed by cardiology teams that are particularly assigned to most hospitals.

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MITRAL VALVE PROLAPSE: UNDERSTANDING CAUSES, SYMPTOMS, AND TREATMENTS

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1. Introduction

Mitral valve prolapses (MVP) occurs when the heart's mitral valve doesn't work well. The mitral valve is located between the left atrium and the left ventricle. It acts like a one-way door; it should allow blood to flow from the left atrium into the left ventricle and then prevent it from flowing backward. MVP affects about 1 in 20 Americans, making it one of the most common forms of heart disease. In MVP, the valve's two flaps can become stretchy and cannot close evenly or smoothly. It is rare for infants to be born with MVP. Instead, the disorder develops in children and adults. In most people, MVP is relatively harmless and does not require any treatment. But in a small minority of patients, MVP can have serious complications or lead to bothersome symptoms. Not long ago, it was widely believed that MVP was overdiagnosed and overtreated. Now experts believe the opposite may be true—that it is underdiagnosed and undertreated. Mitral valve prolapses (MVP) was first described as a distinctive syndrome in the mid-1960s, but the condition has been known since the time of Leenhardt and described as "dysplasia myxoedematous" with reference to marked dilatation of the shelves of the mitral valve. Barlow and colleagues were the first to describe it as a distinct clinical entity in 1963, in a series of elderly women. Mitral valve prolapse refers to an abnormal late systolic movement of the mitral valve leaflets "beyond" the (mitral) annular plane or "billowing" of the mitral valve leaflets. The definition of MVP widely used is the echocardiographic identification of one or more leaflets extending at least 2 mm into the left atrium in the parasternal long-axis view or in apical views or the identification of late systolic displacement of the mitral leaflets extending at least 2 mm beyond the long-axis diameter of the left atrium in apical views.

The scale is also composed of the thickness of the leaflets, which can be normal, thin, or thick, and the appearance of the mitral valve apparatus on movement, which can be normal, flail, or floppy and enlarged. The normal scale includes moderate mitral leaflet motion, usually with a small amount of systolic leaflet curling. The prolapsed scale consists of a combination of late systolic leaflet displacement (≥ 2 mm beyond the annular plane), leaflet thickening (at least two of the mid-portions of the mitral leaflets are > 5 mm in thickness when measured in a parasternal long-axis view), and mitral annular dilatation (defined as MD > 2 SD by age and body surface area) when compared to normal values. Flail leaflet with severe prolapse is relatively rare, present in around 7% of the MVP cases, and is associated with leaflet rupture and severe regurgitation.

Mitral Valve Prolapse (MVP) is a condition where the mitral valve leaflets (the flaps of tissue between the left atrium and left ventricle) become abnormally thickened or elongated, causing them to bulge (prolapse) backward into the left atrium during systole (the phase of the heartbeat when the heart contracts).

PATHOPHYSIOLOGY:

Valve Abnormalities:

Myxomatous Degeneration: In MVP, there is often myxomatous degeneration, where the connective tissue in the valve becomes abnormal. This leads to a softening of the valve leaflets and

elongation of the chordae tendineae (the fibrous cords that anchor the valve leaflets to the papillary muscles in the ventricles). This change reduces the structural integrity of the valve, making it more prone to prolapsing during ventricular contraction.

Thickening and Stretching of Leaflets: The valve leaflets become thickened and stretched, causing them to bulge back into the left atrium. This happens during the contraction of the left ventricle, especially when the pressure in the ventricle increases during systole.

Mitral Regurgitation: The prolapse of the mitral valve may lead to incomplete closure of the valve, causing mitral regurgitation (MR). In MR, blood flows backward from the left ventricle into the left atrium. This can further lead to volume overload in the left atrium, left ventricle, and eventually, pulmonary circulation. In mild cases, MR may be asymptomatic, but in more severe cases, it can lead to symptoms such as shortness of breath, fatigue, or heart failure.

Hemodynamic Changes:

Left Atrial Enlargement: The regurgitation of blood into the left atrium increases the volume of blood in the left atrium, which can lead to left atrial enlargement. This can increase the risk of atrial arrhythmias such as atrial fibrillation.

Left Ventricular Volume Overload: Chronic volume overload in the left ventricle due to regurgitation may eventually lead to left ventricular dilation, which may impair ventricular function.

Increased Risk of Endocarditis: The abnormal valve structure and the turbulent blood flow in the atrium may increase the risk of bacterial endocarditis.

Genetic and Structural Factors:MVP is often associated with genetic conditions, including Marfan syndrome, Ehlers-Danlos syndrome, and other connective tissue disorders, which affect the structural integrity of the valve and other connective tissues in the body.There may be an inherited component in some families with MVP, suggesting a genetic predisposition to abnormal collagen metabolism or structural defects of the mitral valve.

Autonomic Dysfunction: Many individuals with MVP also have an increased sympathetic nervous system activity or autonomic dysfunction, leading to symptoms like palpitations, dizziness, and anxiety. The mechanisms for this association are not fully understood but may relate to abnormal cardiovascular regulation.

CLINICAL MANIFESTATIONS:

Symptoms: Many individuals with MVP are asymptomatic or have mild symptoms. However, those with significant mitral regurgitation or arrhythmias may experience symptoms like palpitations, chest pain (non-cardiac), dyspnea (shortness of breath), and fatigue.

Complications: Severe cases of MVP with significant mitral regurgitation can lead to heart failure, atrial fibrillation, stroke (due to clot formation in the atrium), or endocarditis. In summary, the pathophysiology of mitral valve prolapse is primarily related to structural changes in the mitral valve, such as myxomatous degeneration, elongation of the chordae tendinous, and valve leaflet thickening, which result in abnormal valve function, leading to mitral regurgitation and hemodynamic consequences. The condition can range from mild to severe, and in some cases, it can be associated with other systemic issues, such as connective tissue disorders.

Clinical Presentation and Symptoms of Mitral Valve Prolapse (MVP)- Mitral Valve Prolapse (MVP) can present in a variety of ways, from asymptomatic cases to those with significant symptoms or complications. The severity of MVP can vary based on the degree of mitral

regurgitation, the presence of arrhythmias, and the associated structural changes to the mitral valve.

Asymptomatic vs Symptomatic Patients

Asymptomatic Patients:

Prevalence: The majority of individuals with MVP are asymptomatic, especially in cases of mild mitral regurgitation or when the valve prolapse is not severe.

Incidental Finding: Often, MVP is detected incidentally during a routine physical exam through a murmur (such as a mid-systolic click) or during an echocardiogram performed for another reason. It is frequently diagnosed in healthy individuals without any overt signs or symptoms of heart disease.

Prognosis: Asymptomatic patients with mild MVP generally have a favorable prognosis, with a low risk of developing complications like heart failure, endocarditis, or arrhythmias, though they may need regular monitoring.

Symptomatic Patients:

Prevalence: Symptomatic MVP occurs in a smaller subset of patients. Symptoms may be related to the severity of mitral regurgitation, arrhythmias, or structural changes in the valve.

Types of Symptoms: These patients may experience a variety of symptoms, ranging from mild to severe, affecting their daily activities and quality of life. Symptoms can often be non-specific and may mimic other conditions.

Common Symptoms and Complications

Common Symptoms:

Palpitations: Palpitations are one of the most common symptoms in symptomatic MVP patients. They often occur due to arrhythmias, such as premature ventricular contractions (PVCs) **or** atrial fibrillation (AF**)**. These arrhythmias are more common in individuals with significant mitral regurgitation or structural changes in the mitral valve.

Chest Pain: Some individuals with MVP may report non-cardiac chest pain. This pain is often sharp or stabbing and unrelated to physical exertion. It may occur in episodes and is usually not associated with ischemic heart disease. The exact cause is unclear but may be related to abnormal autonomic nervous system activity or changes in the mitral valve.

Dyspnoea (Shortness of Breath): Dyspnoea can occur due to mitral regurgitation**,** leading to pulmonary congestion. When blood is pushed backward into the left atrium and lungs, it can cause fluid accumulation in the lungs, leading to shortness of breath, especially during exertion or when lying flat (orthopnea).

Fatigue: Fatigue is a common symptom in MVP, especially in patients with more severe mitral regurgitation or left ventricular dysfunction. This may be due to the decreased efficiency of the heart as it pumps blood, leading to less oxygen delivery to the body.

Dizziness or Light-headedness: Some MVP patients experience dizziness or light-headedness, especially when standing up quickly (orthostatic hypotension) or during episodes of arrhythmias like ventricular tachycardia **or** atrial fibrillation**.**

Anxiety: Anxiety and panic attacks are more frequently reported in MVP patients, possibly due to autonomic dysfunction **(**an imbalance between the sympathetic and parasympathetic nervous systems). This can lead to episodes of heightened heart rate or palpitations, which may exacerbate feelings of anxiety.

Syncope: In rare cases, individuals with severe MVP may experience fainting or syncope**,** especially in the context of significant arrhythmias (e.g., ventricular arrhythmias or atrial fibrillation) or if there is significant mitral regurgitation leading to a drop in cardiac output.

COMPLICATIONS:

Mitral Regurgitation:

Chronic Mitral Regurgitation is one of the most significant complications of MVP. Over time, the mitral valve prolapse can worsen, causing the

valve to not close completely, allowing blood to flow backward into the left atrium.

Symptoms of mitral regurgitation can include fatigue**,** shortness of breath**,** and edema (swelling), especially in the lower extremities. Severe regurgitation may lead to heart failure if left untreated.

Arrhythmias: MVP is associated with an increased risk of arrhythmias, particularly atrial fibrillation (AF), which can result in palpitations and an increased risk of stroke due to clot formation in the atrium. In some cases, ventricular arrhythmias may also occur, leading to syncope or sudden cardiac death, though this is rare.

Endocarditis: Infective endocarditis is a potential complication of MVP, particularly in individuals with significant mitral regurgitation or those with a history of valve damage. Bacteria can enter the bloodstream and infect the mitral valve, leading to fever, fatigue, and potentially serious complications like valve destruction or embolic events (e.g., stroke).

Stroke: Atrial fibrillation (AF) associated with MVP increases the risk of embolism, leading to a stroke. Blood clots may form in the left atrium due to stasis from regurgitation, and these clots can travel to the brain, causing ischemic strokes.

Heart Failure: Severe mitral regurgitation, when left untreated, can lead to left-sided heart failure due to volume overload and increased work for the left ventricle. This can cause fluid build-up in the lungs and other organs, resulting in dyspnoea, fatigue**,** edema, and reduced exercise tolerance.

Pulmonary Hypertension: Chronic mitral regurgitation and left atrial enlargement can lead to pulmonary hypertension (high blood pressure in the lungs), further exacerbating shortness of breath and fatigue. Over time, this can strain the right ventricle, potentially leading to right-sided heart failure.

Diagnostic Modalities for Mitral Valve Prolapse (MVP) The diagnosis of Mitral Valve Prolapse (MVP) typically involves imaging studies and electrocardiographic (ECG) testing. Several diagnostic modalities help assess the presence of MVP, the severity of mitral regurgitation, and the risk of complications such as arrhythmias or heart failure. Below are the key diagnostic modalities used to diagnose MVP:

Echocardiography and ECG

Echocardiography is the primary diagnostic tool for MVP, allowing clinicians to visualize the structure and function of the heart, including the mitral valve.

Transthoracic Echocardiography (TTE):

Standard Imaging Tool: TTE is a non-invasive and commonly used method to diagnose MVP. It uses sound waves to create images of the heart's chambers, valves, and blood flow.

Valve Structure and Motion: TTE can identify the characteristic **prolapse** of the mitral valve leaflets into the left atrium during systole. It also helps evaluate the **severity** of **mitral regurgitation** (MR) by assessing the degree of backward blood flow from the left ventricle into the left atrium.

Quantifying Regurgitation: Doppler ultrasound, an integral part of TTE, is used to visualize blood flow and measure the **jet of regurgitation**, which helps assess the severity of mitral regurgitation (mild, moderate, severe). Severe regurgitation is linked to a higher risk of heart failure.

Left Atrial Size and Function: TTE can also assess the left atrium and left ventricle for signs of enlargement due to chronic regurgitation, which is a complication of MVP.

Transesophageal Echocardiography (TEE):

More Detailed Imaging: In some cases, especially if the TTE is inconclusive or if a more detailed view of the mitral valve is needed, a **TEE** may be performed. This involves placing a probe in the esophagus to get closer images of the heart and mitral valve. It is particularly useful for

visualizing the posterior mitral valve leaflet and assessing the severity of regurgitation more accurately.

Indications: TEE is often used in cases of suspected infective endocarditis or when the structure of the valve and mitral regurgitation is difficult to assess through TTE.

Electrocardiogram (ECG):An ECG is often used in the evaluation of MVP, especially to assess for any associated arrhythmias or electrical abnormalities that can be a complication of the condition.

Arrhythmias: MVP patients are at higher risk of arrhythmias, including atrial fibrillation **(**AF**)**, ventricular arrhythmias, and premature ventricular contractions (PVCs**)**. ECG can help identify these arrhythmias, which may be responsible for symptoms like palpitations or dizziness.

QRS Complex and P-Wave Abnormalities: The ECG in MVP may show early repolarization or increased P-wave duration, and prolonged PR interval, which may be associated with changes in left atrial size and function. However, the ECG findings in MVP are often nonspecific.

Cardiac MRI and CT Scans

Cardiac Magnetic Resonance Imaging (MRI): Cardiac MRI is a highly detailed and advanced imaging modality used to assess the structure and function of the heart. It is especially useful when TTE or TEE does not provide enough information or when more precise measurements are required.

Evaluation of Mitral Valve Anatomy: Cardiac MRI can provide high-resolution images of the mitral valve, helping assess the leaflet morphology**,** thickness**,** and chordal elongation. It can also evaluate the degree of prolapse and the mitral valve's ability to close properly during systole.

Quantification of Mitral Regurgitation: MRI can assess **regurgitant volume** and **fraction** by measuring the volume of blood that regurgitates from the left ventricle into the left atrium. This information is critical for evaluating the severity of mitral regurgitation, which influences treatment decisions (e.g., surgical intervention for severe MR).

Left Ventricular Function and Remodelling: MRI provides an accurate assessment of left ventricular size**,** ejection fraction**,** and dilatation, which may be important in determining the long-term impact of MVP, especially in patients with chronic severe regurgitation.

Potential for 3D Imaging: In some cases, cardiac MRI can create three-dimensional images of the heart, providing a more comprehensive view of the mitral valve, its structure, and function.

Cardiac CT Scans: Cardiac CT is another advanced imaging tool that can be used to assess the mitral valve and the surrounding structures.

Assessment of Mitral Valve Anatomy: Although not the first-line imaging method for MVP, cardiac CT can provide detailed anatomical images of the mitral valve, including valve morphology, the presence of any calcifications, or structural abnormalities. It can also identify left atrial enlargement**.**

Evaluation of Coronary Artery Disease (CAD): Cardiac CT is useful in the assessment of coronary artery disease **(**CAD**)**, especially in patients with chest pain. In MVP patients, CAD can be an additional concern, particularly if the patient is older or has risk factors for coronary artery disease.

3D Imaging: Like MRI, **cardiac CT** can provide three-dimensional images of the heart, which can be helpful in planning potential surgical interventions or in assessing complex cases of MVP.

Limitations:

Cardiac MRI and **cardiac CT** are more expensive and less widely available than echocardiography. Additionally, they often require patients to be stable and cooperative (e.g., holding their breath during imaging), and for

MRI, patients with implanted medical devices (e.g., pacemakers) may not be eligible.

Radiation Exposure: While MRI does not involve radiation, **cardiac CT** does use ionizing radiation, which is a consideration, especially in younger patients.

CLASSIFICATION OF MITRAL VALVE PROLAPSE:

Primary (or Idiopathic) Mitral Valve Prolapse: This is the most common type of MVP, where the valve leaflets are abnormally thickened or redundant. It can be due to congenital factors, where the valve's structure is inherently abnormal, leading to its prolapse. Common in individuals with connective tissue disorders like Marfan syndrome, Ehlers-Danlos syndrome, and Osteogenesis imperfecta.

Secondary Mitral Valve Prolapse: This type occurs as a result of another underlying condition, such as:

Ischemic heart disease (heart attacks that damage the mitral valve)

Dilated cardiomyopathy (heart muscle weakness)

Left ventricular dysfunction (reduced function of the left ventricle)

Rheumatic heart disease, though this is less common today due to antibiotics preventing rheumatic fever.

Grading of Mitral Valve Prolapse: Grading of MVP is generally based on the severity of the prolapse and the degree of mitral regurgitation (backward flow of blood into the left atrium), which can be evaluated using echocardiography (especially transthoracic echocardiogram and Transesophageal echocardiogram).

1. **Mild MVP (Grade I):**

The prolapse of the mitral valve is minimal, with only slight bulging of the leaflets into the left atrium. No significant mitral regurgitation or only trivial regurgitation. Often asymptomatic, and no treatment may be required.

2. **Moderate MVP (Grade II):**

There is a more noticeable bulging of the mitral valve leaflets into the left atrium. There may be mild-to-moderate mitral regurgitation, but the function of the heart is generally preserved. Some individuals may experience mild symptoms like palpitations, chest discomfort, or fatigue.

3. **Severe MVP (Grade III):**

The mitral valve leaflets prolapse significantly into the left atrium, potentially leading to severe mitral regurgitation. This can cause a volume overload in the left atrium and ventricle, resulting in increased risk of heart failure, arrhythmias, and other complications. Symptoms may be more pronounced, such as dyspnea (shortness of breath), fatigue, and palpitations. Severe regurgitation may require surgical intervention; such as valve repair or replacement.

Key features to assess during grading include:

Leaflet thickness and redundancy: The more redundant or thickened the valve leaflets, the more likely the prolapse is to be severe.

Degree of prolapse: Measured by the distance the mitral leaflets move into the left atrium.

Degree of mitral regurgitation: Severity is classified from none to severe, which can be determined by Doppler echocardiography.

Associated Complications of MVP:

Mitral regurgitation: This occurs in varying degrees of severity and can eventually lead to heart failure if not managed properly.

Arrhythmias: Atrial and ventricular arrhythmias (such as atrial fibrillation) can be more common in those with severe MVP.

Infective endocarditis: Although rare, MVP with significant mitral regurgitation increases the risk of infective endocarditis.

Stroke: Individuals with MVP may be at a higher risk of stroke, particularly if there is mitral regurgitation and atrial fibrillation.

MANAGEMENT:

Most individuals with mild MVP do not require any intervention.Moderate MVP with mild

regurgitation can usually be managed with regular follow-up and symptom management.

Severe MVP with significant regurgitation may require surgical intervention to repair or replace the mitral valve. Echocardiography is a crucial tool in diagnosing MVP and assessing its severity. Monitoring symptoms and clinical changes is important for determining the appropriate treatment and follow-up for affected individuals.

Medical Management Strategies for Mitral Valve Prolapse (MVP):

Management of MVP focuses on alleviating symptoms, preventing complications, and improving the overall quality of life. The treatment plan often depends on the severity of MVP, the degree of mitral regurgitation, the presence of symptoms, and any associated arrhythmias or other complications.

Pharmacological Interventions

Pharmacological treatment is used to manage symptoms of MVP, mitigate risks such as arrhythmias, and control associated conditions such as mitral regurgitation or heart failure. Below are the key classes of medications used:

Beta-Blockers

Indications: Beta-blockers (e.g., propranolol, metoprolol) are commonly used in patients with MVP who experience palpitations, anxiety, or chest pain. They are effective in managing symptoms related to autonomic dysfunction, which may be present in MVP.

Mechanism of action: These medications slow down the heart rate and reduce the workload on the heart, helping alleviate symptoms like palpitations and anxiety that are often associated with MVP.

Common side effects: Fatigue, dizziness, bradycardia, and sexual dysfunction.

Antiarrhythmic Medications

Indications: Antiarrhythmic drugs (e.g., amiodarone, flecainide) may be prescribed if the patient develops arrhythmias, such as atrial fibrillation **or** ventricular arrhythmias, which can occur in patients with severe MVP.

Mechanism of action: These medications help regulate the heart's electrical activity to prevent irregular heartbeats. In patients with significant mitral regurgitation and left atrial dilation, arrhythmias like atrial fibrillation may become more common.

Common side effects: Fatigue, dizziness, visual disturbances (with amiodarone), and potential proarrhythmic effects.

Diuretics

Indications: Diuretics (e.g., furosemide, spironolactone) may be used in cases of heart failure with pulmonary congestion due to severe mitral regurgitation or other complications. They help reduce fluid retention and ease the burden on the heart.

Mechanism of action: These drugs reduce fluid volume in the body, decrease blood pressure, and relieve symptoms of fluid overload (e.g., shortness of breath, swelling).

Common side effects: Electrolyte imbalances, dehydration, dizziness, and hypotension.

ACE Inhibitors or ARBs (Angiotensin Receptor Blockers)

Indications: In cases of severe mitral regurgitation or heart failure due to MVP, ACE inhibitors (e.g., enalapril, lisinopril) or ARBs (e.g., losartan) may be prescribed. These medications help reduce the heart's workload by lowering blood pressure and preventing further damage to the heart.

Mechanism of action: They relax blood vessels, reduce afterload (resistance the heart must work against), and improve heart function. They may also help reduce the progression of heart failure.

Common side effects: Cough (with ACE inhibitors), hyperkalemia, dizziness, and hypotension.

Anticoagulants

Indications: If the patient with MVP develops atrial fibrillation or other risk factors for thromboembolism**,** anticoagulants (e.g., warfarin, direct oral anticoagulants like apixaban) may be

prescribed to reduce the risk of blood clots and stroke.

Mechanism of action: These medications prevent the formation of blood clots that can lead to stroke or other systemic emboli, particularly in the setting of atrial fibrillation.

Common side effects: Increased bleeding risk, bruising, and gastrointestinal upset.

Antidepressants or Anxiolytics

Indications: Some patients with MVP may also suffer from anxiety or panic attacks, often due to the symptoms of palpitations or chest discomfort. Medications like SSRIs (selective serotonin reuptake inhibitors) or benzodiazepines may be used to manage anxiety.

Mechanism of action: These medications help balance the chemicals in the brain that influence mood and anxiety, offering symptom relief.

Common side effects: Drowsiness, dizziness, gastrointestinal issues, and potential for dependency with long-term benzodiazepine use.

LIFESTYLE MODIFICATIONS:

In addition to pharmacological management, certain lifestyle modifications can help patients with MVP manage symptoms, improve heart health, and reduce the risk of complications.

Regular Exercise

Benefits: Moderate, regular aerobic exercise can improve cardiovascular health, help maintain an optimal weight, and alleviate symptoms of fatigue. It also reduces stress and improves overall well-being.

Recommendations: Activities such as walking, swimming, cycling, or light jogging are generally recommended. However, patients should avoid intense or high-impact activities that might exacerbate palpitations or other MVP-related symptoms.

Stress Reduction Techniques

Benefits: Since stress and anxiety can exacerbate MVP symptoms (such as palpitations and chest discomfort), adopting techniques to reduce stress is important.

Recommendations: Practices like yoga, meditation, deep breathing exercises, and progressive muscle relaxation can help lower stress levels and improve mental health. Cognitive-behavioral therapy (CBT) may also be helpful for patients experiencing significant anxiety or panic attacks related to MVP.

Diet Modifications

Heart-healthy diet: Adopting a balanced diet that is low in saturated fats, sodium, and processed foods can help reduce the risk of hypertension and heart failure, which can exacerbate symptoms of MVP. A diet rich in fruits, vegetables, whole grains, lean proteins, and healthy fats (e.g., omega-3 fatty acids) is ideal for cardiovascular health.

Sodium reduction: Limiting sodium intake can help reduce fluid retention and prevent increased strain on the heart in patients with severe mitral regurgitation.

Weight Management

Benefits: Maintaining a healthy weight can reduce the workload on the heart and lower the risk of hypertension**,** heart failure, and other cardiovascular issues. Being overweight can worsen symptoms like shortness of breath and fatigue in patients with severe MVP.

Recommendations: If necessary, working with a healthcare provider or nutritionist to develop a sustainable weight management plan can help alleviate symptoms and improve overall heart health.

Avoiding Stimulants

Caffeine and Other Stimulants: Some people with MVP are sensitive to caffeine, nicotine, and other stimulants, which can worsen palpitations and other symptoms. It is often recommended to limit or avoid these substances.

Medications or Substances: In some cases, patients should also avoid medications like decongestants or other substances that can stimulate the heart.

Regular Follow-up and Monitoring

Importance of check-ups: Regular follow-ups with a cardiologist are essential to monitor the progression of MVP, particularly for those with moderate-to-severe MVP or mitral regurgitation. This allows for timely adjustments to the management plan and detection of complications, such as arrhythmias or heart failure.

Surgical Intervention and Procedures for Mitral Valve Prolapse (MVP)

In patients with Mitral Valve Prolapse (MVP), surgery is typically considered when the condition leads to significant complications that cannot be effectively managed with medical therapy alone. Surgical intervention is most commonly required when there is severe mitral regurgitation, associated heart failure, or risk of life-threatening arrhythmias.

Indications for Surgery

Surgical intervention for MVP is generally recommended when certain clinical conditions or complications are present. These include:

Severe Mitral Regurgitation

When it occurs: Severe regurgitation often develops in MVP due to the malfunctioning valve, leading to backward flow of blood into the left atrium.

Indications for surgery: If severe mitral regurgitation causes symptoms like shortness of breath, fatigue, or heart failure, or if there is evidence of progressive dilation of the left atrium or left ventricle, surgical intervention is recommended.

Risk: Left untreated, severe regurgitation can lead to significant heart remodeling, worsening heart failure, and other complications, making early intervention essential.

Symptomatic MVP with Significant Mitral Regurgitation

When it occurs: If patients with MVP experience significant symptoms like fatigue, dyspnea (shortness of breath), palpitations, or chest pain, and medical management does not effectively control these symptoms, surgery may be indicated.

Indications for surgery: Persistent symptoms despite optimal medical therapy or deteriorating exercise capacity are strong indications for surgical correction.

Left Ventricular Dysfunction

When it occurs: If severe mitral regurgitation from MVP leads to left ventricular dysfunction (reduced ejection fraction), it may cause further deterioration in heart function and increase the risk of heart failure.

Indications for surgery: Surgery is indicated if left ventricular function declines or if there's progressive worsening of the patient's condition despite medical treatment.

Risk of Arrhythmias

When it occurs: Patients with MVP are at risk for arrhythmias, particularly atrial fibrillation or ventricular arrhythmias, due to structural changes in the heart, such as atrial dilation from chronic regurgitation.

Indications for surgery: Surgery may be needed if arrhythmias cannot be controlled with medications, particularly if they are associated with significant symptoms, such as palpitations, dizziness, or syncope, or if there's an increased risk of stroke or other systemic embolic events.

Progressive Symptoms in Asymptomatic or Mild MVP

When it occurs: In rare cases, asymptomatic or mildly symptomatic MVP can worsen over time, leading to significant mitral regurgitation or other complications that require surgery.

Indications for surgery: Surgery may be considered in patients with progressive worsening of symptoms or worsening structural changes despite close monitoring.

Endocarditis or Other Infections

When it occurs: If infective endocarditis (an infection of the heart valves) occurs on the prolapsed mitral valve, surgery may be needed to remove infected tissue or repair/reconstruct the valve.

Indications for surgery: Surgical intervention is necessary when the infection cannot be controlled with antibiotics alone or when there is significant damage to the valve, leading to severe regurgitation or heart failure.

SURGICAL TECHNIQUES FOR MVP

The goal of surgery for MVP is to restore the normal function of the mitral valve, alleviate symptoms, prevent further complications, and improve long-term outcomes. There are two primary surgical approaches: **mitral valve repair** and **mitral valve replacement**.

Mitral Valve Repair: Mitral valve repair is generally preferred over valve replacement whenever possible because it preserves the patient's own valve and typically provides better long-term outcomes. Repair can involve several techniques depending on the type and severity of the MVP.

Techniques for Mitral Valve Repair:

Annuloplasty: The mitral valve annulus (the ring-like structure around the valve) may be resized and reinforced using a prosthetic ring to restore the valve's shape and function. This is commonly used in cases of mitral regurgitation associated with MVP, where the valve is unable to close properly.

Chordal Repair: In some cases, the chordae tendineae (the fibrous cords that connect the valve leaflets to the heart muscle) may be stretched or torn. These chordae can be repaired or replaced to ensure the valve leaflets close properly.

Leaflet Resection or Plication: In cases where the mitral valve leaflets are excessively thickened or redundant, parts of the leaflet may be removed or reshaped to improve the function of the valve.

Edge-to-Edge Repair: If the leaflets are insufficient or fail to coapt (meet properly), an edge-to-edge repair can be done, where the edges of the leaflets are stitched together to reduce regurgitation.

Posterior Leaflet Enlargement: In some cases, the posterior leaflet may be enlarged to improve the valve's coaptation and reduce regurgitation.

Advantages of Repair:

- Preservation of the native valve, leading to better long-term outcomes and fewer complications.
- Lower risk of clot formation compared to valve replacement (no need for lifelong anticoagulation in most cases).
- Better functional recovery and fewer complications post-surgery.

Mitral Valve Replacement: Mitral valve replacement (MVR) is considered when mitral valve repair is not feasible, either because the valve is too damaged or because other complications are present that require replacement. MVR can involve either a biological (tissue) valve or a mechanical valve

.

Types of Mitral Valve Replacement:

Mechanical Valves: Made from durable materials such as carbon or titanium, mechanical valves are designed to last a long time but require lifelong **anticoagulation therapy** (e.g., warfarin) to prevent clotting on the valve.

Biological (Tissue) Valves: Made from animal tissue (typically porcine or bovine), tissue valves do not require lifelong anticoagulation but may wear out over time and may need replacement after 10–20 years, depending on the patient's age and activity level.

Indications for Valve Replacement:

- Severe MVP with extensive valve damage that cannot be repaired (e.g., calcification, complete rupture of chordae).
- Severe, symptomatic mitral regurgitation where repair is not possible or has failed.
- Infected or non-functional mitral valve in cases of endocarditis or other valvular infections.

Advantages of Valve Replacement:

- Necessary when repair is not possible or the valve is too damaged.

- Provides long-term relief from symptoms and restores normal heart function.
- For mechanical valves, long-term durability, though requiring lifelong anticoagulation.

Minimally Invasive Surgery: In some cases, particularly in patients who are candidates for mitral valve repair, **minimally invasive approaches** may be used to perform the surgery with smaller incisions. This technique may involve the use of robotic assistance or thoracoscopic techniques, reducing recovery time, minimizing pain, and improving cosmetic outcomes.

Transcatheter Mitral Valve Repair/Replacement (TMVR)

For patients who are not candidates for traditional open heart surgery, trans catheter procedures may be an option. These minimally invasive techniques are done via catheterization, often through the groin, to repair or replace the mitral valve.

MitraClip: A device used to reduce mitral regurgitation by clipping together the mitral valve leaflets, making it an option for patients who cannot undergo surgery.

CONCLUSION

Mitral Valve Prolapse is a common but often mild condition that can cause significant morbidity in its more severe forms. Early diagnosis and treatment—ranging from pharmacological management to surgical intervention—can significantly improve outcomes. With appropriate monitoring and intervention, many individuals with MVP can lead normal lives, but severe cases may require surgical repair or replacement of the mitral valve to prevent complications such as heart failure, arrhythmias, or stroke.

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REVIEW ON STROKE: STROKE

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ABSTRACT:
Stroke is a major cause of disability worldwide. Stroke is a condition where blood flow to the brain is disrupted, causing cell death. Neuroimaging techniques like computed tomography and magnetic resonance imaging have improved brain visualization, aiding in diagnosis, prognosis, and treatment of stroke patients. These techniques are crucial in clinical Management [1]. Stroke, the second most common cause of death w orldwide, claims the lives of 5.5 million peope eac h year, and up to 50% of survivors develop perman ent didabilities [2]. Innovative therapies and preventative strategies continue to be a top res earch objective even while breakthroughs in stroke care have improved quality of life for stroke survi vors [3].

INTRODUCTION:
Strokes are the main explanation of longterm depr eciation around the globe and the second most com mon cause of demise after myocardial infarction. Strokes can be classified as either ischemic or hem orrhagic. On the one this point, a hemorrhagic stro ke is caused by a rupture of blood vessels in or nea r the brain, whereas an ischemic stroke originates when a blood clot suddenly interrupts the blood flo w to an area of the brain [1]..Cardiovascular conditions are linkedto a low er quality of life and are the primary cause of death worldwide. Stroke is the second most common cause of mortality worldwide and is known to be the primary cause of long-term physical and cognitive impairment in people [4].

HISTORICAL MILESTONE IN STROKE RESEARCH:

Scientist	Year	Contribution
Hippocrates	460-370BCE	Described occlusion of carotid arteries causing loss of consciousness.
Johann Jacob Wepfer	1658	Reported apoplexy resulted from obstruction of carotid or vertebral artery or bleeding into the brain.
Giovanni Battista Morgagni	1682-1836	Related clinical presentations of stroke to morbid anatomical findings of brain.
John Cheyne	1682-1836	Contributed to understanding stroke pathology.
Moniz and Seldinger	20th century	Developed angiography

TYPES OF STROKE:

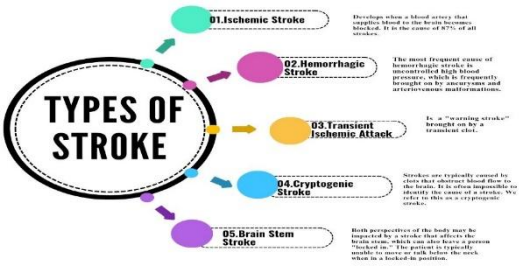


Figure 1: Types of Stroke

ETIOLOGY:

An episode of embolism or thrombosis that impai rs blood flow to a part of the brain is the cause of ischemic stroke. Blood flow to the brain is block ed in a thrombotic event when a thrombus (clot) f orms inside the blood vessel, typically as a result of atherosclerotic disease, arterial dissection, fibr omuscular dysplasia, or inflammatory diseases. B lood flow through the damaged channel is obstru cted by debris from other parts of the body durin g an embolicevent. The proximal artery may be t he cause of emboli, such as an internal carotid art ery atherosclerotic plaque, which can result in an artery-to artery embolic stroke distant from any proximal source, most often the heart [5] [6].

RISK FACTORS:

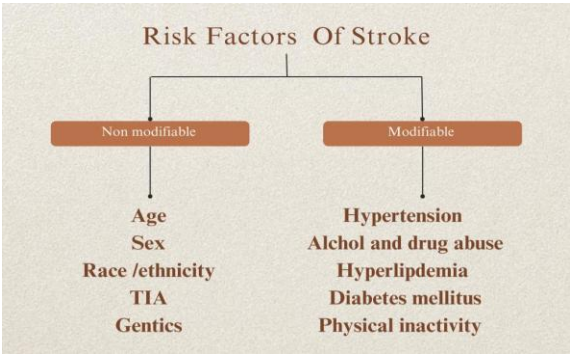
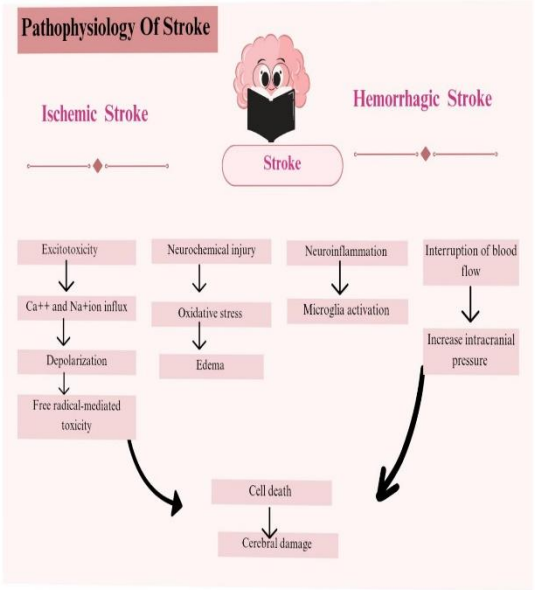


Figure 2: Risk Factors of Stroke



Numerous risk factors, disease processes, and mechanisms can contribute to stroke, which is not a single disease. Although its impact varies depending on the subtype, hypertension is the most significant modifiable risk factor for stroke. The majority of strokes (85%) are ischemic, mostly brought on by big artery atherothromboembolism, cardioembolism, and small vessel arteriolosclerosis [7].

EPIDEMIOLOGY:

According to the 2019 Global Burden of Disease data, one in four people will experience a stroke at some point in their lives. Nine to six million ischemic strokes and four to one million hemorrhagic strokes, including intracerebral and subarachnoid hemorrhages, are thought to occur annually around the world [8]. Stroke caused 1 in 6 cardiovascular disease-related deaths in 2021, with 795,000.Americans experiencing it annually. About 185,000 are ischemic strokes, with 87% stopping blood flow to the brain. The Framingham Heart Study reports a decrease in stroke incidence, but the majority is White[9].

The financial burden of stroke in the US amounted to nearly $56.5 billion between 2018 and 2019, covering healthcare expenses, medication, and lost productivity due to missed workdays. Stroke stands as a primary contributor to severe long-term disability, particularly affecting mobility in over half of stroke survivors aged 65 and Older[10].].Racial and ethnic differences in stroke incidence and outcomes are evident; non-Hispanic Black adults are almost twice as likely as White adults to have their first stroke, and both non-Hispanic Black and Pacific Islander adults have the highest rates of stroke-related death. Additionally, the number of stroke-relateddeaths increased from 38.8 per 100,000 in 2020 to 41.1 per 100,000 in 2021[11].

PATHOPHYSIOLOGY:

Stroke is the second largest cause of death globally in Western nations and one of the most common causes of disability [12].Ischemic occlusions cause 85% of stroke deaths, with intracerebral

hemorrhage accounting for the remaining percentage. The brain experiences thrombotic and embolic conditions due to atherosclerosis-induced vessel narrowing, resulting in thrombotic stroke. Embolic strokes cause reduced blood supply to the brain, leading to extreme stress and premature cell death (necrosis). Following necrosis, the plasma membrane is disrupted, organelles swell, and neurons stop working. Other factors contributing to stroke pathology include inflammation, energy failure, and loss. Hemorrhagic stroke, a high-risk condition, causes blood vessel rupture due to brain stress and injury, leading to infarction. It's classified into intracerebral and subarachnoid hemorrhage, caused by hypertension, disrupted vasculature, and excessive anticoagulant use [13].

Figure 3: Pathophysiology of Stroke

DIAGNOSIS:

The current standard for diagnosing brain and neurovascular diseases is head CT, which can rule in hemorrhagic stroke with over 95% accuracy and major stroke in two-thirds of cases with ischemic changes [14].CT scans for minor strokes do not confirm or exclude ischemia, but MRI offers greater spatial resolution and is preferred for inclusive diagnosis in mild deficits[15].

TREATMENT AND ADVANCED RESEARCH:

One of the most successful medical interventions f or major large vascular occlusions (LVO) that result in a stroke is mechanical thrombectomy (MT), which is currentl y unquestionably the gold standard[16].Thrombect omy device research and development has benefited greatly from preclinical testing utilising in-vitro models. The interplay between the thrombus, devices, and vessel shape during thrombectomy can be visualised using simulations of thrombectomy utilising transparent and synthetic vascular models with thrombus analogues [17].

Stroke Management approaches:

Excitotoxicity: Neuronal depolarization and the cell's incapacity to sustain membrane potential. The glutamate receptors α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) and N-Methyl-D-aspartate (NMDA), which were identified among the initial neuroprotective drugs explored in stroke prevention, mediate this process. Unfettered calcium influx and protein damage result from the premature release of glutamate, which overwhelms the machinery that eliminates glutamate from the cell and causes aberrant release of NMDA and AMPA molecules. Therefore, it has not been demonstrated that these medicines lessen neuronal death in human individuals. Instead of focusing on glutamatergic signaling directly, addressing the molecular pathways downstream of Excitotoxicity signaling may reduce side effects of process [13].

GABA (gamma aminobutyric acid) agonists: Clomethiazole is a GABA agonist that is being tested to assist individuals with stroke symptoms, but it hasn't been able to lessen glutamate receptor toxicity [18].

Calcium (Ca2+) channel blockers: In animal models of brain injury, voltage-dependent Ca2+ ion channel blockers are being demonstrated to reduce the ischemic insult. When given to stroke patients in Phase I and II clinical studies, the Ca2+ ion chelator DP-b99 demonstrated effectiveness and safety. Likewise, in people with stroke treated within 12 hours of start, Phase II trials showed a significant improvement in clinical symptoms [19].

Sodium (Na+) channel blockers: In a number of animal stroke models, Na+ channel blockers are being employed as neuroprotective drugs. They lessen white matter damage and stop neuronal death. Clinical trials have evaluated a variety of voltage-gated Na+ channel blockers, but the majority have shown little efficacy [20]. Although further research is needed to validate its usefulness, mexiletine, a neuroprotectant and Na+ channel blocker, has shown promise in treating grey and white matter ischemic stroke [21].

Intravenous thrombolytics (IVT): Initially created to treat cardiac thrombolysis, the IVT therapy paradigm has been shown to be successful in treating stroke patients. The age of the clot, the thrombolytic agent's specificity for fibrin, and the existence and half-life of neutralizing antibodies are some of the variables that affect how effective thrombolytic medications were [22]. The drugs used to treat IVT function by encourage the production of fibrinolysin, which catalyzes the breakdown of the clot obstructing the cerebral artery. The US National Institute of Neurological Disorders and Stroke (NINDS) undertook research that resulted in the development of the most successful IVT medication, recombinant tissue plasminogen activator (rt-PA, also known as alteplase) [23].

Intra-arterial thrombolytics: Another strategy for treating acute stroke is intra-arterial thrombolysis, or IAT. This treatment, which calls for skilled physicians and angiographic procedures, is most successful within the first six hours following the onset of MCA blockage [19].Middle Cerebral Artery Embolism Local Fibrinolytic Intervention (MELT) and Prolyse in Acute Cerebral Thromboembolism II (PROACT II) were randomized.Two small clinical trials coupled glycoprotein IIb/IIIa antagonists with thrombolytics; this strategy was less successful in treating cardioembolism but beneficial in managing atherosclerotic occlusions [24,25].IVT and IAT were studied in tandem in the Interventional Management of Stroke (IMS) III study to evaluate the advantages of combining improved recanalization methodology for speedier relief (IAT) with rapid delivery of medication (IVT) [26].

Antiplatelet therapy: Antiplatelet therapy including aspirin, clopidogrel, and ticagrelor, is crucial for acute ischemic stroke management, prevention, and control of non-cardioembolic ischemic stroke and TIA in stroke patients [27]. Dual antiplatelet therapy, combining clopidogrel, prasugrel, or ticagrelor with aspirin, is popular and most beneficial after stroke, especially if started within 24 hours and continued for 4-12 week's [28]

Glucose management: Stroke patients often experience hyperglycemia, which can lead to lipid peroxidation, cell lysis, and stroke complications. This condition worsens edema formation, increases cell death, accelerates ischemic injury, and is associated with progression of infarction, reduced recanalization, and poor clinical outcomes [29].Both diabetic and non-diabetic stroke patients can lower their risk of stroke by using continuous glucose monitoring devices [30].

Antihypertensive therapy:

The multi-center Acute Candesartan Cilexetil Therapy in Stroke Survivors (ACCESS) Phase II research demonstrated that taking candesartan for blood pressure during a stroke was safe; no planned cerebrovascular complications resulting from hypotension were documented. Antihypertensive medications have also been the subject of similar studies. For example, the Scandinavian Candesartan Acute Stroke Trial (SCAST) sought to assess the impact of the medication candesartan on stroke and cardiovascular disease; the Control of Hypertension and Hypotension Immediately Post Stroke (CHHIPS) study sought to establish the cut-off value for blood pressure during an attack; and the Continue or Stop Post Stroke Antihypertensives Collaborative Study (COSSACS) examined the effectiveness of antihypertensive therapy in stroke [19].

The SCAST trial indicated that a cautious BP-lowering treatment was linked to a higher risk of a poor clinical result [32], while the CHHIPS study showed that a very moderate fall in blood pressure decreased the death rate [33

Stem cell therapy: Stem cell therapy offers promising therapeutic opportunities for stroke patients, with research showing potential for tissue regeneration, maintenance, migration, proliferation, and neurocircuitry rewiring. A new type of mesenchymal stem cells, multilineage differentiating stress-enduring (Muse)cells, have

been tested as a stroke treatment, demonstrating functional recovery in damaged tissue [35,36]

.**Stroke rehabilitation:**Stroke rehabilitation aims to reduce stroke-related disability through a structured process of assessment, goal setting, intervention, and reassessment by a multidisciplinary team. Even with improvements in acute stroke care, man stroke victims still suffer from severe disabilities

It is anticipated that the prevalence of stroke related impairment would

rise globally over the next several decades, having a significant effect on families, healthcare systems, and economy.

Reducing impairment following a stroke is largely dependent on effective neurorehabilitation [36].

TRENDS IN STROKE RESEARCH:

Stroke Incidence Trends in highincome economi es in the TwentyFirst Century:

A Systematic Assessment and Population-Based study: Population-based studies offer reliable data on stroke incidence, showing a 42% decrease in high-income countries between the 1970s and early 2000s, but the trend's sustainability remains uncertain. The study used data from OCSP and OXVASC, along with other studies, to calculate age-standardized relative incidence rate ratios and projection estimates for stroke incidence in the UK from 2015 to 2045[37]. Stroke-related emergencies have decreased due to improved understanding, new drugs, and technological advancements like telestroke [38] and mobile stroke units, reducing mortality and morbidity [39]. Stroke Recovery and Rehabilitation Roundtables bring together physiotherapists and experts to recommend research directions and guide post-stroke healthcare systems, focusing on optimized care delivery and rehabilitation access [40]

Regenerative therapy, which rebuilds brain networks and repairs damaged neurons caused by ischemia insult, is the result of breakthroughs in stem cell technologies and genetics [41,42]. Natural compounds like Honokiol, synthesized at lower costs, have shown neuroprotective effects in animal models, reducing oxidative stress and inhibiting inflammatory responses, making them a cost-effective and safe alternative [43].

Future clinical trials should describe recovery and the clinical consequences in addition to evaluating the safety and effectiveness of medications. The recommendations should be followed in clinical studies of pharmaceutical treatments for stroke recovery [44].Recent years have seen rapid advancements in stoke management research, with new technologies expected to lead to further valuable discoveries through hypothesis-driven clinical trials.

FUTURE DIRECTION:

Investigate genetic and environmental stroke risk factors. Develop novel imaging techniques for early detection. Explore stem cell therapy, gene editing, and neuroprotection.

CONCLUSION:

Stroke remains a significant global health burden, affecting millions worldwide. Advances in imaging, thrombolytic therapy, and endovascular treatment have improved Outcomes. Emerging therapies, such as stem cell therapy and gene editing, hold promise.

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CONGENITAL HEART DEFECTS

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ABSTRACT:
The congenital heart disease includes abnormalities in heart structure that occur before birth. Such defects occur in the fetus while it is developing in the uterus during pregnancy. 1 in every 100 children has defects in their heart due to genetic or chromosomal abnormalities, such as Down syndrome. The excessive alcohol consumption during pregnancy and use of medications, maternal viral infection, such as Rubella virus, measles (German), in the first trimester of pregnancy, all these are risk factors for congenital heart disease in children, and the risk increases if parent or sibling has a congenital heart defect. These are heart valves defects, atrial and ventricular septa defects, stenosis, the heart muscle abnormalities, and a hole inside wall of the heart which causes defect in blood circulation, heart failure, and eventual death. There are no particular symptoms of congenital heart disease, but shortness of breath and limited ability to do exercise, fatigue, abnormal sound of heart as heart murmur, which is diagnosed by a physician while listening to the heart beats. The echocardiogram or transesophageal echocardiogram, electrocardiogram, chest X-ray, cardiac catheterization, and MRI methods are used to detect congenital heart disease. Several medications are given depending on the severity of this disease, and catheter method and surgery are required for serious cases to repair heart valves or heart transplantation as in endocarditis. In this review the causes, diagnosis, symptoms, and treatments of congenital heart disease are described.[1]

INTRODUCTION:
Congenital heart defects (CHDs) are structural abnormalities in the heart. "Congenital" denotes that the disorders exist at birth. These problems occur when a baby's heart does not develop properly during pregnancy. Congenital cardiac problems are the most prevalent type of birth defect. Congenital cardiac abnormalities can affect how the heart pumps blood. They may cause blood to flow too slowly, in the wrong direction, or entirely block it. There are various types of congenital cardiac abnormalities. They can occur in one or several areas of the heart. The most prevalent varieties include: Septal defects are openings in the heart's wall between the left and right sides. Heart valve defects are issues with the valves that control blood flow. Defects in the large blood vessels that carry blood in and out of the heart Septal defects are holes in the heart's wall between the left and right sides. Heart valve defects are issues with the valves that govern the heart. Congenital heart defects can range from very mild problems that never need treatment to life-threatening problems at birth. The most serious congenital heart defects are called critical congenital heart disease. Babies with these defects usually need surgery in the first year of life. But the symptoms of milder heart defects may not show up until childhood or adulthood.[2]

CAUSES:
Researchers often don't know what causes congenital heart defects. They do know that changes in a baby's genes sometimes cause a heart defect. The changed genes may come from the parents, or the changes may happen during pregnancy.[2]

DIAGNOSIS:
To check for heart problems, your healthcare professional may use ultrasound images of the baby's heart prior to delivery. We refer to this as a fetal echocardiography. Between weeks 18 and 22 of pregnancy, it is carried out. All infants are examined for congenital cardiac

abnormalities in the initial days following delivery. To monitor blood oxygen, a pulse oximeter is attached to your baby's hands or feet. Additional testing will be required to determine whether your baby has a cardiac problem if the blood oxygen levels are insufficient. A physical examination is one of the various methods a healthcare professional may employ to identify congenital heart abnormalities in infants, kids, and adults. Specific cardiac tests to assess the heart's function. [2]

TREATMENT:

The kind and severity of the congenital cardiac abnormality determine the course of treatment. Among the potential therapies are:

Cardiac catheterization to fix minor flaws like a tiny hole in the heart's inner wall. A tiny tube is inserted into the heart through a vein during a catheterization. It may be necessary to have cardiac surgery to: Fix heart and blood vascular defects. Fix or swap out a heart valve. To assist the heart in pumping blood, insert a device into the chest. Perform heart transplantation. If your child has patent ductus arteriosus, a particular kind of congenital cardiac abnormality, medication is frequently utilized. All adults and children with congenital cardiac abnormalities require routine follow-up care. [2]

EPIDEMIOLOGY:

Congenital heart disease (CHD) is the most prevalent birth defect, with an estimated global incidence of 8 per 1,000 live births, while more recent investigations have found rates as high as 9.5 per 1,000. This aggregate estimate obscures significant regional variation. Although different criteria make quantification challenging, regional studies have indicated incidence rates ranging from 1.2 to 17 per 1,000 live births. As detection improved, reported incidences grew throughout time, and it is presently estimated that 1.35 million children are born with CHD each year. In 2017, at least 260,000 people died from CHD, with 180,000 of them being newborns. [3]

SYMPTOMS:

Some people born with a heart problem don't notice symptoms until later in life. Symptoms also may return years after a congenital heart defect is treated.Common congenital heart disease symptoms in adults include:

- Irregular heartbeats, called arrhythmias.
- Blue or gray skin, lips, and fingernails due to low oxygen levels. Depending on the skin color, these changes may be harder or easier to see.
- Shortness of breath.
- Feeling tired very quickly with activity. Swelling due to fluid collecting inside body tissues, called edema.[3]

RISK ELEMENTS:

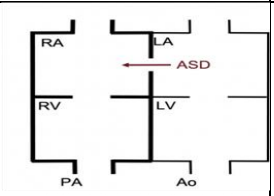
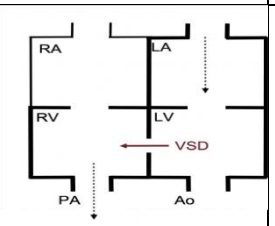
Congenital heart disease risk factors include: genetics. Congenital cardiac disease is hereditary since it seems to run in families. Gene alterations have been connected to birth defects in the heart. People with Down syndrome, for example, frequently have heart problems from birth. Rubella is another name for German measles. Pregnancy-related rubella may have an impact on the developing fetus's heart. You can determine whether you are immune to rubella with a blood test performed before becoming pregnant. For individuals who are not immune, there is a vaccination. diabetes. Pregnancy-related type 1 or type 2 diabetes may also alter the development of the fetus's heart. In most cases, congenital cardiac disease is not made more likely by gestational diabetes.[3]

COMPLICATIONS:

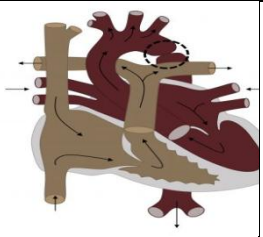
Arrhythmias are irregular heartbeats. Changes in heart signaling may result from scar tissue in the heart after procedures to treat a congenital cardiac disease. The heart may beat excessively quickly, too slowly, or irregularly as a result of the

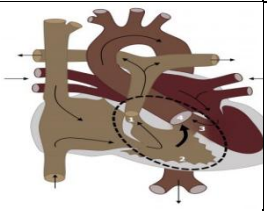
alterations. If left untreated, certain irregular heartbeats can result in stroke or abrupt cardiac death. Endocarditis is an infection of the heart's lining and valves. If left untreated, this infection can lead to a stroke or damage or destruction of the heart valves. Before receiving dental care, antibiotics could be suggested in order to avoid this illness. Frequent dental examinations are essential. Endocarditis risk is decreased by having healthy teeth and gums. [3]

PATHOPHYSIOLOGY [4]:

Type	Diagram	Pathophysiology
Atrial Septal Defect (ASD)	 Figure 6.1: Schematic of ASD with left-right shunt. Thicker lines imply volume overload.	Blood flow between the atria is permitted by ASDs. Blood flows from left to right because the pressure in the left atrium is greater than the pressure in the left (figure 6.1). The right side of the heart experiences volume overload as a result. Reduced right ventricular compliance could result from this high load as Remodeling takes place. Reduced compliance can increase right-side pressure, hence reducing the left-right shunt.
Ventricular Septal Defect (VSD)	 Figure 2: Schematic of a VSD with a left-right shunt that can cause volume overload in the RV, LA, LV, and pulmonary circulation. Dotted lines indicate blood recirculation back through the pulmonary circulation. Thicker lines indicate volume overload.	The manifestations of a VSD depend on the VSD size and the relative resistance of the pulmonary and systemic circulations—all of which will determine the direction of blood flow. During fetal development, the pulmonary and systemic circulations have equivalent resistances, so there may be very little shunting through the VSD, particularly if it is small. After birth, however, the resistance of the pulmonary system falls dramatically, so right ventricular pressure is lower and below left

		ventricular pressure (which still has to contend with systemic resistance)—consequently a left–right shunt is established. If this shunt is large (depending on the size of the defect), then blood returning from the pulmonary circulation to the left atrium can pass into the left ventricle, through the VSD into the right ventricle and head back into pulmonary circulation to start this loop again (figure 6.2). When a large VSD is present, the recirculated blood causes volume overload of the right ventricle and the pulmonary circulation and subsequently b oth chambers

		of the left heart (figure 6.2). This can eventually cause chamber dilation and lead to heart failure. The extra volume load in the pulmonary circulation can also lead to early onset of pulmonary vascular disease.
Coarctation of the Aorta	 Figure 3: AortaCoarctation (circled).	The reduced lumen generates an increase in afterload on the left ventricle. Vessels branching from the aorta before the coarctation can get normal blood flow; therefore, the head (carotid) and upper extremities (subclavian) are typically well perfused; however, branching arteries after the coarctation may be under perfused. Consequently,

		differentiated cyanosis is a probable symptom.
Tetralogy of Fallot (ToF)	 Figure 4 depicts Tetralogy of Fallot with 1) pulmonic stenosis, 2) RV hypertrophy, 3) VSD, and 4) overriding aorta.	The high resistance of the stenosed pulmonic valve (#1, figure 6.4) forces blood in the right ventricle to depart through VSD (#3, figure 6.4) and enter the left ventricle, generating a right-left shunt that bypasses the pulmonary circulation. As a result, blood containing venous PO2 reaches the systemic circulation, causing hypoxemia/cyanosis. The degree of hypoxemia/cyanosis observed is determined by the degree of pulmonic stenosis.

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VASCULITIS

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ABSTRACT:
Vasculitis encompasses various disorders that involve inflammation of blood vessels, potentially resulting in damage to vessel walls, thrombosis, and decreased blood flow. The disease can affect various organs and systems, and its manifestations depend on the type and location of the inflamed vessels. This review provides an overview of the key aspects of vasculitis, including its epidemiology, etiology, pathophysiology, clinical features, diagnosis, treatment options, and non-pharmacological interventions. We also explore the role of Ayurvedic treatments and recent advances in targeted drug therapies.

INTRODUCTION:
Vasculitis includes a range of conditions marked by the inflammation observed in blood vessels, which can cause damage to the vessel walls, lead to blood clots, and reduce blood circulation. The severity of vasculitis can vary widely, ranging from mild forms, such as hypersensitivity vasculitis, to more serious conditions like Granulomatosis with Polyangiitis (GPA) and Behçet's disease. Because its symptoms can be nonspecific, diagnosing vasculitis can be challenging, but early recognition is crucial to prevent organ damage and improve prognosis. The disease can affect both large and small blood vessels, and treatment typically involves immunosuppressive medications to manage inflammation and prevent further damage. [1]

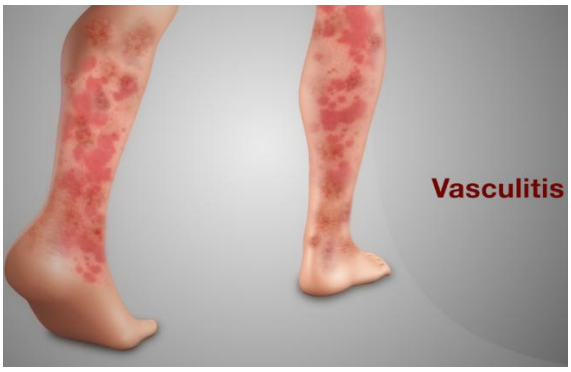


Fig 1:Vasculitis

EPIDEMIOLOGY: [2-6]
Vasculitis is a rare condition with annual incidence rates of 10 to 100 cases per million, varying by subtype. It often shows geographic and ethnic variation, such as higher rates of giant cell arteritis in Scandinavia and Takayasu arteritis in Southeast Asia. Risk factors include age (with peaks in childhood and older adulthood), gender (more common in females for certain types), genetic predispositions, and environmental exposures like silica dust.
Recent studies suggest a potential rise in the incidence of specific vasculitis types, possibly due to improved diagnostic practices and changes in environmental factors, including climate change.

TYPES OF VASCULITIS: [7-12]

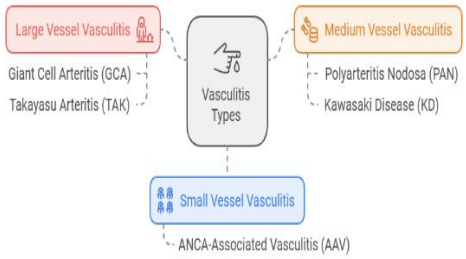


Fig 2: Types of Vasculitis

Vasculitis is typically classified based on the size of the affected blood vessels:

Vasculitis Type	Vessel Size	Synonyms	Major Affected Areas	Key Symptoms
Giant Cell Arteritis (GCA)	Large	Temporal Arteritis, Horton's Disease	Aorta, Cranial Vessels	Headaches, vision issues, scalp tenderness
Takayasu Arteritis (TAK)	Large	Pulseless Disease	Aorta, Major Branches	Reduced pulses, limb BP differences
Polyarteritis Nodosa (PAN)	Medium	-	Medium Arteries, Skin, Kidneys	Skin lesions, neuropathy, abdominal pain
Kawasaki Disease (KD)	Medium	Mucocutaneous Lymph Node Syndrome	Coronary Arteries, Skin, Mucous Membranes	Fever, rash, lymphadenopathy
Granulomatosis with Polyangiitis (GPA)	Small	Wegener's Granulomatosis	Respiratory Tracts, Kidneys	Sinusitis, haemoptysis, renal issues
Microscopic Polyangiitis (MPA)	Small	-	Kidneys, Lungs	Glomerulonephritis, pulmonary haemorrhage
Eosinophilic Granulomatosis with Polyangiitis (EGPA)	Small	Churg-Strauss Syndrome	Lungs, Skin, Heart, Nerves	Asthma, eosinophilia, neuropathy

ETIOLOGY: [13-17]

Several genetic variants, including HLA-DP, SERPINA1, and PRTN3, are linked to increased susceptibility to vasculitis, alongside strong associations between HLA-B51 and Behçet's disease. Environmental triggers include infections (like Epstein-Barr and hepatitis), certain drugs (such as propylthiouracil and hydralazine), and exposure to silica dust, though clear causal links are often lacking. The pathophysiology of vasculitis is primarily driven by immune dysregulation, with mechanisms varying by type, involving T-cell damage in giant cell arteritis and ANCA-mediated neutrophil activation in small vessel vasculitis.

PATHOPHYSIOLOGY:

The activation of immune cells, including B cells, T cells, and macrophages, leads to the formation of immune complexes and the production of pro-inflammatory cytokines, a hallmark of the immunopathogenesis associated with vasculitis.[18] In genetically predisposed people, environmental variables like infections are believed to set off the immunological response.[19] Endothelial cells are activated by the ensuing inflammation, which causes them to release chemokines and produce adhesion molecules, thus sustaining the inflammatory response.[20] Inflammation and damage to the blood vessel wall, which results in vascular stenosis, occlusion, and aneurysm formation, are the pathological characteristics of vasculitis.[21] Immune cells including neutrophils and macrophages and the accumulation of immune complexes and complement components are among the histological characteristics of vasculitis.[22]

SIGN & SYMPTOMS OF VASCULITIS: [23-28]

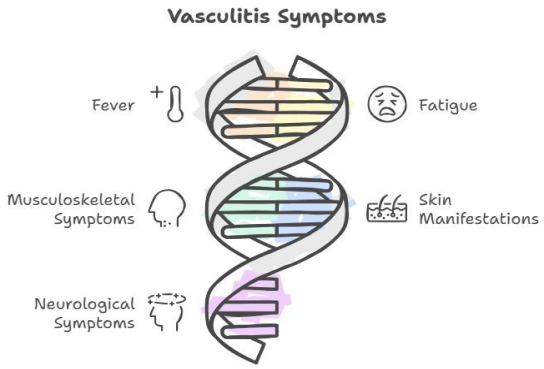


Fig 3: sign & symptoms of vasculitis

DIAGNOSIS: [29], [30]

Blood tests can indicate elevated inflammatory markers, such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), in vasculitis cases.

Step 1: Excluding Vasculitis Mimics and its Secondary Causes

Blood Tests → If positive, treat the infection.
Echocardiogram → To rule out heart involvement.
Hepatitis Screen (B & C) → To exclude viral causes.
HIV Test → To rule out HIV-related vasculitis.
Antiglomerular Basement Membrane Antibody → To exclude Goodpasture syndrome.

Step 2: Assess Extent of Vasculitis

Urine Tests and Microscopy → Detect kidney involvement.
Chest: Radiography → To identify lung

Step 3: Confirm Diagnosis of Vasculitis

Biopsy and/or Angiogram → Tissue biopsy or

Step 4: Identify Specific Type of Vasculitis

TREATMENT: (Specific Therapies) [30],[31]

PRECAUTIONS:

When caring for patients on immunosuppressant, it is vital to monitor them for infections because their immune systems are weakened. It is also important to avoid live vaccines, as they can pose significant risks to individuals with compromised immunity. Regular follow-up appointments are essential to evaluate the effects of immunosuppressive therapy and to ensure that any potential complications are addressed promptly.

NON-PHARMACOLOGICAL TREATMENT: [32]

Non-pharmacological interventions are essential in managing vasculitis alongside medication. These include physical therapy to preserve joint and muscle function, dietary changes to reduce inflammation, and quitting smoking to prevent disease flare-ups. Psychosocial support can also help address emotional challenges. Regular monitoring and follow-up care are crucial for assessing treatment effectiveness and identifying complications early.

Type of Vasculitis	Treatment
Behcet's Disease	- **Initial:** Oral colchicine with topical steroids- **If Ineffective:** Azathioprine, IFN alpha, TNF inhibitors, Thalidomide, apremilasnippst (30 mg twice daily)
ANCA-Associated Vasculitis (AAV)	- **Inducing Phase:** Cyclophosphamide (CYC) or Rituximab (RTX) - -**Maintenance Phase:** RTX (0.5-1 gm every 6 months), co-trimoxazole for respiratory infections - **Alternatively:** Avacopan (CCX168): a glucocorticoid alternative
Cryoglobulinaemic Vasculitis (CV)	- **Hepatitis C-induced:** Ribavirin with Sofosbuvir - **Non-Hepatitis C:** Rituximab, CYC, steroids, azathioprine for severe cases.
LargeVessel Vasculitis (GCA, Takayasu)	- **Steroids:** High-dose prednisolone (1 mg/kg/day, maximum 60 mg/day) for GCA. - **Steroid-Sparing Agents:** Methotrexate, cyclophosphamide, and mycophenolate mofetil. - **Biologics:** Tocilizumab, infliximab, adalimumab, and abatacept.
Hypersensitivity Vasculitis	- **Mild Cases:** Leg elevation, rest, antihistamines - **Severe Cases:** Steroids (1 or 2 mg/kg/day), Tapered for 8 to 12 weeks. - **Steroid-Sparing:** Dapsone, Colchicine, Hydroxychloroquine - **Other Options:** Cyclosporine, Azathioprim, Methotrexate.
Henoch-Schoenlein Purpura (HSP)	- **Severe Disease:** Cyclophosphamide (100-200 mg per day for 6-12 months) with Prednisolone (1-2 mg/kg/day) - **Steroid-Sparing:** Dapsone, Rituximab, Azathioprim, Mycophenolate mofetil - **Renal Failure occurs:** Plasmapheresis, dialysis and renal transplant.
Urticarial Vasculitis	- **Starting Treatment:** Colchicine, Prednisolone - **Steroid-Sparing:**
	Hydroxychloroquine/Mycophenolate mofetil/Methotrexate - **Biologics:** Canakinumab, Rituximab, Anakinra.
Kawasaki Disease	- **First-line To Start with:** IV Immunoglobulin (IVIG) (2g/kg) + aspirin (80-100 mg/kg/day) - **Second Dose:** If the fever continues, administer IVIG again. - **Long-term:** Low-dose aspirin (5 mg/kg/day)

AYURVEDIC TREATMENT: [33],[34]

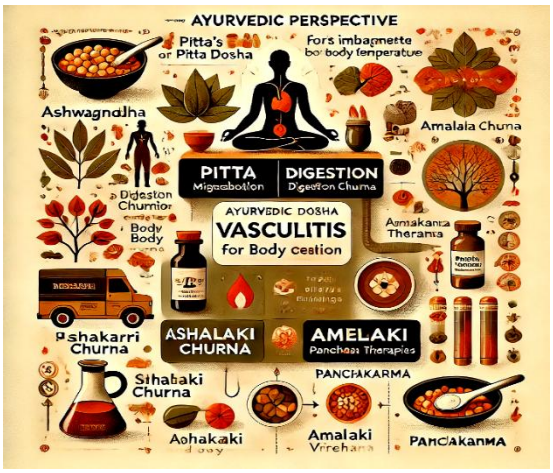
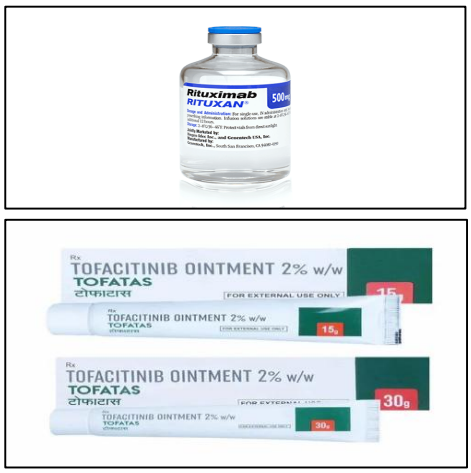


Fig 4: Ayurvedic treatment

RECENT TARGET OF DRUG ACTION: [35-37]

Rituximab – Monoclonal Antibody.
Janus Kinase(JAK)inhibitors- Tofacitinib



Depletes B-Cells
Blocks Janus kinase enzymes for Cytokinin
For Relapsing AAV Cases.
Signaling Pathways. (Mitigates Inflammation).

CONCLUSION:

Vasculitis is a complex group of disorders that can lead to significant health complications and high mortality if not addressed promptly. Advances in immunosuppressive therapies, biologics, and targeted treatments have improved outcomes for many patients. However, managing vasculitis is multifaceted, requiring both medication and non-medication approaches. Ongoing research into the underlying mechanisms and treatment options for vasculitis, including alternative therapies such as Ayurveda, is essential for enhancing patient outcomes.

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DILATED CARDIOMYOPATHY

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ABSTRACT:
Cardiomyopathy can be defined as a myocardial condition where the heart is structurally and functionally abnormal. DCM has a high risk of developing heart failure without any specific symptoms. The etiology both at the genetic and non-genetic level has been studied depend on their genetic linkage analysis and non-inflammatory or viral toxins. Patients of DCM may show thromboembolism or abrupt death when ignored earl signs of fatigue, dyspnea, dicrotic/ hypokinetic pulse or decreased blood supply to brain which may lead to serious conditions. Genetic diagnosis can help predict prognosis, especially with regard to arrhythmia risk for certain subtypes. There are diagnostic criteria and imaging modalities which are used to diagnose the disease, including echocardiography, MRI, biopsy etc. Different types of pharmacological and non-pharmacological treatments have been discussed over here and these are considered for both the genetic and acquired factors.

INTRODUCTION:
Dilated cardiomyopathy (DCM) is a myocardial disease that is identified by dilatation of the left ventricle along with impaired systolic function. Globally, dilated cardiomyopathy (DCM) is one of the most common reasons for a heart transplant in both adults and children and contributes majorly to heart failure (HF) and sudden cardiac death (SCD). The World Health Organization (WHO) defines DCM as a serious cardiac disorder in which structural or functional abnormalities of the heart muscle can lead to substantial morbidity and mortality owing to complications such as heart failure and arrhythmia [1] . Several pathogenic mechanisms have been identified, such as autoimmune reactions to acute viral myocarditis or genetic alterations in cytoskeletal components. Decades of research have revealed diverse aetiologies for DCM, including genetic mutations, infections, inflammation, autoimmune diseases, exposure to toxins, and endocrine or neuromuscular causes [2]. In the majority of cases, DCM is caused by idiopathic and familial illness. According to the definition of the WHO, primary cardiomyopathies are classified as dilated cardiomyopathy, hypertrophic cardiomyopathy, restrictive cardiomyopathy, and arrhythmogenic right ventricular cardiomyopathy [3].

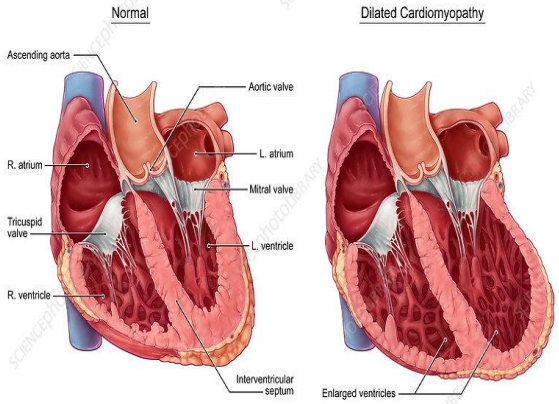

Fig 1: Introduction of Dilated cardiomyopathy

EPIDEMIOLOGY:
DCM is one of the most common causes of heart failure, with estimated prevalence of approximately 1:250–400 and up to 1:2500 in the general population [4,5]. 20%-50% of DCM cases are inherited, and more frequent abnormalities are visible in echocardiograms of asymptomatic relatives. In South Africa and Uganda, DCM accounts for 10% to 17% of all cardiac conditions encountered at autopsy [6,7,8] and in many parts of Africa, for 17% to 48% of patients who are hospitalized for heart failure. Whereas the

incidence and prevalence of DCM in the United States and elsewhere are reported to be 4 to 8 per 100,000 person-years and 36.5 per 100,000 individuals, respectively [9]. In the western world, up to 36% of DCM cases are associated with alcohol abuse [12]. If the 15 cases diagnosed only after death during the 12-year study period were included, the occurrence increased to 0.74 per 100,000 populations per year. Fifty-six new cases of dilated cardiomyopathy and 40 new cases of hypertrophic cardiomyopathy were diagnosed during the study period, giving average annual occurrences of 0.34/100,000/year (95% CI 0.26–0.44) and 0.24/100,000/year (95% CI 0.17–0.33) for new cases of dilated and hypertrophic cardiomyopathies, respectively.

ETIOLOGY:

The cause of DCM can be classified into genetic DCM and non-genetic or acquired DCM.

Genetic DCM

More than 30 genes have been involved in the development of DCM through genome-wide linkage analyses, candidate gene sequencing, and genetic association studies. These genes can be grouped into four major categories depending on their development and functioning with its implication on DCM: proteins forming the myocyte cytoskeleton, sarcomeric proteins, nuclear envelope proteins, and calcium homeostasis and mitochondrial function regulators. Genetic alterations of an autosomal dominant trait are mostly inherited; inheritance in an X-linked, autosomal recessive, or mitochondrial pattern is rare. The most common gene in which is a variant associated with DCM is found to be the *TTN* (Titin) gene, the largest known human gene. Additional genes frequently linked to DCM include variations in BAG3, MYH7 (β-myosin heavy chain), MYH6 (α-myosin heavy chain), and LMNA (Lamin A/C).

Non-genetic DCM

Idiopathic-inflammatory, viral, or autoimmune-mediated cardiomyocyte destructions are mediated directly via viropathic effects (acute phase) or indirectly via T-cell-mediated cytotoxicity (subacute phase) [10]. Fungi, parasites, and chemotoxins may lead to inflammation, which can be one of the causes of primary acute cardiomyopathy. In some families, a predisposition for autoimmune-mediated cardiomyocyte destruction has been observed. These patients' first-degree relatives had a higher frequency of autoimmune disorders, including juvenile diabetes, rheumatoid arthritis, thyroiditis, psoriasis, and asthma, pointing towards a possible role for MHC class II DQ polymorphisms in these familiar forms of autoimmune-mediated IDC [11].

PATHOPHYSIOLOGY:

Mutations in cytoskeletal components such as actin, desmin, and α-tropomyosin have been found [12]. The secondary causes include chemotherapy, excess alcohol consumption, and peripartum. Sometimes patients having DCM are unmasked due to myocardial insult or stress. Patient having DCM with no family history arises from acute myocarditis. An initial myocardial insult led by chronic inflammation, which leads to ventricular remodeling and dysfunction.

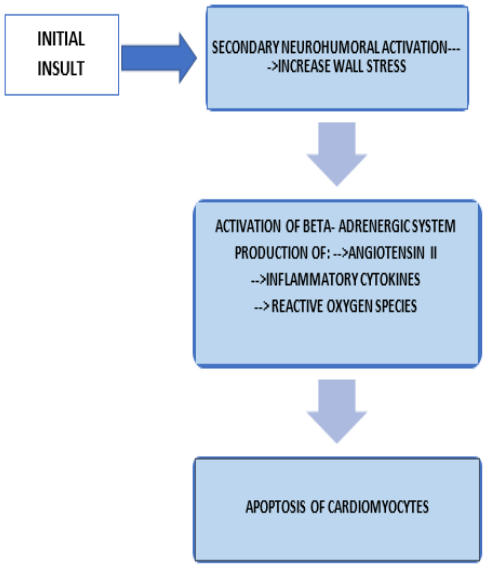

Fig 1: Pathophysiology

PATHOPHYSIOLOGICAL CHANGES INCLUDE:

- DECREASE IN STROKE VOLUME
- DECREASE IN CARDIAC OUTPUT
- IMPAIRED VENTRICULAR
- INCREASE IN END-DIASTOLIC

SIGNS AND SYMPTOMS:

The signs and symptoms of DCM are similar to conventional CHF, even though patients are less symptomatic. Decreased cardiac output, which is one of the pathological changes, leads to fatigue, cachexia, narrow pulse pressure, dicrotic pulse/hypokinetic pulse, cool extremities, decreased blood supply to the brain (cognitive dysfunction), dyspnea, and reduced blood supply to the kidney (renal failure). DCM may show as thromboembolism, abrupt death, or heart failure. As an alternative, patients who have subclinical DCM discovered during family exams may receive a diagnosis. Even within the same family, clinical manifestations might differ significantly, as explained in the section above.

DIAGNOSIS:

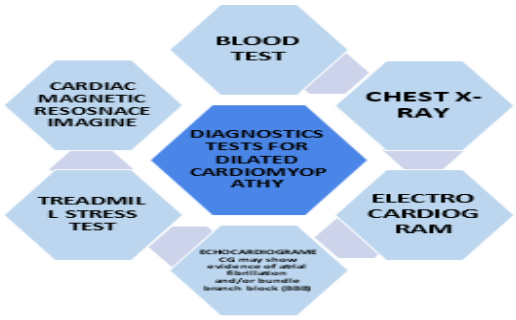

Fig 2: Diagnosis

In patients with DCM, the left ventricle will be dilated, with LVEDD >5cm [women] or 6 cm [men], though it should be connected to body surface area [BSA]. This will be associated with impaired systolic function. Cardiopulmonary exercise (CPEX) testing plays a role in measuring the adequacy of cardiac response to exertion and is a predictor of risk [13]. In individuals whose echocardiograms have less-than-ideal image quality, cardiac magnetic resonance imaging (MRI) may offer additional information and a more precise evaluation of chamber capacity and function. Electrocardiographic findings are nonspecific in most familial DCM; however, associated conduction disease should be considered. Prolongation of the PR interval is often the earliest manifestation of genetic DCM+E, and less common manifestations, such as atrial standstill, may develop with progressive disease [14]. Stress tests serve a dual role in the evaluation of a patient with presumed familial dilated cardiomyopathy: detection of coronary heart disease, along with quantification of exercise capacity.

TREATMENT:

The treatment of DCM aims to reduce the symptoms of heart failure and improve cardiac function. The standard drug therapy for heart failure in DCM patients includes beta-blockers and ACE inhibitors. Combined angiotensin receptor–neprilysin inhibitors reduce total mortality and hospital admissions compared with ACE inhibitors and could replace ACE inhibitors as one of the cornerstones of drug therapy in chronic heart failure [15]. Diuretics and mineral corticoid receptor antagonists should be given to treat symptomatic heart failure NYHA II-IV. Digoxin is primarily used only in the treatment of NYHA III and IV and can be recommended in the event of atrial fibrillation. Patient with sinus rhythm and pulse rate of > 70/min are recommended Ivabradine. The two major surgical options for DCM patients with heart failure are heart transplantation and implantation of long-

term mechanical circulatory support, which would be a temporary measure while awaiting transplantation or permanently.

NON-PHARMACOLOGICAL TREATMENT:

Paying attention to nutrition, diet, and exercise can help patients with heart failure. Practice of aerobic exercise training, over the long term, can help reverse left ventricular remodeling. Importantly, exercise is contraindicated in the active phase of inflammatory cardiomyopathy, both in athletes and non-athletes, and in DCM due to lamin A/C mutations [16]. Dietary sodium restriction to 2-3 g/day is recommended, and fluid restriction to 2 L/day is recommended for patients with the condition of hyponatremia. Aim for a healthy weight by consuming no more calories than you require each day. When engaging in physical exercise, balance the calories you consume with the calories you expend. Obesity and excess weight can increase cardiac workload.

CONCLUSION:

In today's modern lifestyle, dilated cardiomyopathy (DCM) is a globally challenging disease that has a high risk of heart failure, sudden cardiac death, and the need for heart transplantation. DCM arises from comorbidities that include genetic and non-genetic factors, and sometimes it's idiopathic. Despite advancements in diagnostics, including imaging and genetic testing, a considerable number of cases remain undiagnosed until the disease progresses to severe stages.Current treatment modalities, including pharmacological therapy with beta-blockers, ACE inhibitors, and emerging options like angiotensin receptor inhibitors, have shown promise in improving survival and reducing hospitalizations. However, for those with advanced disease, heart transplantation and mechanical circulatory support remain critical interventions. Heart devices are considered as a life-supporting treatment. Early diagnosis through improved screening tools, like MRI and ECHO, especially for at-risk populations, can help us prevent worsening of the condition.

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RHEUMATIC FEVER

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ABSTRACT:
An untreated Group a Streptococcus skin or throat infection can result in rheumatic fever, an autoimmune disease. Frequent bouts of rheumatic fever can result in heart failure, irreversible damage to the heart valves, and even death. Children between the ages of 4 and 19 are most commonly affected by rheumatic fever, an autoimmune disease that develops in response to an untreated Group A streptococcal (GAS) skin or throat infection.

KEY WORDS: Rheumatic fever, epidemiology, diagnosis of rheumatic fever, treatment of rheumatic fever

INTRODUCTION:

Most frequently manifesting between the ages of 5 and 14, acute rheumatic fever (ARF) is an immune-mediated illness caused by infection with Streptococcus pyogenes (Group A Streptococcus). While the RHD Australia Guidelines are applied in the Australian context, the updated Jones Criteria (2015) are used to diagnose ARF, a clinical syndrome. Approximately 60% of people who have an ARF episode go on to acquire chronic rheumatic heart [1]. Rheumatic heart disease, which can cause heart valve damage, heart failure, and even death, can be brought on by recurrent bouts of rheumatic fever [2]. One known cause of the immune-mediated reaction that leads to acute rheumatic fever is gas pharyngitis [9]. primary prevention of acute rheumatic fever (ARF), which includes prompt detection and appropriate management of impetigo and pharyngitis caused by superficial group streptococcus (GAS) infections. In the pathogenesis of the disease, GAS is the sole known inciting agent [10].

EPIDEMIOLOGY:

ARF incidence varies by area, with indigenous groups and low- and middle-income nations having the highest rates. In regions of hyperendemicity, the incidence of ARF can reach 300–500 cases per 100,000 children, although the global incidence is 8–51 cases per 100,000 people. Less than two incidents per 100,000 school-age children is the incidence in the United States and comparable high-resource nations. According to recent estimates, 33.4 million individuals worldwide suffer from rheumatic heart disease, and between 300,000 and 500,000 new episodes of rheumatic fever occur each year, with around 60% of those cases progressing to rheumatic heart disease. Its consequences cause 230,000 deaths [3].

PATHOPHYSIOLOGY:

Nonsupportive inflammatory lesions of the heart, joints, subcutaneous tissue, and central nervous system are the hallmarks of ARF. According to a thorough review of the literature, rheumatic fever usually follows pharyngeal infection with rheumatogenic group Some streptococci, at least in industrialized nations. Following an episode of streptococcal pharyngitis, there is an estimated 0.3–3% chance of getting rheumatic fever [4].

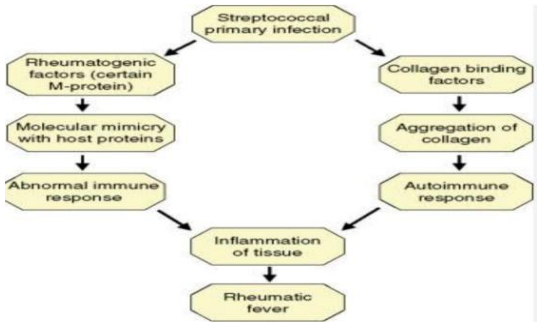

Figure no 1: Diagrammatic representation of pathogenesis of rheumatic fever [5,6]

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Streptococcal skin infections may also be linked to the development of rheumatic fever, according to research on the disease that affects Australia's indigenous tribes. And that streptococci of groups C and G might also act as starting infections. In Oceania and Hawaii, group A streptococcal strain not often linked to rheumatic fever have been reported to cause the illness, despite the fact that numerous classic group A streptococcal am types are thought to be rheumatogenic and most likely to be associated with acute rheumatic fever. It also seems that lower- and middle-income nations frequently experience this kind of potentially provoking group A streptococcal strain. The tissue damage that results from rheumatic fever is explained by molecular mimicry. A genetically susceptible host's humoral and cellular defenses are both engaged. Both B- and T-cell-mediated immune responses in the patient are unable to discriminate between the invasive microorganism and specific host tissues throughout this process. The cytokines Th17 and T helper 1 seem to be important mediators of rheumatic heart disease. The resulting inflammation causes the many symptoms of rheumatic fever and may last long after the initial illness has passed [5]

RISK FACTORS:

Poverty, overcrowding, and restricted access to healthcare are recognized risk factors. It is believed that cuts and abrasions on the skin can also allow streptococcal germs to enter the body. The high prevalence of rheumatic fever in isolated Australian communities may be partially explained by the high incidence of scabies [6].

DIAGNOSIS AND TREATMENT:

Children with suspected acute rheumatic fever, ages 3 to 17, for a conclusive diagnosis based on the Jones Criteria [7]. Two levels of diagnosis are used in ARF: primary care, when penicillin prophylaxis is initiated for suspected cases, and secondary/tertiary care, where echocardiography is done and the diagnosis is either confirmed or ruled out. Identification and therapy: It is necessary to classify all children with unexplained joint symptoms and heart murmurs that suggest mitral and/or aortic valve dysfunction as "suspected ARF." Additionally, unless there is a clear alternative reason for this symptom, people with chorea must be classed as ARF. Referral to a higher center should be organized, and standard ARF care, including BPG prophylaxis, should begin. It is anticipated that this streamlined method will identify more ARF cases in primary care settings, which could enhance RHD prevention, early diagnosis, and treatment. Refer to the section on BPG administration for information on the dosage and injection techniques. As secondary prophylaxis, BPG should be used every three weeks for children under the age of eighteen and every four weeks for adults. To confirm or rule out ARF, an echocardiogram and a review of the patient's medical history should be conducted at the secondary/tertiary center.

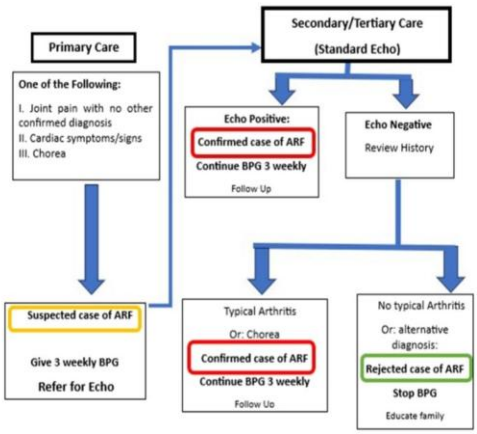

Figure no 2: Diagnosis and management of acute Rheumatic fever [9]

The standard Jones Criteria can be used if the patient arrived at the secondary or tertiary care unit while experiencing acute symptoms. If there is no or cured carditis, the prophylactic period lasts until age 25, and if there is persistent valve disease, it lasts forever. Oral penicillin V is an alternative if BPG is contraindicated; for individuals under 27 kg, the dosage is 250 mg

twice daily, and for those over 27, it is 500 mg twice daily for the duration of prophylaxis. Refer to the section on BPG allergy if you are an allergic patient. Anti-inflammatory drugs are necessary for patients with ARF. Ibuprofen 10 mg/kg/day every 8 hours for two weeks is the first-line treatment. For two weeks, aspirin (60 mg/kg/day) taken every eight hours is the second-line treatment. Steroids can be used if nonsteroidal anti-inflammatory drugs are not well tolerated, although there is no evidence to support their usage. For two weeks, prednisolone dosages ranging from 2 mg/kg/day to 80 mg/day can be given before being tapered and stopped. Steroids can be used if nonsteroidal anti-inflammatory drugs are not well tolerated, although there is no evidence to support their usage. After two weeks, prednisolone dosages ranging from 2 mg/kg/day to 80 mg/day can be reduced and stopped. Two daily doses of carbamazepine (5–10 mg/kg) or sodium valproate (5–10 mg/kg) are used to treat rheumatic chorea [8].

CONCLUSION:

In low-resource environments and among some indigenous groups in high-income nations, ARF remains a significant public health concern. The environment, host genetic vulnerability, and GAS-related variables all influence the development of ARF. The pathophysiology is probably connected to molecular mimicry, in which antibodies against the GAS M protein react with the tissues of the valves. Carditis, chorea, and arthritis are the most prevalent clinical manifestations of ARF.

KEY POINTS:

1) Children aged 5 to 15 are primarily affected by rheumatic fever.
2) The goal of treatment is to control symptoms and inflammation.
3) Treating scarlet fever or strep throat infections as soon as possible and taking all of the recommended antibiotics is the best defense against rheumatic fever.
4) Long-term cardiac damage, such as heart failure or irregular heartbeat, is one of the serious risks.

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AORTIC ANEURYSMS

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ABSTRACT:
An aneurysm is a localized, abnormal dilation or ballooning of a blood vessel wall, typically occurring in arteries. It results from weakening of the vessel wall due to factors like high blood pressure, atherosclerosis, or genetic predisposition. Aneurysms are most commonly found in the aorta, brain (cerebral aneurysms), and peripheral arteries. They can remain asymptomatic or cause symptoms based on size and location. If untreated, aneurysms risk rupture, leading to life-threatening bleeding. Diagnosis relies on imaging modalities such as CT, MRI, or ultrasound. Treatment involves monitoring small aneurysms or surgical intervention, such as clipping or stent placement, for larger or symptomatic cases.

INTRODUCTION TO ANEURYSM [2,3]

An aneurysm is a pathological condition characterized by the abnormal enlargement or ballooning of a blood vessel wall, primarily due to structural weakening. It commonly occurs in arteries, where blood pressure is higher, and can lead to severe complications if untreated. Aneurysms are categorized based on their location, with the most prevalent types being aortic aneurysms (thoracic or abdominal), cerebral aneurysms (affecting brain arteries), and peripheral aneurysms (occurring in limbs). The etiology of aneurysms involves a combination of genetic, mechanical, and environmental factors. Conditions such as hypertension, atherosclerosis, connective tissue disorders (e.g., Marfan syndrome), infections, and trauma can contribute to the degradation of vessel walls. Lifestyle factors like smoking and chronic stress further elevate the risk.Clinically, aneurysms may remain asymptomatic for years, being detected incidentally during imaging studies. However, large aneurysms or those near sensitive structures may cause pain, pulsatile masses, or neurological deficits. The most critical risk is rupture, which can lead to catastrophic internal bleeding and is often fatal without immediate medical intervention. Diagnosis is achieved through advanced imaging techniques such as CT scans, MRIs, or ultrasounds. Management ranges from vigilant monitoring to surgical repair, including open surgery or minimally invasive endovascular procedures, depending on size and symptoms.

EPIDEMIOLOGY OF ANEURYSM [1,4]

The epidemiology of aneurysms varies significantly depending on the type and location of the aneurysm, as well as the population studied. The prevalence, risk factors, and demographic distribution provide insights into the burden of the disease and guide prevention and management strategies.

1. Overall Prevalence

Aortic Aneurysms: These are relatively common in the elderly population, with abdominal aortic aneurysms (AAAs) being more prevalent than thoracic aortic aneurysms (TAAs). The global prevalence of AAA in adults over 65 years' ranges from 4-8%, with a higher incidence in males.

Cerebral Aneurysms: Approximately 2-5% of the general population is thought to harbour cerebral aneurysms. Most are asymptomatic and unruptured, but rupture leads to subarachnoid haemorrhage, which carries high morbidity and mortality.

Peripheral Aneurysms: These are less common, accounting for fewer than 5% of all aneurysms. The popliteal artery is the most frequently affected site**. [4,5]**

2. Age and Gender Distribution

Age: Aneurysms are predominantly a disease of older individuals, with risk increasing significantly after age 55. This is due to cumulative vascular stress, degenerative changes in the arterial walls, and associated comorbidities such as hypertension and atherosclerosis.

Gender: Men are more likely to develop aneurysms, especially AAAs, at a ratio of 4:1 compared to women. However, women with aneurysms are at higher risk of rupture, particularly in the case of cerebral aneurysms.

3. Geographic and Racial Differences

The incidence and prevalence of aneurysms differ globally. In Western countries, the prevalence of AAAs has declined in recent decades, likely due to improved cardiovascular risk factor management and decreased smoking rates. In contrast, regions with limited access to healthcare may see higher mortality from undiagnosed or untreated aneurysms. Racial disparities are also notable. For instance, Caucasian males have the highest rates of AAA, while cerebral aneurysms appear more frequently in populations of East Asian descent.

4. Risk Factors [6]

The epidemiology of aneurysms is closely tied to modifiable and non-modifiable risk factors:

Non-Modifiable: Age, male gender, family history, and genetic predisposition.

Modifiable: Hypertension, smoking, hyperlipidaemia, and alcohol use. Smoking is the most significant risk factor for AAAs, while hypertension plays a pivotal role in cerebral aneurysms.

5. Incidence of Rupture

The risk of rupture varies by type, size, and location of the aneurysm. Small aneurysms (e.g., AAAs <5 cm) have a low annual rupture risk, whereas larger aneurysms (>5.5 cm) or those growing rapidly carry an exponentially higher risk. Cerebral aneurysms under 7 mm in diameter have a low rupture risk, but those >10 mm, especially in high-pressure areas like the anterior communicating artery, are at greater risk.

ETIOLOGY OF ANEURYSM [7,8]

The etiology of an aneurysm involves a multifactorial process where genetic, structural, and environmental factors contribute to the weakening of the blood vessel wall. Understanding the causes and underlying mechanisms is crucial for preventing, diagnosing, and managing this condition.

1. Structural and Mechanical Factors

Aneurysms result from the failure of the vessel wall to withstand intraluminal pressure. This failure occurs due to structural defects in the arterial wall, particularly the media layer, which consists of smooth muscle and elastic fibres. Over time, these layers may degrade due to:

Chronic Hemodynamic Stress: Persistent high blood pressure exerts excessive force on arterial walls, particularly at bifurcations where turbulent blood flow creates localized stress. This is a significant factor in the formation of cerebral and aortic aneurysms.

Atherosclerosis: Plaque build-up leads to inflammation and degradation of the vessel wall. It is a common cause of abdominal aortic aneurysms (AAAs).

Trauma: Direct injury to the vessel can weaken the wall, leading to traumatic aneurysms.

2. Degenerative Changes

Degeneration of the vascular wall, often age-related, plays a central role in aneurysm development:

Elastic Fiber Degradation: Loss of elastin weakens the wall's ability to recoil, making it prone to dilation.

Collagen Breakdown: Enzymes like matrix metalloproteinase (MMPs) degrade collagen, reducing the tensile strength of the wall.

Inflammation: Chronic inflammation, as seen in conditions like atherosclerosis or infection, leads to remodelling and weakening of the vessel wall.

3. Genetic Factors

Genetic predisposition is a significant contributor to aneurysm development, particularly in

individuals with a family history of the condition. Specific genetic and connective tissue disorders include:

Marfan Syndrome: A defect in fibrillin-1, a protein critical for elastin integrity, predisposes individuals to thoracic aortic aneurysms.

Ehlers-Danlos Syndrome (Type IV): A collagen synthesis defect increases the risk of arterial wall rupture and aneurysm formation.

Bicuspid Aortic Valve (BAV): Associated with thoracic aortic aneurysms due to abnormal hemodynamic stress on the ascending aorta.

4. Infection

Infectious or mycotic aneurysms arise from weakened vessel walls due to infection. Common causes include:

Bacterial Endocarditis: Bacteria can seed arterial walls, leading to localized destruction.

Syphilitic Aneurysms: Although rare today, tertiary syphilis historically caused aneurysms of the thoracic aorta.

Fungal Infections: Immunocompromised individuals are particularly at risk of fungal infections affecting arterial walls.

5. Inflammatory and Autoimmune Conditions

Non-infectious inflammatory conditions can also weaken the vessel wall:

Takayasu Arteritis: Inflammation of large vessels can result in aneurysmal dilation.

Giant Cell Arteritis: This condition affects medium-to-large arteries, increasing the risk of aneurysm formation.

6. Environmental and Lifestyle Factors

Several modifiable lifestyle factors significantly contribute to the etiology of aneurysms:

Smoking: The most important risk factor for AAAs, smoking accelerates elastin degradation, promotes inflammation, and raises blood pressure.

Hypertension: A major contributor to both aortic and cerebral aneurysms, high blood pressure creates mechanical stress on vessel walls.

Hyperlipidemia: Cholesterol buildup in vessel walls promotes atherosclerosis and weakens the arterial structure.

7. Congenital Causes

Congenital anomalies in vascular structure may predispose individuals to aneurysms. Examples include:

Berry Aneurysms: Linked to congenital defects in the Circle of Willis, where structural abnormalities in arterial walls predispose to cerebral aneurysms.

Coarctation of the Aorta: This congenital narrowing of the aorta may lead to downstream aneurysm formation due to increased wall stress.

8. Iatrogenic Causes

Medical interventions or procedures can inadvertently lead to aneurysms:

Post-surgical Trauma: Procedures involving vascular grafts or catheters may damage the vessel wall.

Radiation Therapy: Radiation-induced vascular damage can lead to long-term weakening and aneurysm formation.

9. Rare and Miscellaneous Causes

Polyarteritis Nodosa: A vasculitis affecting medium-sized arteries can cause aneurysms.

Fibromuscular Dysplasia: This rare condition leads to abnormal cellular growth in artery walls, potentially resulting in aneurysmal dilation.

PATHOPHYSIOLOGY OF ANEURYSM [1,3,9,10,11]

The pathophysiology of an aneurysm is a complex process involving the weakening of the blood vessel wall, its subsequent dilation, and eventual risk of rupture. It occurs through an interplay of structural, biochemical, genetic, and environmental factors that disrupt the normal architecture and function of the vessel wall. The process varies depending on the type and location of the aneurysm but shares common pathological mechanisms.

1. Normal Vascular Wall Structure

The arterial wall is composed of three layers:

Intima: Innermost layer with endothelial cells, providing a barrier and regulating vascular tone.
Media: Middle layer with smooth muscle cells, elastin, and collagen, responsible for strength and elasticity.
Adventitia: Outer layer with connective tissue, providing structural support.
The integrity of these layers is crucial for maintaining vascular resilience to blood pressure and flow dynamics.

2. Pathogenesis of Aneurysm Formation

Aneurysm development involves progressive damage to the arterial wall. Key steps include:

Mechanical Stress and Hemodynamic Forces

Arteries experience cyclic stress due to blood pressure and flow. Chronic hypertension increases wall tension (as per **Laplace's Law**), making vessels susceptible to dilation.
Areas of turbulence, such as arterial bifurcations or curvatures, are more prone to localized wall stress, especially in cerebral aneurysms.

Structural Degradation

The structural integrity of the arterial wall is compromised due to:
Elastin Fragmentation: Elastin provides elasticity, allowing vessels to recoil. Degradation of elastin fibers leads to loss of elasticity, wall dilation, and thinning.
Collagen Breakdown: Collagen provides tensile strength. Enzymes such as **matrix metalloproteinase (MMPs)**, particularly MMP-2 and MMP-9, degrade collagen, weakening the wall.
Medial Necrosis: Smooth muscle cells in the media die due to hypoxia, oxidative stress, or inflammation, further compromising wall strength.

C. Inflammatory Response

Inflammation plays a pivotal role in aneurysm formation. Activated immune cells (e.g., macrophages, T-cells) release cytokines and enzymes like MMPs, which degrade the extracellular matrix. Chronic inflammation induces vascular remodelling and adventitial fibrosis, destabilizing the wall.

D. Vascular Smooth Muscle Cell (VSMC) Dysfunction

VSMCs are essential for maintaining vascular tone and repairing damage. In aneurysms, VSMCs undergo apoptosis or phenotypic changes, impairing their ability to synthesize extracellular matrix proteins like collagen and elastin.Loss of VSMCs leads to thinning of the media and reduced repair capacity.

3. Specific Mechanisms by Aneurysm Type

A. Abdominal Aortic Aneurysm (AAA)

Atherosclerosis: Plaque buildup in the intima initiates chronic inflammation, leading to medial thinning and weakening.
Smoking and Elastase Activation: Smoking induces elastin degradation and inflammation, contributing to AAA progression.
Intraluminal Thrombus: AAAs often develop thrombi, which exacerbate hypoxia in the vessel wall, promoting VSMC apoptosis and matrix degradation.

B. Thoracic Aortic Aneurysm (TAA)

Genetic Disorders: Mutations in structural proteins (e.g., fibrillin-1 in Marfan syndrome) impair connective tissue integrity.
Cystic Medial Necrosis: Seen in aging and genetic conditions, it involves the loss of smooth muscle cells and accumulation of mucoid material in the media, weakening the wall.

C. Cerebral Aneurysm

Hemodynamic Stress: Cerebral arteries experience high pulsatile flow, particularly at bifurcations, making them prone to localized wall stress.
Endothelial Dysfunction: Damage to endothelial cells leads to increased permeability, inflammation, and weakened wall structure.
Genetic Susceptibility: Alterations in collagen synthesis predispose individuals to aneurysm formation in intracranial arteries.

D. Peripheral Aneurysm

Peripheral arteries, like the popliteal artery, are less affected by systemic factors like atherosclerosis. However, local trauma, infection, or connective tissue disorders can contribute to aneurysm formation.

4. Progression of Aneurysm

Once an aneurysm forms, it tends to grow due to ongoing wall stress and structural degradation:

Expansion: The aneurysm enlarges as intraluminal pressure exerts radial stress on the weakened wall. Larger aneurysms are at greater risk of rupture due to exponential increases in wall tension.

Thinning: The vessel wall becomes progressively thinner as the media and adventitia weaken.

Aneurysm Rupture: The wall eventually fails, causing catastrophic bleeding. The risk of rupture depends on:

Aneurysm size (e.g., AAAs >5.5 cm).

Growth rate (rapidly enlarging aneurysms are more prone to rupture).

Location (cerebral aneurysms often rupture at smaller sizes).

5. Molecular Pathways

The molecular basis of aneurysm formation includes:

Oxidative Stress: Reactive oxygen species (ROS) damage endothelial cells and promote VSMC apoptosis.

Cytokines and Proteases: Inflammatory cytokines (e.g., IL-6, TNF-α) and proteases (e.g., cathepsins, MMPs) degrade the extracellular matrix.

Genetic Mutations: Mutations in genes encoding structural proteins (e.g., COL3A1 in Ehlers-Danlos syndrome) weaken the arterial wall.

6. Protective Mechanisms

Collateral Reinforcement: Adventitial fibrosis can temporarily stabilize the wall.

Vascular Remodeling: Compensatory thickening of the intima may delay rupture but also contributes to luminal narrowing and ischemia.

SIGNS AND SYMPTOMS OF ANEURYSM [1,2,6,7]

The clinical presentation of an aneurysm varies widely depending on its size, location, and whether it has ruptured. Many aneurysms remain asymptomatic until they become large or rupture, leading to life-threatening complications. Below is a detailed overview of the signs and symptoms based on different types of aneurysms.

1. General Features of Aneurysms

Aneurysms share some common characteristics in their presentation:

Asymptomatic Phase: Small, unruptured aneurysms often go unnoticed and are detected incidentally during imaging for unrelated conditions.

Symptoms Due to Expansion: As an aneurysm grows, it may compress surrounding structures, leading to localized pain or dysfunction.

Symptoms of Rupture: Ruptured aneurysms cause sudden, severe symptoms and can be life-threatening.

2. Abdominal Aortic Aneurysm (AAA)

Unruptured AAA

Asymptomatic: Most small AAAs are asymptomatic and discovered during imaging.

Pulsatile Abdominal Mass: A classic sign, patients may feel a throbbing mass in the abdomen, especially in thin individuals.

Abdominal or Back Pain: Pain may develop due to pressure on nearby tissues, nerves, or organs.

Symptoms of Compression: Large AAAs may compress adjacent structures, causing symptoms such as:

Gastrointestinal symptoms (nausea, vomiting).

Leg swelling from compression of venous structures.

Ruptured AAA

Severe Abdominal or Back Pain: Sudden, intense, and tearing pain, often radiating to the back or groin.

Shock: Symptoms of hypovolemic shock include rapid heart rate, low blood pressure, cold and clammy skin, and fainting.
Grey-Turner Sign: Flank discoloration due to retroperitoneal hemorrhage.
Cullen's Sign: Periumbilical bruising, although rare.

3. Thoracic Aortic Aneurysm (TAA)

Unruptured TAA

Chest or Back Pain: Dull, aching pain in the chest or between the shoulder blades due to expansion.
Hoarseness: Compression of the recurrent laryngeal nerve can cause voice changes.
Difficulty Swallowing (Dysphagia): Pressure on the esophagus.
Shortness of Breath: Compression of the trachea or main bronchus.
Superior Vena Cava Syndrome: Compression of the superior vena cava (SVC) leads to facial swelling, cyanosis, and distended neck veins.

Ruptured TAA

Sudden Severe Chest Pain: Tearing or ripping pain that radiates to the back.
Hemodynamic Instability: Hypotension, shock, and loss of consciousness.
Haemoptysis: Coughing up blood, if the aneurysm ruptures into the lungs or airways.

4. Cerebral (Intracranial) Aneurysm

Unruptured Cerebral Aneurysm

Asymptomatic: Many small aneurysms are silent and discovered incidentally during imaging.
Neurological Symptoms: Large aneurysms may compress nearby structures, causing:
Headache: Persistent or localized pain.
Visual Disturbances: Blurred vision, double vision, or loss of vision due to pressure on the optic nerves or chiasm.
Cranial Nerve Palsies: Eye movement abnormalities due to compression of cranial nerves (e.g., oculomotor nerve).
Seizures: Rarely, large aneurysms can trigger seizures.

Ruptured Cerebral Aneurysm

Sudden Severe Headache: Often described as the "worst headache of my life."
Neurological Deficits: Weakness, numbness, difficulty speaking, or loss of consciousness due to subarachnoid hemorrhage.
Nausea and Vomiting: Often accompanies the headache.
Neck Stiffness: Due to irritation of the meninges (meningismus).
Seizures: Resulting from cortical irritation.
Coma or Death: Severe cases with extensive haemorrhage may result in rapid deterioration.

5. Peripheral Aneurysms

Popliteal Aneurysm

Limb Pain or Swelling: Due to compression of adjacent nerves or veins.
Distal Ischemia: Embolization from thrombus within the aneurysm may cause cold, pale, or painful feet.
Pulsatile Mass: A pulsating swelling in the popliteal fossa (behind the knee).

Femoral Aneurysm

Groin Mass: A pulsatile lump in the groin.
Limb Symptoms: Similar to popliteal aneurysms, including pain and ischemia.

6. Visceral Aneurysms

Splenic Artery Aneurysm

Abdominal Pain: Often in the upper left quadrant.
Hemodynamic Instability: If ruptured, it can cause severe pain and shock.

Renal Artery Aneurysm

Flank Pain: Persistent pain in the side.
Hypertension: Due to renal ischemia or arterial compression.
Haematuria: Blood in the urine, if rupture occurs.

7. Mycotic (Infectious) Aneurysms

Fever and Malaise: Due to underlying infection.
Localized Pain: Corresponding to the affected vessel.
Sepsis: If rupture occurs, it can lead to septicaemia and multi-organ failure.

Key Differences Between Symptomatic and Asymptomatic Aneurysms

Feature	Asymptomatic Aneurysm	Symptomatic Aneurysm
Size	Small, often stable.	Larger, expanding rapidly.
Symptoms	None or minimal.	Pain, compression, or neurological deficits.
Detection	Incidentally during imaging.	Presents with clinical complaints.

Diagnosis and Treatment of Aneurysm [11,12,13, 14, 15]

The diagnosis and treatment of aneurysms require a systematic approach involving imaging, risk assessment, and therapeutic strategies tailored to the aneurysm type, size, location, and risk of rupture. Below is a detailed explanation of the diagnostic techniques and treatment options.

Diagnosis of Aneurysm

1. Clinical Evaluation

History and Symptoms: Initial assessment includes documenting risk factors (e.g., smoking, hypertension, family history) and symptoms (e.g., pulsatile mass, pain, neurological deficits).

Physical Examination:

Abdominal Aortic Aneurysm (AAA): A pulsatile abdominal mass may be palpable in thin individuals.

Peripheral Aneurysms: Swelling, pain, or pulsation in affected limbs (e.g., popliteal, femoral regions).

Neurological Exam: For cerebral aneurysms, assess cranial nerve function and neurological deficits.

2. Imaging Techniques

A. Non-Invasive Imaging

Ultrasound (US)

Abdominal Aneurysm: Abdominal ultrasonography is the preferred screening tool for AAAs due to its high sensitivity, specificity, and cost-effectiveness.

Peripheral Aneurysms: Doppler ultrasound helps visualize arterial flow and aneurysm morphology.

Computed Tomography Angiography (CTA)

Provides detailed images of aneurysm size, shape, and surrounding structures.

Widely used for thoracic, abdominal, and cerebral aneurysms.

Magnetic Resonance Angiography (MRA)

Offers high-resolution imaging without ionizing radiation. Preferred in patients with renal impairment or when CTA is contraindicated.

CT or MRI Brain

For cerebral aneurysms, identifies unruptured aneurysms or subarachnoid hemorrhage (SAH) in case of rupture.

B. Invasive Imaging

Digital Subtraction Angiography (DSA)

Gold standard for evaluating cerebral aneurysms. Provides detailed vascular anatomy, aiding in treatment planning.Used selectively due to its invasive nature and associated risks.

3. Screening Guidelines

AAA Screening:

Recommended for men aged 65-75 who have ever smoked. Selective screening for women or non-smokers with strong family history.

Cerebral Aneurysm Screening:

Considered in individuals with familial predisposition or genetic disorders (e.g., Marfan syndrome, Ehlers-Danlos syndrome).

Treatment of Aneurysm

1. General Principles

The choice of treatment depends on:

Size and Growth Rate: Large or rapidly growing aneurysms are at higher risk of rupture.

Symptoms: Symptomatic aneurysms generally warrant treatment.

Rupture Status: Ruptured aneurysms require emergency intervention.

Location: The treatment modality varies by location (e.g., aortic vs. cerebral aneurysms).

Patient Factors: Age, comorbidities, and surgical risk influence treatment decisions.

2. Treatment Modalities

A. Medical Management

Risk Factor Modification

Control **hypertension** with beta-blockers or angiotensin receptor blockers (ARBs) to reduce wall stress. Encourage smoking cessation to slow aneurysm growth. Manage hyperlipidaemia with statins. Address diabetes and other comorbidities.

Surveillance

Regular imaging to monitor size and growth for small, asymptomatic aneurysms.

AAA: Ultrasound every 6-12 months for AAAs <5.5 cm.

Cerebral Aneurysm: Periodic MRI or CTA for aneurysms <7 mm.

B. Surgical and Endovascular Interventions

Open Surgical Repair

Procedure: Involves replacing the weakened segment with a synthetic graft.

Indications:

Large aneurysms (e.g., AAAs >5.5 cm or TAAs >6 cm).

Rapidly expanding aneurysms (>0.5 cm per year).

Symptomatic or ruptured aneurysms.

Advantages: Durable, with low recurrence risk.

Limitations: Invasive, longer recovery time, higher perioperative risks.

Endovascular Repair (EVAR/TEVAR)

Procedure: Minimally invasive approach using stent grafts inserted via femoral artery.

Indications: AAAs or TAAs unsuitable for open surgery. High-risk surgical patients.

Advantages: Lower perioperative mortality, shorter recovery.

Limitations: Requires regular follow-up to detect endoleaks or graft migration.

Cerebral Aneurysms

Clipping: Open surgical procedure involving a metal clip placed at the aneurysm neck to prevent blood flow.

Coiling: Endovascular procedure using detachable coils to fill the aneurysm and induce clotting.

Flow Diversion: Stent-like devices redirect blood flow away from the aneurysm sac.

Peripheral Aneurysms

Surgical bypass or endovascular stenting is preferred for limb-threatening aneurysms.

3. Emergency Management of Ruptured Aneurysms

Hemodynamic Stabilization:

Intravenous fluids and blood transfusion for shock. Emergency antihypertensive for cerebral aneurysms to prevent rebreeding.

Definitive Repair:

Open or endovascular repair for ruptured aortic aneurysms. Urgent clipping or coiling for ruptured cerebral aneurysms.

4. Emerging Therapies

Pharmacological Interventions: MP inhibitors and anti-inflammatory drugs to slow aneurysm growth (under research).

Gene Therapy: Targeting genetic pathways involved in connective tissue degradation.

Biodegradable Grafts:

Novel materials that integrate with native tissue, reducing complications.

Prognosis: Unruptured Aneurysms: Excellent prognosis with timely monitoring and elective repair.

Ruptured Aneurysms: High mortality (50-80%), emphasizing the importance of early detection and management.

Non-Pharmacological and Alternative Treatments of Aneurysm [13, 16, 17, 18]

Non-pharmacological and alternative treatments focus on lifestyle modifications, monitoring, and holistic approaches that complement medical or surgical interventions. These strategies aim to

reduce aneurysm progression and improve overall vascular health.

1. Lifestyle Modifications

Lifestyle changes play a crucial role in managing aneurysm progression and preventing complications.

Smoking Cessation: Smoking accelerates aneurysm growth and weakens the vessel wall by increasing inflammation and oxidative stress. Smoking cessation is one of the most effective non-pharmacological interventions for aneurysm management.

Blood Pressure Management: Controlling blood pressure reduces mechanical stress on the arterial walls, slowing aneurysm expansion. Non-pharmacological methods to lower blood pressure include:

Diet: Low-sodium, heart-healthy diets like the DASH (Dietary Approaches to Stop Hypertension) diet.

Exercise: Moderate aerobic activities, such as walking or cycling, improve cardiovascular health without exerting excessive strain on the aneurysm.

Stress Reduction: Practices like meditation, yoga, and mindfulness help reduce stress-induced hypertension.

Weight Management: Maintaining a healthy weight alleviates pressure on the cardiovascular system and reduces inflammation associated with obesity.

Diet and Nutrition

Antioxidant-Rich Foods: Diets high in fruits, vegetables, nuts, and seeds provide antioxidants that combat oxidative stress.

Omega-3 Fatty Acids: Found in fish or flaxseeds, omega-3s have anti-inflammatory properties that may benefit vascular health.

Limit Alcohol Consumption: Excessive alcohol can elevate blood pressure, increasing aneurysm risk.

2. Regular Monitoring and Surveillance

Imaging: Regular imaging (ultrasound, CTA, or MRA) is a key non-pharmacological approach for aneurysms that do not immediately require surgery.

Frequency of Monitoring: Small aneurysms: Ultrasound every 6-12 months. Moderate-sized aneurysms: More frequent imaging to assess growth rate.

B. Risk Assessment

Regular check-ups for blood pressure, cholesterol, and other cardiovascular risk factors. Genetic counselling and screening for individuals with a family history of aneurysms.

3. Exercise Therapy

Moderate Aerobic Exercise: Low-impact activities like walking, swimming, or yoga can improve circulation without exerting high mechanical stress on the aneurysm.

Avoid High-Intensity Exercise: Strenuous activities, such as heavy lifting, can increase blood pressure and aneurysm rupture risk.

4. Alternative and Complementary Therapies

While alternative therapies do not replace conventional medical treatments, they may support vascular health.

Acupuncture: May help manage high blood pressure and stress, indirectly reducing aneurysm risk.

Potentially beneficial for pain management in symptomatic aneurysms.

Herbal Remedies

Hawthorn (Crataegus): Used traditionally to improve heart and vascular health, hawthorn may have vasodilatory and antioxidant properties.

Garlic (Allium sativum): May reduce blood pressure and inflammation, improving cardiovascular health.

Curcumin: The active compound in turmeric, curcumin has anti-inflammatory and antioxidant effects.

Omega-3 Supplements

Omega-3 fatty acids from fish oil supplements may reduce systemic inflammation and improve endothelial function.

Mind-Body Practices

Yoga and Tai Chi: Help reduce stress and improve blood pressure regulation.

Meditation: Reduces sympathetic nervous system activity, lowering blood pressure and heart rate.

5. Psychosocial Support

Living with an aneurysm can cause anxiety or depression, particularly when under surveillance. Addressing psychological health is an essential aspect of non-pharmacological care.

Counselling or Support Groups: Helps patients cope with the emotional impact of living with an aneurysm.

Cognitive Behavioral Therapy (CBT): May alleviate stress and anxiety associated with aneurysm diagnosis.

6. Environmental and Behavioral Factors

Limit Exposure to Toxins: Reduce exposure to pollutants and chemicals that can exacerbate oxidative stress.

Adequate Sleep: Poor sleep patterns are linked to hypertension and cardiovascular disease.

7. Emerging Non-Pharmacological Strategies

Elastin Reinforcement: Experimental therapies include the use of biomaterials or scaffolds to reinforce weakened vessel walls.

Endovascular Monitoring Devices: Devices to monitor aneurysm size and pressure non-invasively in real time.

Limitations of Non-Pharmacological and Alternative Treatments: These approaches are not curative for aneurysms and cannot replace surgical or endovascular repair when indicated.

Alternative therapies, such as herbal supplements, should be used with caution, as they may interact with medications or have inconsistent efficacy.

Recent advancements in the pharmacological treatment of aneurysms focus on targeted and innovative approaches, including repurposing existing drugs and exploring novel therapeutic pathways: [14,15]

Metformin: Known primarily as a treatment for diabetes, metformin is under investigation for its potential to slow the progression of abdominal aortic aneurysms (AAA). Its anti-inflammatory and anti-oxidative properties may inhibit pathways involved in aneurysm development

Nanoparticle-based Therapies: Experimental studies highlight the use of nanoparticles to deliver drugs or therapeutic agents directly to aneurysmal sites. These approaches aim to stabilize the vascular wall and prevent further dilation

Prolylcarboxypeptidase (PRCP) Targeting: Advanced genetic studies suggest that targeting PRCP and related proteins could provide a molecular basis for reducing the risk of intracranial aneurysm rupture. This represents a significant step in using genetic and protein-level data to identify druggable targets

Gene Therapy and Biologics: Research into gene therapy for aneurysms is identifying specific genetic mutations and pathways that could be corrected or modulated. Biologics, such as antibodies against matrix metalloproteinase, are also under evaluation to prevent tissue degradation in the aneurysm wall

CONCLUSION:

An aneurysm is a potentially life-threatening condition characterized by the weakening and bulging of blood vessel walls. It can occur in various arteries, such as the aorta, cerebral arteries, and peripheral vessels, and may remain asymptomatic until it ruptures, causing severe complications like internal bleeding, stroke, or even death.

Ways to Treat Aneurysms

Treatment options depend on the aneurysm's size, location, and rupture status:

Surgical Interventions:

Open Surgery: Involves graft replacement of the weakened vessel.

Endovascular Repair: Minimally invasive stent placement to reinforce the vessel wall.

Medical Management:
Blood pressure control and risk factor management (e.g., smoking cessation, cholesterol reduction). Use of medications like beta-blockers and emerging therapies like metformin.

Alternative Treatments:
Lifestyle modifications and dietary changes can support vascular health. Experimental options like gene therapy and nanotechnology-based approaches show promise.

Effects of the Disease
Untreated aneurysms can lead to complications such as rupture, organ damage, and death. Even non-ruptured aneurysms may cause symptoms like pain, nerve compression, or ischemia, affecting quality of life. Early detection and appropriate management are critical for reducing morbidity and mortality.

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