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A basic review of cardiovascular diseases

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Abstract
Cardiovascular diseases (basic review) are to understand very easily for graduate, post graduate and post-doctoral ayush, dental, medical etc., students. I am explaining main and important diseases in cardiovascular system in day to day practical life for medical students and professionals. Diseases are ischemic heart disease, Raynaud's disease, aortic aneurism, rheumatic heart disease, valve heart diseases, infective endocarditis, Cardiomyopathy, pericarditis.

Keywords: cardiovascular diseases diseases, causes, clinical features, investigation

Introduction

Ischaemic heart disease

Ischemic heart disease also called as coronary heart disease. In the India one in three men and one in four women die from this disease. Chronic coronary artery disease (CAD) is estimated to affect 16.8 million people in the United States; of these, 9.8 million have angina pectoris, and nearly 8 million have had a myocardial infarction (MI). In 2002, out of 57 million deaths worldwide, approximately 16.7 million were due to cardiovascular disease (as compared with approximately 5 million due to tuberculosis, human immunodeficiency virus, and malaria combined), and 80% of these cardiovascular deaths were in the developing world. The article provides a state-of-the-art review of the literature on cardio vascular diseases for interested medical, dental, ayush physicians; appropriate articles were identified by searching the international journal database for the following terms: cardio vascular disease, causes [1].

Aetiology

Age and male sex: Males are more risk when compared to females.

Family history: Family aetiology may be due to genetic factors or the effects of a shared environment (diet, smoking habits etc). Hyperlipidaemia, hyperfibrinogenaemia and abnormalities of other coagulation factors are often genetically determined.

Smoking: Tobacco is probably the most important avoidable cause of coronary disease.

Hypertension: The incidence of coronary artery disease increase as blood pressure rises and excess risk is related to both systolic and diastolic blood pressure.

Hypercholesterolaemia: Patient with familial hyperlipidaemia have a high incidence of premature coronary disease and many epidemiological studies have demonstrated a positive correlation between mean population and plasma cholesterol concentration and morbidity and death from coronary disease.

Diabetes mellitus: This is associated with a increased incidence of ischaemic heart disease (IHD) and with a tendency to diffuse coronary atheroma [2].

Haemostatic factors, physical inactivity, obesity, alcohol, mental stress may leads to ischemic heart disease.

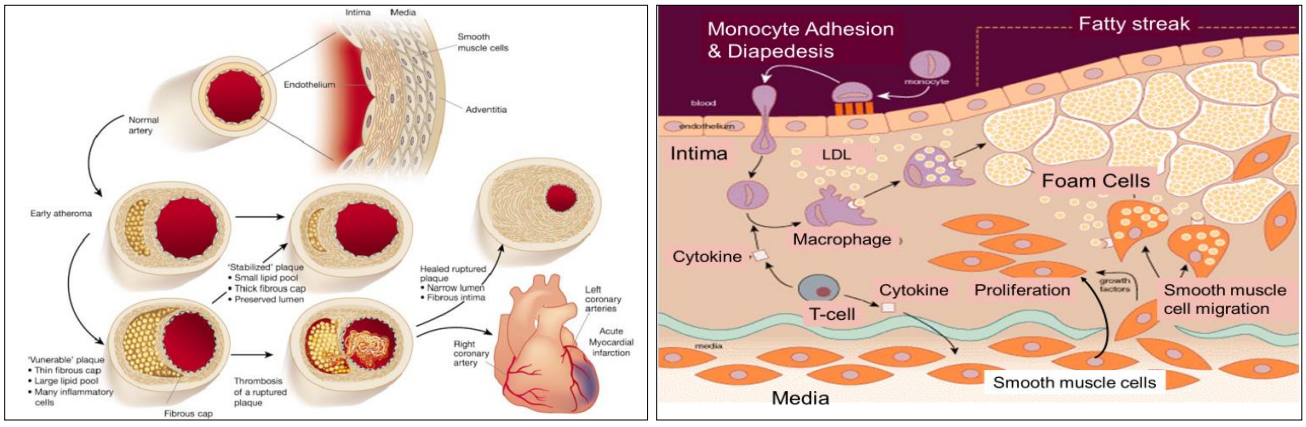

Fig 1: The evolution of an atheromatous plaque

Pathophysiology

Mechanisms that account for a minority of fatal coronary thromboses include superficial erosion, intraplaque hemorrhage, and the erosion of a calcified nodule. Thus, physical disruption of the atherosclerotic plaque accounts for almost all acute coronary thromboses. Disrupted plaques provoke thrombosis in several ways. First, contact with collagen in the plaque's extracellular matrix can trigger platelet activation. Second, TF produced by macrophages and SMCs activates the coagulation cascade. The disrupted plaque thereby represents a "solid-state" stimulus to both thrombosis and coagulation; these pathways reinforce each other, as thrombin generation amplifies the activation of platelets and other cells in the lesion. On version of fibrinogen to fibrin and release of von Willebrand factor from activated platelets can provide the cross-linking molecular bridges between platelets that yield the dense, 3-dimensional network of platelets entrapped in fibrin characteristic of the "white" arterial thrombus. In addition to the solid state of the disrupted plaque, the "fluid phase" of blood can predispose toward coronary thrombosis. Plasminogen activator inhibitor-1 (PAI-1) extinguishes the body's natural fibrinolytic mechanism that combats the persistence and accumulation of thrombi by inhibiting urokinase-like and tissue-type plasminogen activators. Circulating levels of PAI-1 increase in diabetes and obesity, and mediators of hypertension such as angiotensin II can augment PAI-1 expression by various cell types. Furthermore, disrupted plaques can elaborate particulate TF, which can heighten the thrombogenicity of blood. These fluid-phase changes led to the concept of the "vulnerable patient," thus augmenting our appreciation of the so-called "vulnerable plaque". In the context of ACS, the distal embolization of TF-rich debris spewing into the bloodstream from the core of the suddenly disrupted plaque may promote distal thrombosis in the microcirculation. uch distal embolization explains in part the "no-reflow" phenomenon that can complicate both spontaneous and iatrogenic plaque disruption and prevent effective reperfusion of the distal microcirculation [3].

Coronary heart disease may leads to angina. Angina causes the following feeling across the chest.

Squeezing, pressure, heaviness, tightening, burning, aching. Angina might also cause the following symptoms: Indigestion, heart burn, weakness, sweating, nausea, cramping.

Diagnosis

ECG may show evidence of previous myocardial infarction. The most convincing ECG evidence of myocardial ischaemia is obtained by demonstrating reversible ST segment depression or elevation with or without T wave inversion at the time the patient is experiencing symptoms. Coronary artery calcium scanning with CT is a screening tool that has no role in patients with established CAD in whom the presence of coronary artery calcification is a given. Furthermore, the specificity of the coronary calcium score for obstructive coronary lesions is low. Although CT coronary angiography is showing promise for noninvasive detection of obstructive CAD in major epicardial arteries, it is still limited by a high number of false-positive results (up to 50% with severe calcification and coronary stents), specific patient selection (heart rate must be regular and <70 beats/min; patient must hold breath for 15 seconds), and high dose radiation exposure. Magnetic resonance imaging may be used for stress perfusion or stress wall motion imaging as well as noninvasive coronary angiography. Most heart valve prostheses and vascular stents are compatible with MRI; however, MRI cannot be used in the presence of certain implanted metal objects or medical devices, such as pacemakers or implantable cardioverter defibrillators. However, electronic rhythm management devices and other cardiovascular devices are being developed that could be compatible with MRI. Exercise tolerance test is usually performed using a standard treadmill or bicycle ergometer protocol to ensure a progressive and reproducible increase in workload while monitoring the patients ECG, blood pressure and general condition.

Raynaud's diseases [4, 6]

It is caused by intense vasospasm of peripheral arteries. Raynaud's is a rare disorder that affects the arteries. Arteries are blood vessels that carry blood from your heart to different parts of your body.

Causes: Many causes of Reynaud's disease. Example includes:

- Diseases and conditions that directly damage the arteries or damage the nerves that control the arteries in the hands and feet.
- Repetitive actions that damage the nerves that control the arteries in the hands and feet
- Injuries to the hands and feet
- Exposure to certain chemicals. E.g. beta adrenoceptor, ergotamine and derivatives.
- Medicines that narrow the arteries or affect blood pressure.
- Occupational exposure to vibrating tools and cold.
- Cryoglobulinaemia.
- CREST syndrome.

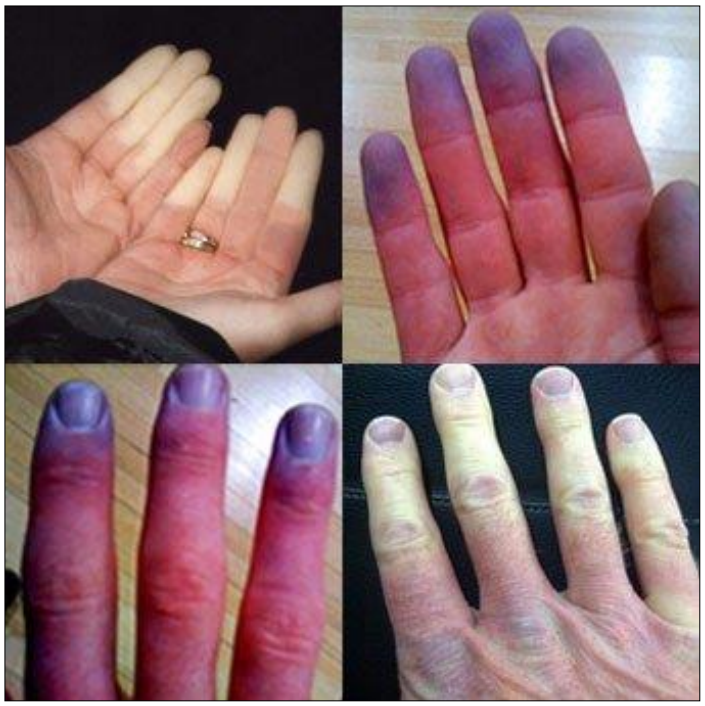

Fig 2: Raynaud's disease

Clinical features

- Turn pale or white and then blue.
- Feel numb, cold, or painful.
- Turn red, throb, tingle, burn, or feel numb as blood flow returns to the affected areas.

Diagnosis

Your doctor will look at your fingers and toes to check the health of your skin and nails and to check blood flow to these areas. Cold stimulation test can be used to trigger Raynaud's symptoms. For this test, a small device that measures temperature is taped to your fingers. Your hands are then exposed to cold—they're usually briefly put into ice water. Your hands are then removed from the cold, and the device measures how quickly your fingers return to their normal temperature. If you have Raynaud's, it may take more than 20 minutes for your fingers to return to their normal temperature. Because results of this type of test are not always consistent, your doctor may do other tests to check for Raynaud's.

Aortic aneurysm [7, 9]

An aortic aneurysm is an enlargement (dilatation) of the aorta to greater than 1.5 times normal size. They usually cause no symptoms except when ruptured. Occasionally, there may be abdominal, back, or leg pain.

Causes

Atheromatous disease – affects ascending or descending aorta, aortitis and collagen vascular diseases – affect thoracic aorta: cystic medial necrosis, marfan's syndrome, ehlers – danlos syndrome.

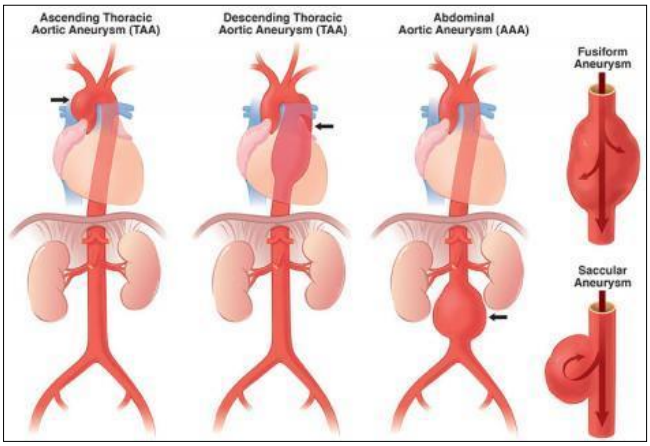

Fig 3: Aortic aneurysm

Clinical features

Most intact aortic aneurysms do not produce symptoms. As they enlarge, symptoms such as abdominal pain and back pain may develop. Compression of nerve roots may cause leg pain or numbness. Untreated, aneurysms tend to become progressively larger, although the rate of enlargement is unpredictable for any individual. Rarely, clotted blood which lines most aortic aneurysms can break off and result in an embolus. Aneurysms can be found on physical examination. Medical imaging is necessary to confirm the diagnosis and to determine the anatomic extent of the aneurysm. Signs are aneurysm may be palpable in abdominal aorta, evidence of widespread vascular disease, stigmata of distal embolisation, haemodynamic collapse (hypotension, tachycardia, shock), with rupture of aneurysm.

Management:

Emergency surgery.

Rheumatic heart disease [10, 12]

Rheumatic heart disease (RHD) is characterised by permanent damage to the valves of the heart that develops as a serious consequence of repeated episodes of acute rheumatic fever (ARF), an autoimmune reaction to a Group A streptococcus (GAS) bacterial infection. Rheumatic heart disease is a chronic cardiac condition with an infectious aetiology, causing high disease burden in low-income settings. Affected individuals are young and associated morbidity is high. However, RHD is relatively neglected due to the populations involved and its lower incidence relative to other heart diseases.

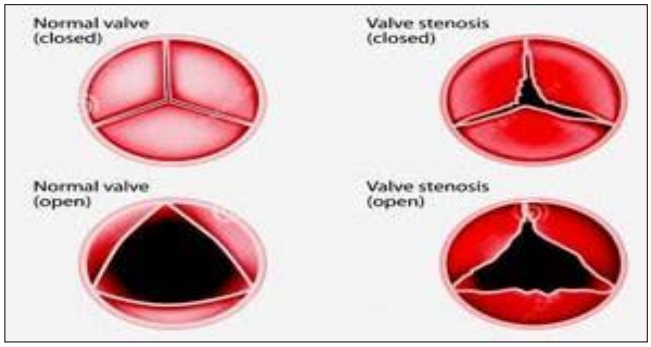

Fig 4: Rheumatic Heart disease

The resulting immune response targets both the bacteria and some of the body's own tissues that contain similar molecules to those in the bacteria, including the heart, skin, joints and nervous system. Rheumatic heart disease results from persisting inflammation of the heart after acute or recurrent episodes of rheumatic fever. It typically affects the valves of the heart, especially the mitral and aortic valves. Chronic inflammation may cause narrowing of the valves resulting in decreased blood flow through the heart or leakage of the valves causing blood to flow in the wrong direction. This may eventually lead to arrhythmias, such as atrial fibrillation, or heart failure, where the heart is unable to pump enough blood to meet the body's needs. Rheumatic fever typically affects children between five and 15 years old for the first time but the effects of rheumatic heart disease often first present in adulthood. Environmental factors such as poor sanitation and crowded living conditions increase the transmission of the bacteria that cause rheumatic heart disease.

Clinical features

Rheumatic fever is a systemic illness typically presenting with fever, anorexia, lethargy and joint pains. Arthritis includes skin rashes (erythema marginatum), carditis (breathlessness, palpitations, chest pain), chorea, subcutaneous nodules and neurological features. Pulses are supporting evidence of preceding streptococcal infection: recent scarlet fever, raised

antistreptolysis O or other streptococcal antibody titre, positive throat culture.

Investigations

Blood tests may be performed in order to detect components of the inflammatory and immune responses that cause rheumatic heart disease. Diagnosis of damage to the heart is primarily achieved by echocardiography, which is an ultrasound imaging of the heart. This can detect abnormally narrow, thickened or leaky valves as well abnormal function of the heart's chambers. Rheumatic fever patients have raised ESR or c reactive protein, leuocytosis, first degree or second degree AV block.

Management

Prevention of rheumatic heart disease centers on early detection and treatment of streptococcal throat infections that cause rheumatic fever. This involves appropriate antibiotic therapy. If moderate or severe heart disease is established, an operation may be necessary to repair or replace the damaged heart valves. This may involve the insertion of a tube and balloon into the heart to dilate a narrowed heart valve through 'balloon valvotomy'. Alternatively, surgical repair or replacement of a damaged heart valve may be performed. The selection of the appropriate procedure is dependent on a number of factors, including the extent of disease of the patient and the level of expertise of the treating doctor. These procedures aim to improve symptoms and quality of life, restore heart function and prevent deterioration of the heart that may lead to complications such as arrhythmias and heart failure.

Valve heart disease

Valvular heart disease (VHD) encompasses a number of common cardiovascular conditions that account for 10% to 20% of all cardiac surgical procedures in the United States. A better understanding of the natural history coupled with the major advances in diagnostic imaging, interventional cardiology, and surgical approaches have resulted in accurate diagnosis and appropriate selection of patients for therapeutic interventions. A thorough understanding of the various valvular disorders is important to aid in the management of patients with VHD. Appropriate work-up for patients with VHD includes a thorough history for evaluation of causes and symptoms, accurate assessment of the severity of the valvular abnormality by examination, appropriate diagnostic testing, and accurate quantification of the severity of valve dysfunction and therapeutic interventions, if necessary. It is also important to understand the role of the therapeutic interventions vs the natural history of the disease in the assessment of outcomes. Prophylaxis for infective endocarditis is no longer recommended unless the patient has a history of endocarditis or a prosthetic valve[13].

AR = aortic regurgitation; AS = aortic stenosis; AVR = aortic valve replacement; CAD = coronary artery disease; CMR = cardiac magnetic resonance imaging; CT = computed tomography; ECG = electrocardiography; LV = left ventricular; MR = mitral regurgitation; MS = mitral stenosis; MV = mitral valve; RV = right ventricular.

Etiology and pathophysiology:

Isolated AR is significantly less common than pure AS. Degenerative and bicuspid aortic valve disease shows a different degree of both regurgitation and left ventricular obstruction; however, stenosis is usually pre-eminent. More frequently, AR is a consequence of aortic dilation and the deformation of the annulus valve. Overall prevalence of significant native AR has been reported in between 2.0% and 2.5% of patients 70 years to 83 years of age, without gender differences.

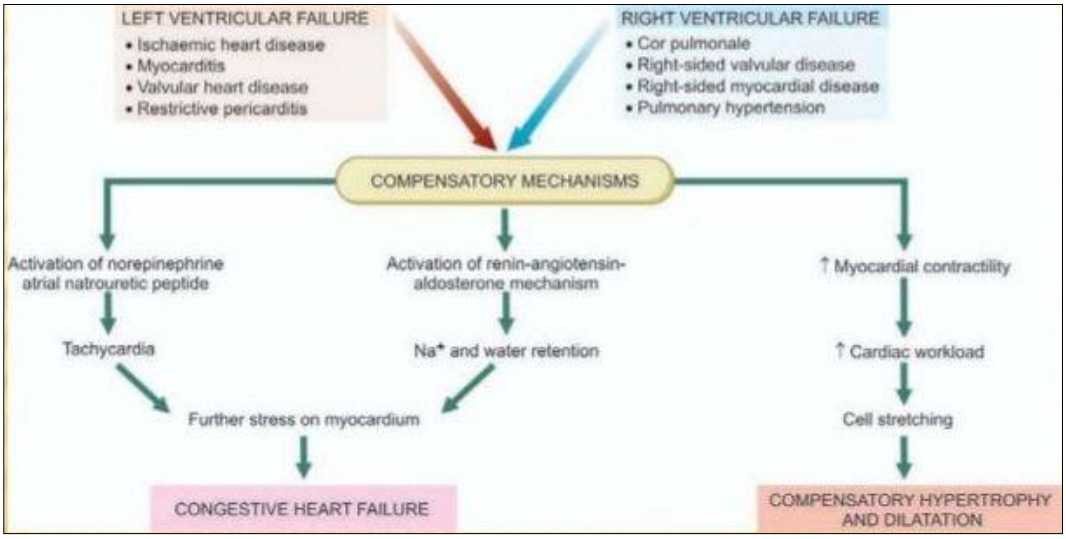

Fig 5: Valves diseases

Although smaller studies reported a higher incidence of up to 13%. Age, aortic valve fibrocalcification, and female sex were considered independent factors related to AR, while several studies failed to find a relationship with arterial hypertension.

Aortic regurgitation

The incidence of clinically significant aortic regurgitation (AR) increases with age, typically peaking in the fourth to sixth decade of life. It is more common in men than women. The prevalence of AR in the Framingham study was reported to be 4.9%, with

regurgitation of moderate or greater severity occurring in 0.5%. AR may be caused by malfunction of the valve leaflets themselves, by dilatation of the aortic root and annulus, or may be due to a combination. Rheumatic disease is still the most common aetiology of AR in developing countries; however, in Western Europe and North America the leading cause of AR is either congenital (particularly due to bicuspid leaflets) or degenerative disease, including annuloaortic ectasia. Understanding the mechanism leading to AR is essential for proper patient management, including the surgical approach. Thus, knowledge of the morphology of the valve leaflets, the annulus and the ascending aorta are essential [14].

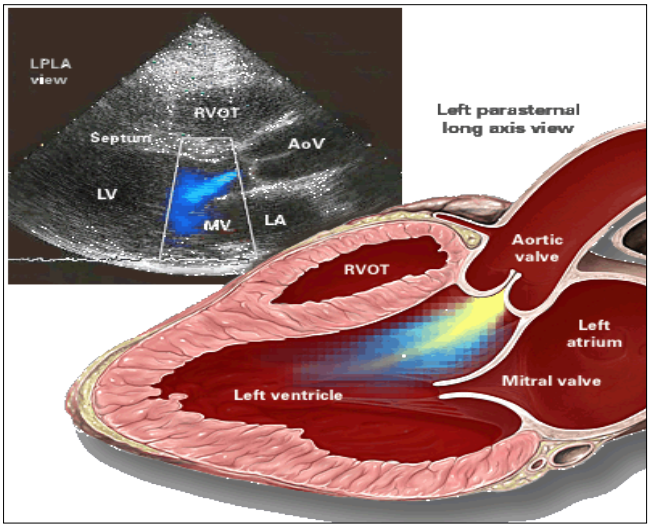

Fig 6: Aortic Regurgitation

Clinical features

Aortic regurgitation clinical features are mild to moderate of aortic regurgitation, awareness of heart beat, palpitations, sever aortic regurgitation, heart failure, angia. Signs are large volume or 'collapsing pulse', bounding peripheral pulses, capillary pulsation in nail beds – quincke's sign, femoral bruit (pistol shot)-duroziez's sign, head nodding with pulse – de musset's sign, early diastolic murmur, systolic murmur of increased stroke volume, Austin flint murmur (soft mid diastolic), thrusting apex, fourth heart sound, enlarge LV, heart failure.

Diagnosis

The ECG may be normal in mild AR. With greater degrees of regurgitation LV hypertrophy with or without strain pattern can be seen. Chest *x* ray shows evidence of LV enlargement. Dilatation of the ascending aorta and aortic knob may be seen. Aneurysmal dilatation of the aorta can be present, particularly in patients in whom the AR is related to primary disease of the aortic wall. Echocardiography presently is the principal tool for diagnosis and grading of AR severity as well as for serial follow-up. Colour Doppler is a highly sensitive and specific technique for detecting AR and provides visualisation of the regurgitant jet. Continuous and pulsed wave Doppler offer additional haemodynamic information and aid quantitation. Importantly, two dimensional echocardiography permits evaluation of LV size and function as well as visualisation of valve structures and of the aorta. Three dimensional echocardiography may play an increasing role in obtaining more precise measurements of ventricular volumes and may offer enhanced images of valve morphology. Aortic root angiography and cardiac magnetic resonance imaging (MRI) are alternative imaging techniques, particularly in rare instances when echocardiography is technically impossible or technically limited. Radionuclide ventriculography can be used to serially assess LV ejection fraction at rest and during exercise [15].

Management

Treatment may be required for underlying conditions such as endocarditis or syphilis. Aortic valve replacement is indicated if aortic regurgitation causes symptoms.

Aortic stenosis

Although valvular heart disease (VHD) is less frequent than coronary artery disease (CAD), heart failure or hypertension, it is of interest for several reasons. Aortic stenosis (AS) is a common valvular heart disease in the Western populations, with an estimated overall prevalence of 3% in adults over 75 years. To understand its patho-biological processes represents a priority. In elderly patients, AS usually involves trileaflet valves and is referred to as degenerative calcific processes. Rheumatic fever as a cause of AS already had begun to wane in developed countries and was replaced pathogenetically by degenerative calcific disease. The ambiguous term "degenerative" suggested that AS stemmed from wear and tear on the valve over time, perhaps explaining its greater incidence in older patients. Although calcification of the aortic valve is a disease of the elderly population, there is evidence that it is not simply a consequence of aging [16].

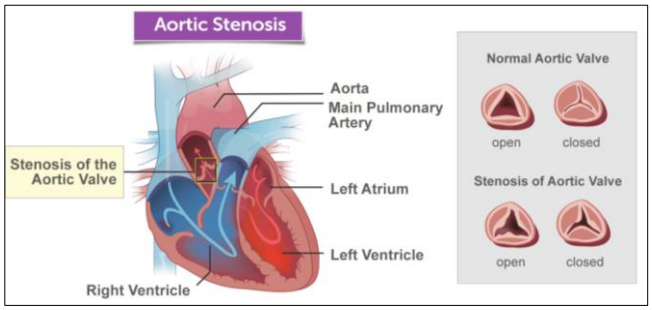

Fig 7: Aortic stenosis

Causes

In infants, children, adolescents are congenital aortic stenosis, congenital subvalvular aortic stenosis, congenital supravalvular aortic stenosis. In young adults to middle aged are calcification and fibrosis of congenitally bicuspid aortic valve, rheumatic aortic stenosis. In middle aged to elderly causes are calcification of bicuspid valve, senile degenerative aortic stenosis, rheumatic aortic stenosis.

Clinical features

Aortic stenosis are exertional dyspnoea, pulmonary oedema, angina, exertional syncope, sudden death. Signs of aortic stenosis are ejection systolic murmur, slow rising crotid pulse, reduced pulse pressure, LVH, thrusting left ventricle, signs of left ventricular failure (crepitations, pulmonary oedema).

Diagnosis

Electrocardiogram (ECG)-triggered CT scan of the heart and the whole aorta, including femoral and subclavian arteries, is performed. Not only can aortic annulus size be studied using MSCT but also leaflet and annulus calcification. The latter can be removed during surgery but, if present, might stand in the way of TAVI. Other important characteristics to be taken into account are distances between the annulus and the coronary ostia that could differ from standard and could result in ostial occlusion after implantation. On the other hand, left ventricular outflow tract (LVOT) and proportions of the ascending aorta are mandatory to achieve a precise and safe implantation. Moreover, the peripheral access site and the descending aorta can be evaluated for anomalies such as major calcification, stenosis, and other factors that could hinder the procedure. MSCT is now an essential tool in terms of access site evaluation, prosthesis sizing, and reducing the paravalvular leakage and risk of complications.

Management

Patients with symptomatic aortic stenosis and a valve gradient indicative of moderate or severe stenosis should have aortic valve replacement.

Mitral stenosis

Mitral stenosis (MS) is a form of valvular heart disease. Mitral stenosis is characterized by narrowing of the mitral valve orifice. Today, the most common cause of mitral stenosis is rheumatic fever, but the stenosis usually appears clinically relevant only after several decades.

Causes

The most common cause of mitral stenosis is rheumatic fever. Uncommon causes of mitral stenosis are calcification of the mitral valve leaflets and congenital heart disease. Other causes of mitral stenosis include infective endocarditis, mitral annular calcification, endomyocardial fibroelastosis, malignant carcinoid syndrome, systemic lupus erythematosus, Whipple disease, Fabry disease, and rheumatoid arthritis. He prevalence of rheumatic disease in developed countries is declining with an estimated incidence of 1 in 100,000. The prevalence is higher in developing nations than in the United States. In Africa, for example, the prevalence is 35 cases per 100,000. Rheumatic mitral stenosis is more common in females. The onset is usually between the third and fourth decade of life [17].

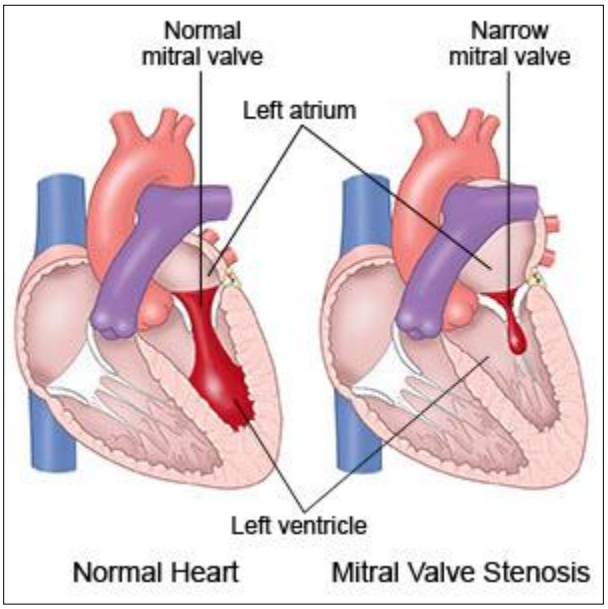

Fig 8: Mitral stenosis

Clinical features

Mitral stenosis is exertional dyspnoea, nocturnal dyspnoea, cough, ankle/leg oedema, abdominal swelling (right heart failure), acute pulmonary oedema symptoms, secondary to arterial/venous emboli symptoms (stoke, haemoptysis, chest pain). signs of mitral stenosis are atrial fibrillation, mitral facies, loud 1st heart sound, opening snap mid diastolic murmur, raised pulmonary capillary pressure crepitations, pulmonary oedema, effusions, pulmonary hypertension signs, RV heave, loud P2.

Diagnosis

Mitral stenosis is evaluated using noninvasive and invasive measures. Noninvasive tests are the electrocardiogram (ECG), chest x-ray, echocardiogram, and exercise echocardiogram. An invasive test for mitral stenosis would include a cardiac catheterization. On the ECG, the P wave changes suggest left atrial enlargement. A presence of right axis deviation and right ventricular hypertrophy suggest severe pulmonary hypertension. ECG frequently detects atrial arrhythmias such as atrial fibrillation. On the chest x-ray, the early stages of mitral stenosis findings are normal heart size, straightening of the left border of the cardiac silhouette, prominent main pulmonary arteries, dilatation of the upper pulmonary veins, and displacement of the esophagus by an enlarged left atrium. During the severe chronic stage of mitral stenosis, the chest x-ray will have enlargement of all the chambers, pulmonary arteries, and pulmonary veins.

Management

Mitral stenosis is by mitral valvotomy, balloon valvuloplasty or mitralvalve replacement. Tricuspid stenosis:

Tricuspid valve stenosis (TS) is rare, affecting less than 1% of patients in developed nations and approximately 3% of patients worldwide. Detection requires careful evaluation, as it is almost always associated with left-sided valve lesions that may obscure its significance. Primary TS is most frequently caused by rheumatic valvulitis. Tricuspid valve stenosis is usually progressive when due to rheumatic disease or carcinoid, versus a fixed stenosis in the setting of congenital abnormalities. Moreover, most stenotic tricuspid valves have some element of tricuspid regurgitation. This is in contrast to purely regurgitant tricuspid valves, which have no element of TS. Stenotic tricuspid valves always demonstrate structural abnormalities, such as fibrous thickening of the leaflets or subvalvular mural plaque as seen in carcinoid. Each etiology of TS has its own distinct pattern of leaflet and chordal pathology [18].

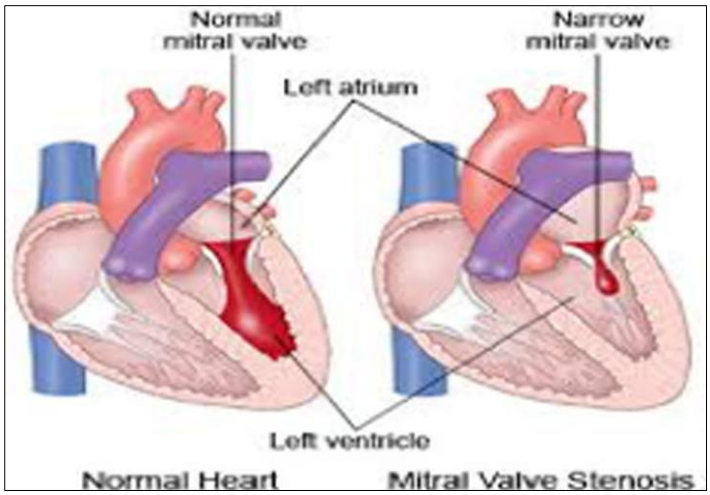

Fig 9: Tricuspid stenosis

Tricuspid stenosis is an uncommon valvular abnormality commonly associated with other valvular lesions. Ebstein's anomaly is a rare congenital heart malformation characterized primarily by abnormalities of the tricuspid valve and right ventricle. Endomyocardial fibrosis is a restrictive cardiomyopathy observed in tropical and subtropical regions. It may cause right ventricular distortion with apparent apical displacement of the tricuspid valve, mimicking Ebstein's anomaly. Eosinophilia, rheumatic in origin are the most commonly cited aetiological link in endomyocardial fibrosis.

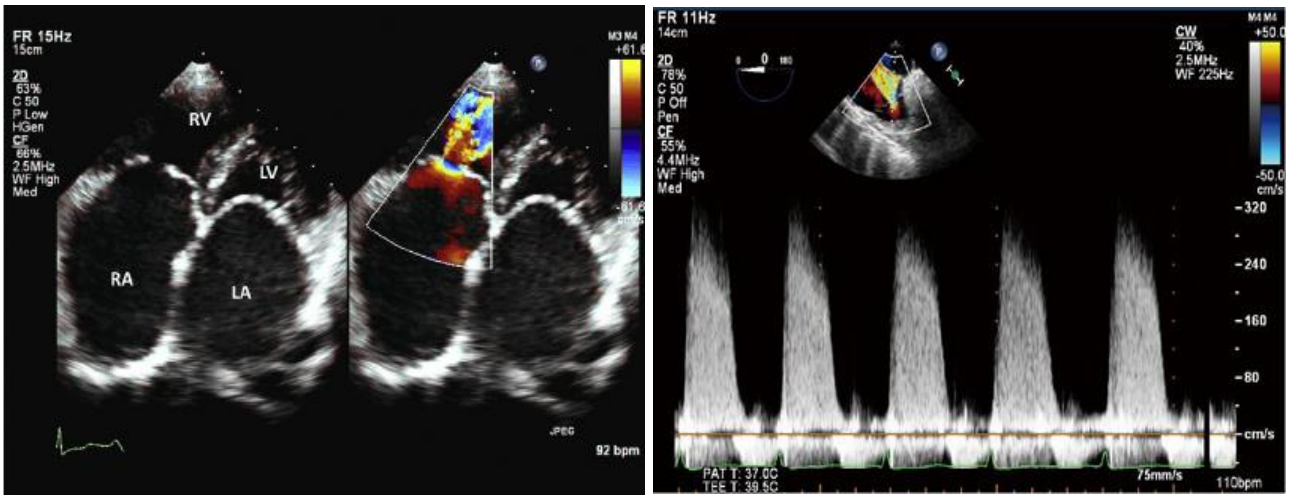

Fig 10: Tricuspid stenosis echocardiogram

Clinical features

Tricuspid valve clinical features are mitral or aortic disease symptoms, abdominal swelling, hepatic discomfort, peripheral oedema, fatigue. Signs are raised JVP, mid diastolic murmur is increased by inspiration, right heart failure – ascities, peripheral oedema.

Management

Tricuspid stenosis is requires surgery. Either replaced or subjected to valvotomy at the time of surgery. Balloon valvuloplasty can be used to treat rare cases of isolated tricuspid stenosis.

Tricuspid regurgitation

ricuspid valve regurgitation (TR) presents challenges to modern day clinical practice. Its natural history is not well understood. Previously, uncertainty existed as to whether TR is an independent factor of outcome or rather a surrogate marker of right ventricular disease and other co-morbid conditions including pulmonary hypertension. However, more recently studies has shown increasing TR severity is associated with worse survival regardless of left ventricular (LV) function and pulmonary hypertension [19].

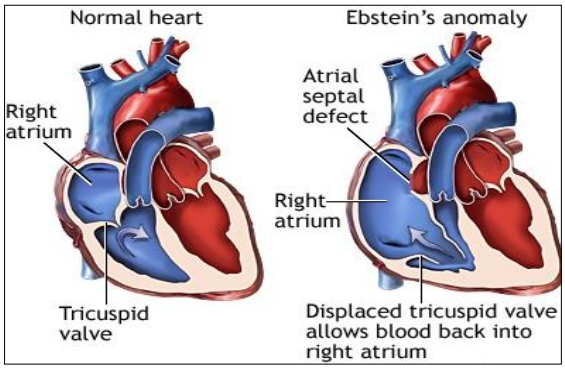

Fig 11: Tricuspid regurgitation

Primary valvular disease accounts for 10% of cases of TR in adults. Patients with congenital disease may have primary TV disease such as in Ebstein's anomaly, atrioventricular defects and myxomatous prolapse. Acquired primary conditions include endocarditis, rheumatic disease, carcinoid or flail leaflet caused by trauma. There is an increasing population of patients with isolated primary TR caused by endomyocardial biopsy or intracardiac leads. Secondary TR results from annular dilation and leaflet tethering leading to malcoaptation.

Clinical features:

TR are rheumatic heart disease, endocarditis, particularly in intravenous drug abusers, ebstein's congenital anomaly(primary) and RV dilation due to chronic LHF, right ventricular infarction, pulmonary hypertension (secondary). Signs of tricuspid regurgitation are raised JVP, large systolic wave in JVP, pansystolic murmur (left sterna edge), systolic hepatic pulsation.

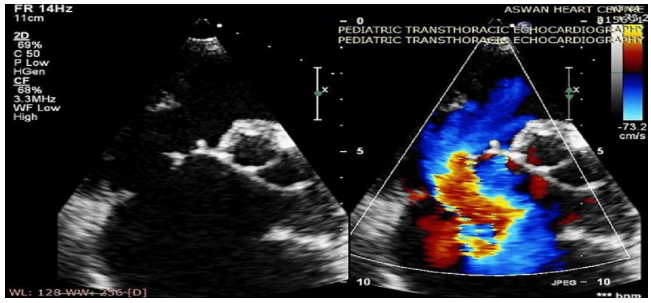

Fig 12: Tricuspid regurgitation echocardiography

Management

Tricuspid regurgitation, which is due to RV dilation, gets better when the cause of right ventricular over load is corrected.

Pulmonary stenosis

Pulmonary stenosis is a condition characterized by obstruction to blood flow from the right ventricle to the pulmonary artery. This obstruction is caused by narrowing (stenosis) at one or more points from the right ventricle to the pulmonary artery. Areas of potential narrowing include thickened muscle below the pulmonary valve, stenosis of the valve itself, or stenosis of the pulmonary artery above the valve. The most common form of pulmonary stenosis is obstruction at the valve itself, referred to as pulmonary valvar stenosis [20].

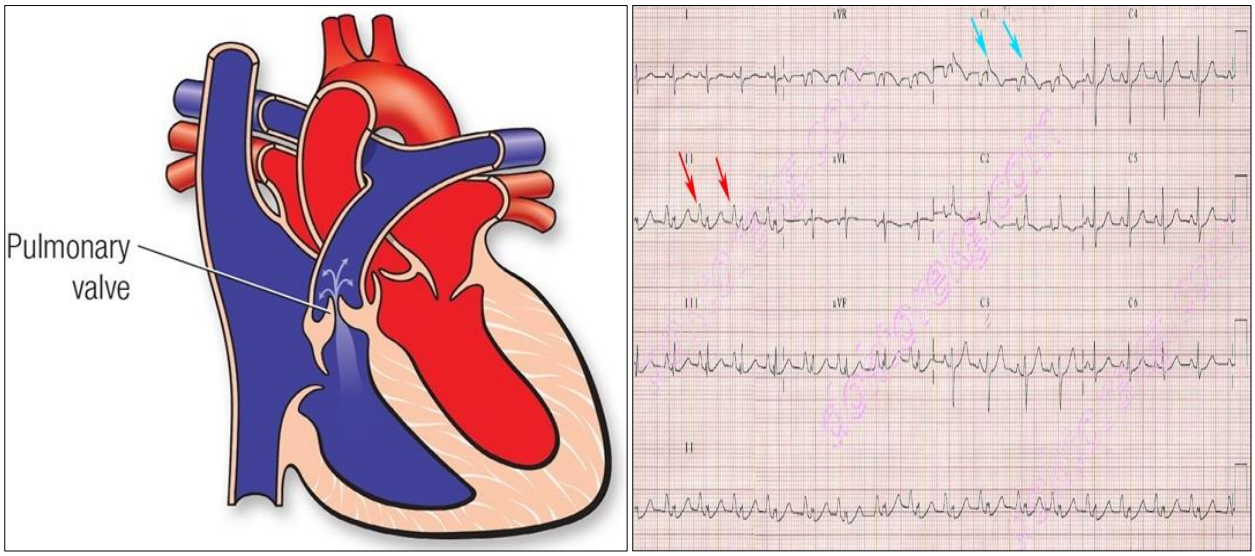

Fig 13: Pulmonary stenosis & ECG changes

Causes

Pulmonary stenosis occurs when the pulmonary valve doesn't grow as it should or the area below or above the valve doesn't grow fully in a baby during the first 8 weeks of pregnancy. Why this happens isn't known. Some congenital heart defects are passed down through families (genetic defects).

Clinical features

Symptoms are right heart failure, carcinoid syndrome and signs are giant a wave in the JVP, RV hypertrophy as well as dilation, systolic murmur, systolic thrill over pulmonary outflow, P2 soft and delayed, valvular PS may have an ejection click.

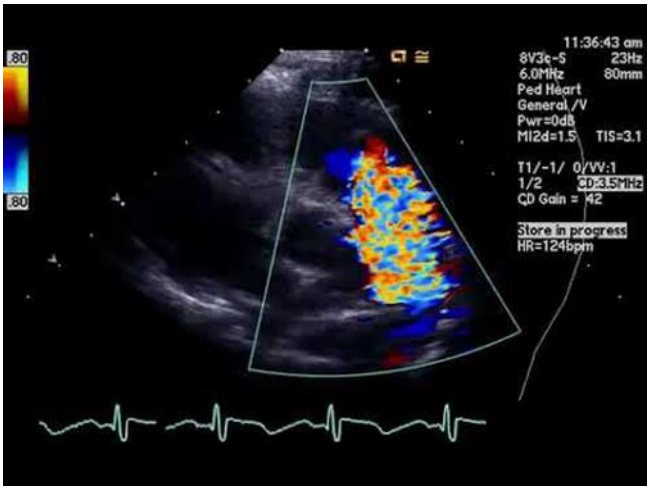

Fig 14: Pulmonary stenosis echocardiogram

Diagnosis

Echocardiogram is typically normal in the presence of mild pulmonary stenosis. With moderate-to-severe pulmonary stenosis the electrocardiogram may show enlargement of the right ventricle and thickening of its muscle. An echo uses sound waves (ultrasound) to make a moving picture of the heart and heart valves. This test is most helpful in diagnosing pulmonary stenosis.

Chest X ray: A chest X-ray may show changes of the heart or pulmonary artery.

ECG: An ECG records the electrical activity of the heart. It shows abnormal rhythms (arrhythmias), and finds heart muscle stress. Although the ECG is often normal, it may show abnormalities that are found with pulmonary stenosis.

Management

Mild pulmonary stenosis often does not need treatment. Moderate or severe stenosis needs repair.

Valvotomy. This is surgery to remove scar tissue from the pulmonary valve leaflets. This lets the valve open as it should.

Balloon dilation or valvuloplasty: A cardiac cath is done as in a diagnostic test. The catheter has a balloon on the tip. When the catheter reaches the narrowed valve or area, the provider inflates the balloon for a short time to stretch it open. Children who have had balloon dilation may need to take antibiotics to prevent heart infection after being discharged from the hospital.

Valvotomy**:** This is surgery to remove scar tissue from the pulmonary valve leaflets. This lets the valve open as it should [20].

Pulmonary regurgitation

Isolated pulmonary regurgitation, in an otherwise normal heart, is well tolerated for decades. However, in a meta-analysis reported in the literature, 29% of patients had developed symptoms within 40 years. Many patients with a right ventricle to pulmonary artery conduit develop a mixture of obstruction and regurgitation across the conduit. However, some of these patients have regurgitation as the dominant lesion, and feature in pulmonary valve replacement series. In pulmonary regurgitation secondary to pulmonary hypertension, the clinical picture is dominated by the primary lung disease or the high pulmonary vascular resistance rather than the volume load. Severe acute pulmonary regurgitation driven by a large duct can occur in neonatal Ebstein's anomaly or following balloon dilation of critical pulmonary stenosis or perforation of valvar pulmonary atresia.

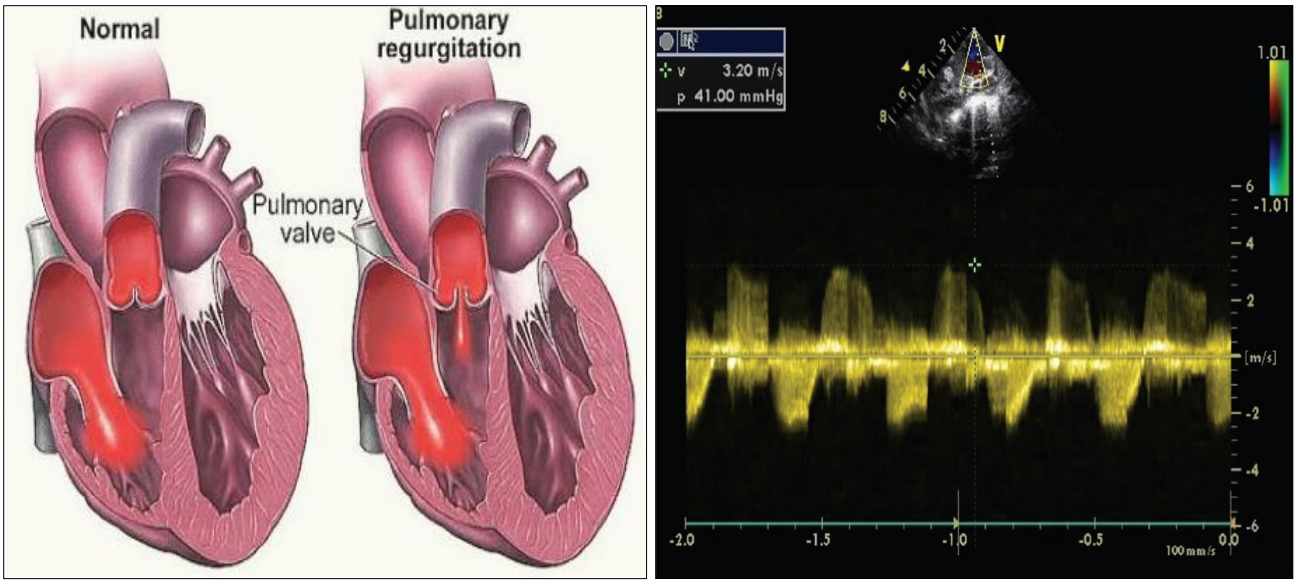

Fig 15: Pulmonary regurgitation & echocardiogram

Causes

Infection endocarditis, complication after surgery to repair tetralogy of fallot, carcinoid syndrome, rheumatic fever and complications after catheterization are rare causes in the India.

Clinical features: murmurs, chest pain, discomfort, fatigue, lightheadedness or fainting.

Management

Pulmonary regurgitation is usually focused on the underlying cause that created the valve problem (e.g. pulmonary hypertension). The need to replace the pulmonary valve is very rare [20].

Infective endocarditis (IE) [21, 23]

It is due to rare, life-threatening disease that has long lasting effects even among patients who survive and are cured. IE disproportionately affects those with underlying structural heart disease and is increasingly associated with healthcare contact, particularly in patients who have intravascular prosthetic material. In the setting of bacteraemia with a pathogenic organism, infected vegetation may form as the end result of complex interactions between invading microorganisms and the host immune system. Once established, IE can involve almost any organ system in the body. The diagnosis of IE may be difficult to establish and a strategy that combines clinical, microbiological and echocardiography results has been codified in the modified Duke criteria. In cases of blood culture-negative IE, the diagnosis may be especially challenging and novel microbiological and imaging techniques have been developed to establish its presence. Once diagnosed, IE is best managed by a multidisciplinary team with expertise in infectious diseases, cardiology and cardiac surgery. Antibiotic prophylaxis for the prevention of IE remains controversial. Efforts to develop a vaccine targeting common bacterial causes of IE are ongoing, but have not yet yielded a commercially available product. IE is a relatively rare but life-threatening disease. In a systematic review of the global burden of IE, crude incidence ranged from 1.5 to 11.6 cases per 100,000 person-years, with high quality data available from only 10 — mostly high-income — countries.

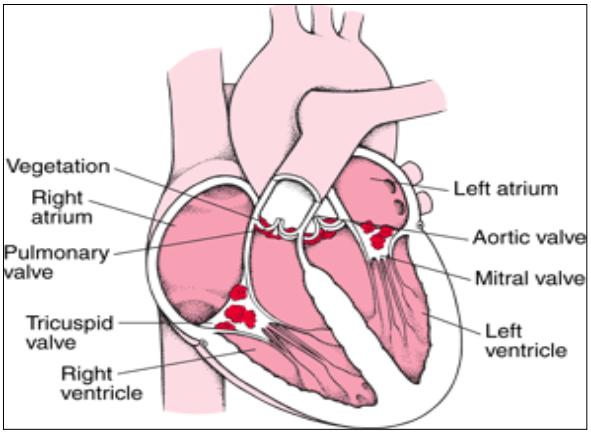

Fig 16: Endocarditis

Causes

It is cause are bacteria like streptococci (viridians 30 -40%), enterococci (10-15%), other streptococci (20-25%).

Pathophysiology

Experimentally, the normal valvular endothelium is resistant to bacterial colonization upon intravascular challenge. Thus, the development of IE requires the simultaneous occurrence of several independent factors: alteration of the cardiac valve surface to produce a suitable site for bacterial attachment and colonization; bacteraemia with an organism capable of attaching to and colonizing valve tissue; and creation of the infected mass or 'vegetation' by 'burying' of the proliferating organism within a protective matrix of serum molecules (for example, fibrin) and platelets.

Clinical features

Clinical features of endocarditis are cerebral emboli, subconjunctival haemorrhages, varying murmurs, conduction disorders, cardiac failure, haematuria, osler's nodes, systemic emboli, petechial rash, loss of pulses, roth's spos in fundi, petechial haemorrhages on mucous membranes and fundi, splenomegaly, digital clubbing, splinter haemorrhages, weight loss, night sweats, fever, tiredness, develops new signs of valve dysfunction or heart failure.

Investigations

The diagnosis of IE typically requires a combination of clinical, microbiological and echocardiography results. Historically, and as is probably still the case in resource-limited settings, IE was diagnosed clinically based on classic findings of active valvulitis (such as cardiac murmur), embolic manifestations and immunological vascular phenomena in conjunction with positive blood cultures. These manifestations were the hallmarks of subacute or chronic infections, most often in young patients with rheumatic heart disease. In the modern era in developed countries, however, IE is usually an acute disease with few of these hallmarks because the epidemiology has shifted towards healthcare-associated IE, often with early presentations due to *S. aureus.* Blood culture is the most important initial laboratory test in the workup of IE. Bacteraemia is usually continuous and the majority of patients with IE have positive blood cultures. Echocardiography is the second cornerstone of diagnostic efforts and should be performed in all patients in whom IE is suspected. Transthoracic echocardiography (TTE) may enable visualization of vegetations in many patients. These include 3D TEE, cardiac CT, cardiac MRI and F-fluorodeoxyglucose PET-CT.

Blood culture positivity for either of the following:

- Typical microorganism (viridans group streptococci, *S. gallolyticus*, HACEK organisms, *S. aureus*, community acquired enterococci in the absence of a primary focus) from 2 separate blood cultures.
- Persistent bactaeremia (two positive cultures >12 hours apart or three positive cultures or a majority of ≥4 culture positive results >1 hour apart).

Serology

Single positive blood culture for *C. burnetii* or antiphase 1 IgG antibody titre of ≥1:800.

Thus, IE diagnosis cannot be made on the basis of a single symptom, sign or diagnostic test. Rather, the diagnosis requires clinical suspicion, most commonly triggered by systemic illness in a patient with risk factors, followed by evaluation according to

the diagnostic schema outlined in the modified Duke criteria. It is worth keeping in mind that the Duke criteria were originally developed to facilitate epidemiological and clinical research efforts and the application of the criteria to the clinical practice setting is more difficult.

Management

In the modern era, management of IE typically requires a multidisciplinary team including, at a minimum, an infectious disease specialist, a cardiologist and a cardiac surgeon. All patients should receive antimicrobial therapy and a subset may benefit from cardiovascular surgical intervention.

Cardiomyopathy [24, 26]

It is a genetic disorder of cardiac myocytes that is characterized by cardiac hypertrophy, unexplained by the loading conditions, a non-dilated left ventricle and a normal or increased ejection fraction. Cardiac hypertrophy is usually asymmetric with greatest involvement most commonly of the basal interventricular septum subjacent to the aortic valve. It is occasionally restricted to other myocardial regions, such as the apex, the mid-portion as well as the posterior wall of the left ventricle. At the cellular level, cardiac myocytes are hypertrophied, disorganized, and separated by areas of interstitial fibrosis.

A diverse array of mechanisms, mirroring the diversity of the causal genes and mutations, are implicated in the pathogenesis of HCM. The mechanistic events in HCM might be categorized into four sets of interlocking mechanisms.

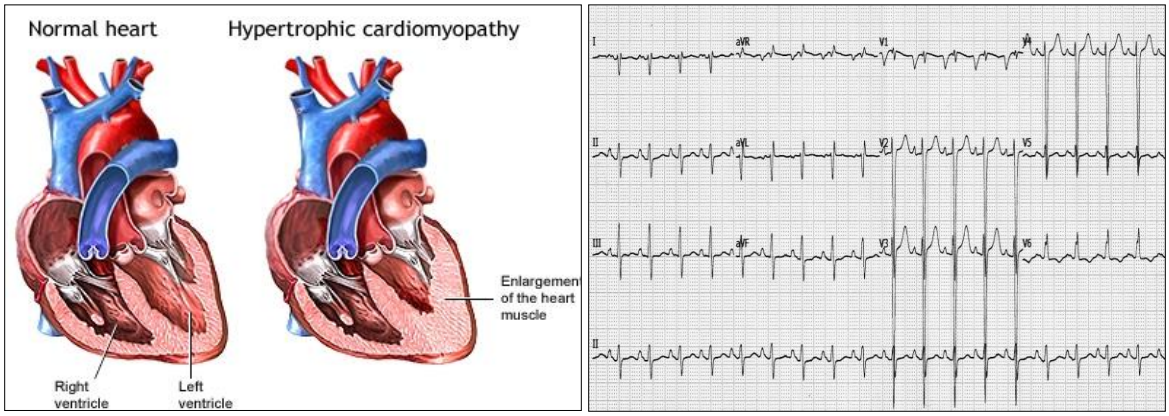

Fig 17: Cardiomyopathy & ECG

The primary defect is the mutation. Initial or proximal phenotypes are defined as those resulting from the direct effects of the mutations on the structure and function of the sarcomere proteins. The intermediary (or secondary) phenotypes include the molecular changes that occur in response to the changes in the sarcomere protein structure and function. Examples of the latter include altered gene expression and activation of the signaling pathways, such as the MAPK and TGFB1 pathways. The tertiary effects are the ensuing histological and pathological phenotypes, which are the consequence of perturbation of a myriad of secondary molecular events in the myocardium, such as activation of the hypertrophic signaling pathways. These molecular and histological changes lead to the clinical phenotypes of HCM (quaternary). It is important to note that there is a mechanistic distinction between cases of HCM caused by sarcomere protein mutation and the phenocopy conditions, since ventricular hypertrophy in the latter may, at least in part, result from storage of material, such as glycogen and in part because of functional defects in myocytes, such as impaired contraction.

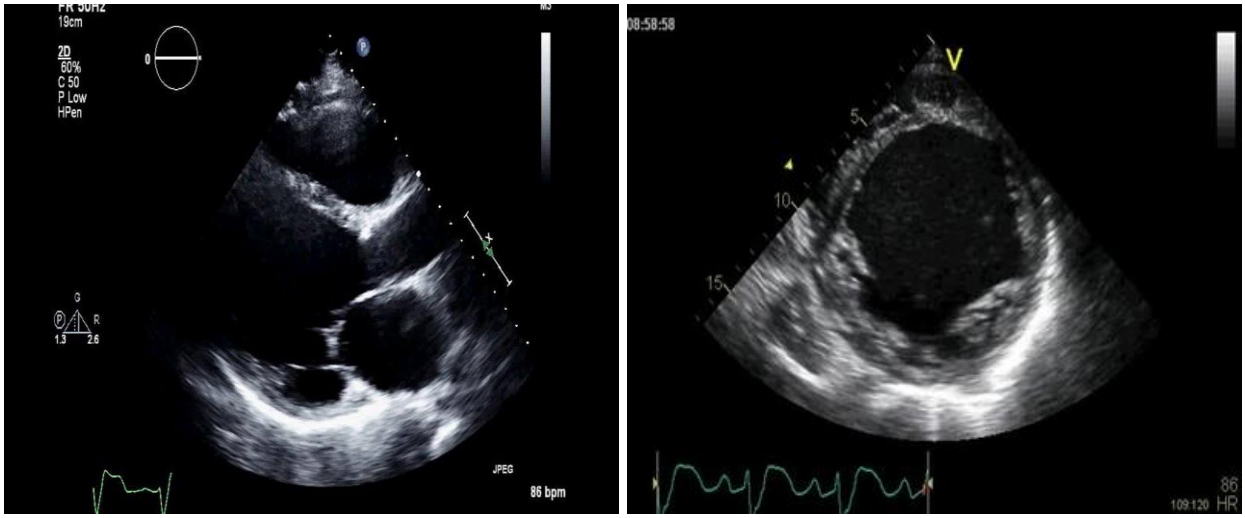

Fig 18: Cardiomyopathy echocardiogram

Clinical features

Angina on effort, dyspnoea on effort, syncope, sudden death. Signs of Cardiomyopathy is jerky pulse, palpable left ventricular hypertrophy, double impulse at the apex, mid systolic murmur at the base, pansystolic murmur, signs of left ventricular out flow tract obstruction which may be augmented by standing up, inotropes and vasodilators.

Diagnosis

ECG in patients with idiopathic DCM has no specific diagnostic role, and abnormalities ranging from isolated T wave and ST segment changes to septal pathological Q waves, wide QRS complex in patients with LV fibrosis might be present. Prolongation of atrioventricular (AV) conduction, and bundle branch block can be observed. Echocardiography in DCM has characteristic patterns, although it is not possible to make differential diagnosis by echocardiography between idiopathic and other secondary LV dilation with dysfunction. M-mode echocardiography shows LV dilation with diffuse hypokinetic walls. Although cardiomyopathy is diffuse pathology, there may be segmental differences of the degree of hypokinesis revealed by two-dimensional echocardiography, which causes difficulties for differentiation from ischemic cardiomyopathy. Ventricular dilation usually is not accompanied by sufficient hypertrophy, which causes increase of volume-to-mass ratio.

Management

Beta adrenoceptor antagonists help to relieve angina and sometimes prevent syncopal attacks but no pharmacological treatment is definitely known to improve prognosis.

Pericarditis

Pericarditis is a common disorder caused by inflammation of the pericardium and can occur as an isolated entity or as a manifestation of an underlying systemic disease. It is diagnosed in approximately 0.1% of hospitalized patients and in 5% of patients admitted to the emergency department with noncardiac chest pain [2]. In most patients, the cause of acute pericarditis is thought to be idiopathic because the yield of diagnostic tests to confirm etiology has been relatively low[27].

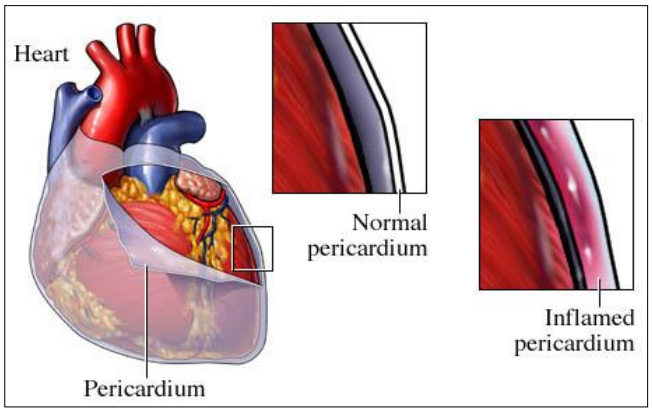

Fig 19: Pericarditis

Etiology

Acute myocardial infraction, viral, uraemia, malignant disease, trauma (blunt chest injury), connective tissue disease (SLE), bacterial infection, rheumatic fever, tuberculosis[28].

Clinical features

Pericarditis symptoms are retroseternal pain and radiates to the shulders and neck. Pain worst by deep breathing, movement, a change of position, exercise and swallowing, low grade fever[29].

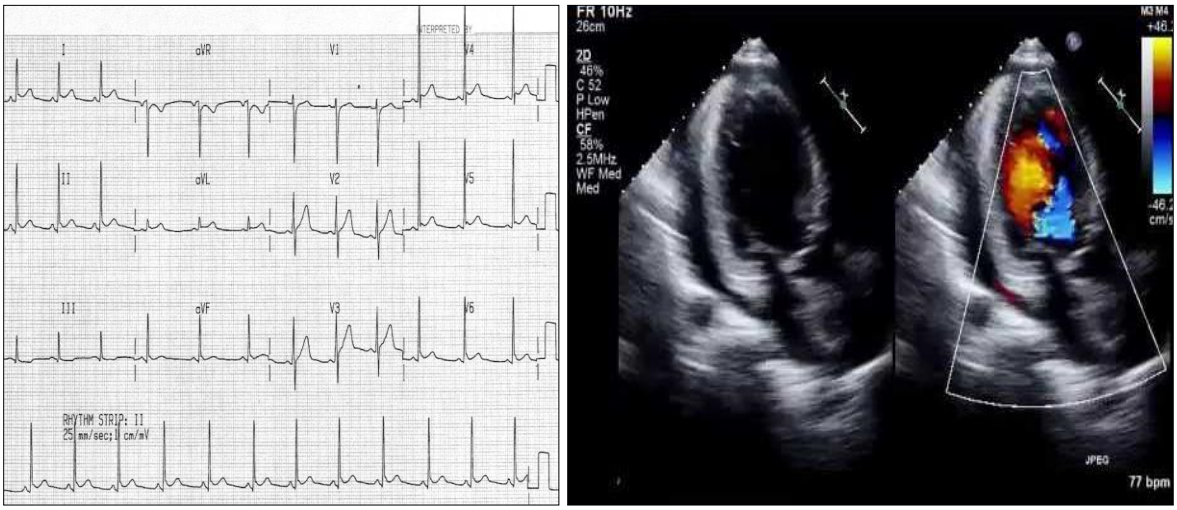

Fig 20: Pericarditis ECG & echocardiogram

Diagnosis

Typical ECG changes in acute pericarditis include wide-spread upward concave ST-segment elevation and PR-segment depression. Transthoracic echocardiography is recommended in patients with suspected acute pericarditis who have evidence of hemodynamic compromise. The finding of a significant pericardial effusion supports the diagnosis and guides further management, especially if there is evidence of cardiac tamponade and a need for emergent pericardiocentesis [30].

Management

The pain can usually be relieved by NSAID's, anti inflammatory agents may be required. Purulent pericarditis requires treatment with antimicrobial therapy, if necessary, surgical drainage.

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