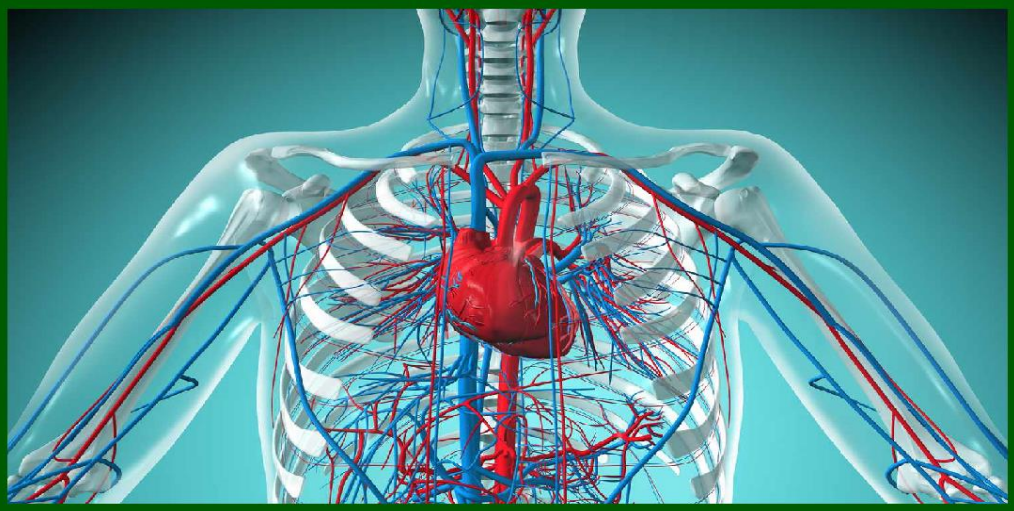
March-24

S.TENTISHEV ASIAN MEDICAL INSTITUTE

KANT KYRGYZSTAN

Inter-Professional Discipline Department

Cardiovascular System



T.S.A.M

" THE STUDENTS ABSTRACT OF MEDICINE"

For

Interprofessional Communication And Partnership In Health Care & Medical Education

3rd

Dr.AFTAB SHEIKH
Senior lecturer
Orthopedic Surgeon & Traumatologist

AsMI
PHARMACOLOGICAL
SOCIETY

S. Tenishev Asian Medical Institute

Department of Interprofessional Disciplines

A collection of works by AzMI students under the guidance of

Senior lecturer Dr.Aftab Sheikh in the specialty

General Medicine & Dentistry

1-Collection of works by AzMI students under the guidance of Senior lecturer ***Dr.Aftab Sheikh*** was approved and recommended in meeting of
The Department of Interprofessional Disciplines ,Protocol No.01 ,dated 5th of September 2023 academic year.
2-***Approved by Head of the Department Ryspekova Altynay Erkinbekovna***
3-The collection consists of 40-45 pages.
4-The collection will be published 7 times in an academic year
5-Release date from the 20th to 30th of the month.
6-Work of students of 3-4-5 years of 5 and 6 year programs as included and recommended

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Aortic Valve Stenosis

OVERVIEW

Aortic valve stenosis — or aortic stenosis — is a type of heart valve disease (valvular heart disease). The valve between the lower left heart chamber and the body's main artery (aorta) is narrowed and doesn't open fully. This reduces or blocks blood flow from the heart to the aorta and to the rest of the body.Treatment of aortic stenosis depends on the severity of the condition. You may need surgery to repair or replace the valve. Without treatment, severe aortic valve stenosis can lead to death.

Aortic valve disease

Cardiologist explains aortic valve disease, including aortic stenosis.
Show transcriptfor video Aortic valve disease

Symptoms

Aortic valve stenosis ranges from mild to severe. Symptoms generally occur when narrowing of the valve is severe. Some people with aortic valve stenosis may not have symptoms for many years.

Symptoms of aortic valve stenosis may include:

- ✓ An irregular heart sound (heart murmur) heard through a stethoscope
- ✓ Chest pain (angina) or tightness with activity
- ✓ Feeling faint or dizzy or fainting with activity
- ✓ Shortness of breath, especially with activity
- ✓ Fatigue, especially during times of increased activity
- ✓ Rapid, fluttering heartbeat (palpitations)
- ✓ Not eating enough (mainly in children with aortic valve stenosis)
- ✓ Not gaining enough weight (mainly in children with aortic valve stenosis)

Aortic valve stenosis may lead to heart failure. Heart failure symptoms include fatigue, shortness of breath, and swollen ankles and feet.

Causes

To understand the causes of aortic valve stenosis, it may be helpful to know how the heart and heart valves typically work.
The heart has four valves that keep blood flowing in the correct direction:

- ✓ Aortic valve
- ✓ Mitral valve
- ✓ Tricuspid valve
- ✓ Pulmonary valve

Each valve has flaps (cusps or leaflets) that open and close once during each heartbeat. Sometimes, the valves don't open or close properly. If a valve doesn't fully open or close, blood flow is reduced or blocked.

In aortic valve stenosis, the valve between the lower left heart chamber (left ventricle) and the aorta does not open completely. The area through which blood moves out of the heart to the aorta is narrowed (stenosis).
When the aortic valve opening is narrowed, the heart must work harder to pump enough blood into the aorta and to the rest of the body. The extra work of the heart can cause the left ventricle to thicken and enlarge. Eventually the strain can cause a weakened heart muscle and can ultimately lead to heart failure and other serious problems.
Aortic valve stenosis causes include:

Congenital heart defect. Some children are born with an aortic valve that has only two cusps (bicuspid aortic valve) instead of three (tricuspid aortic valve). Rarely, an aortic valve may have one (unicuspid) or four (quadricuspid) cusps.

Having a congenital heart defect such as a bicuspid aortic valve requires regular medical checkups. The valve condition may not cause any problems until adulthood. If the valve begins to narrow or leak, it may need to be repaired or replaced.

Calcium buildup on the valve (aortic valve calcification). Calcium is a mineral found in the blood. As blood repeatedly flows over the aortic valve, calcium deposits can build up on the heart valves.

The calcium deposits may never cause any problems. Aortic valve stenosis that's related to increasing age and calcium deposit buildup usually doesn't cause symptoms until age 70 or 80. However, in some people — particularly those with congenital aortic valve defects — calcium deposits result in stiffening of the valve cusps at a younger age.

Rheumatic fever. This complication of untreated strep throat can damage the heart valves. It may cause scar tissue to form on the aortic valve. Scar tissue can narrow the aortic valve opening or create a rough surface on which calcium deposits can collect.

Rheumatic fever may damage more than one heart valve, and in more than one way. While rheumatic fever is rare in the United States, some older adults had rheumatic fever as children.

Risk factors

Risk factors of aortic valve stenosis include:

- Older age
- Certain heart conditions present at birth (congenital heart defects), such as a bicuspid aortic valve
- Chronic kidney disease
- Having heart disease risk factors, such as diabetes, high cholesterol and high blood pressure
- History of infections that can affect the heart, such as rheumatic fever and infective endocarditis
- History of radiation therapy to the chest

Complications

Aortic valve stenosis can cause complications, including:

- Heart failure
- Stroke

- Blood clots
- Bleeding
- Irregular heart rhythms (arrhythmias)
- Infections that affect the heart, such as endocarditis
- Death

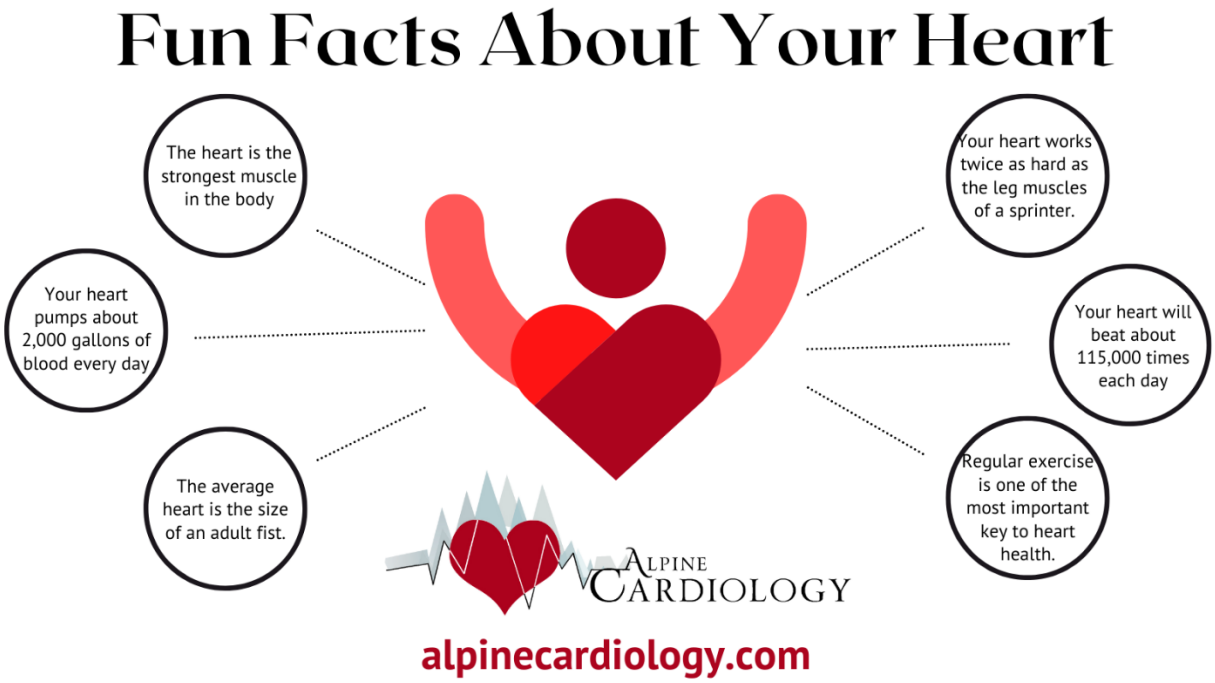
Prevention

Taking steps to prevent rheumatic fever. See your health care provider when you have a sore throat. Strep throat can usually be easily treated with antibiotics. Untreated strep throat can develop into rheumatic fever. Rheumatic fever is more common in children and young adults.

- **Keeping the heart healthy.** Talk to your health care provider about risk factors for heart disease and how to prevent or manage them. They include high blood pressure, obesity and high cholesterol levels. These risk factors may be linked to aortic valve stenosis.
- **Taking care of the teeth and gums.** There may be a link between infected gums (gingivitis) and infected heart tissue (endocarditis). Inflammation of heart tissue caused by infection can narrow arteries and worsen aortic valve stenosis.

If you have aortic valve stenosis, your health care provider may recommend that you limit strenuous activity to avoid overworking your heart.

REFERENCE:- https://www.mayoclinic.org/diseases-conditions/aortic-stenosis/symptoms-causes/syc-20353139



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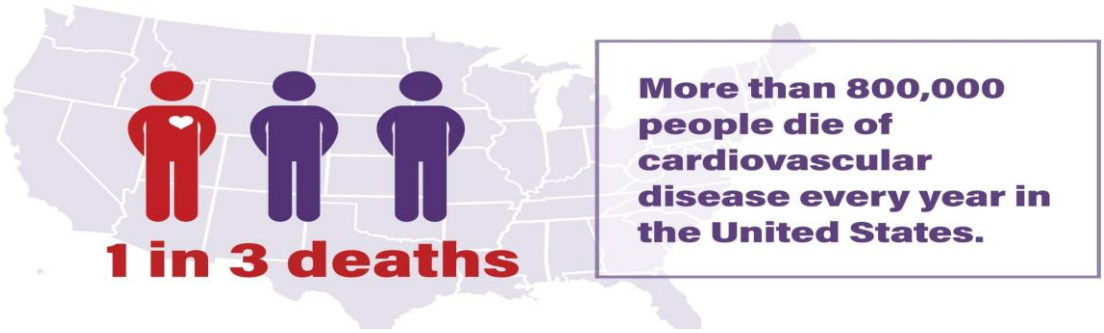
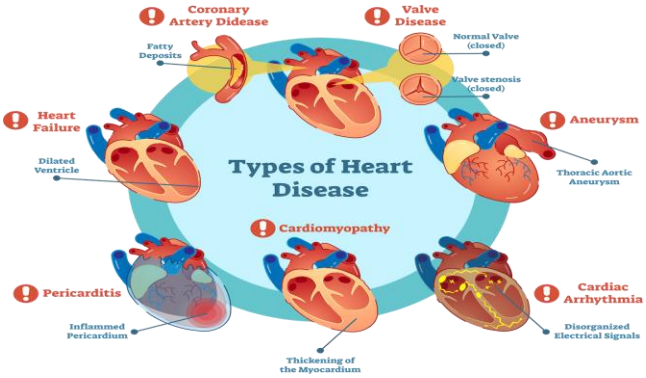
Cardiovascular Diseases

Cardiovascular disease is the term for all types of diseases that affect the heart or blood vessels, including coronary heart disease (clogged arteries), which can cause heart attacks, stroke, heart failure, and peripheral artery disease.



1. **Coronary Artery Disease (CAD):** A leading cause of heart attacks, CAD results from the buildup of plaque in coronary arteries, restricting blood flow to the heart.
2. **Myocardial Infarction (Heart Attack)**:Occurs when blood flow to a part of the heart muscle is blocked, leading to damage or death of the affected tissue.
3. **Heart Failure**: A condition where the heart is unable to pump blood effectively, causing symptoms like shortness of breath and fatigue.
4. **Hypertension (High Blood Pressure)**: Persistent elevated blood pressure that can lead to serious complications, including heart disease.
5. **Arrhythmias**: Irregular heart rhythms, which can include conditions like atrial fibrillation, bradycardia, and tachycardia.
6. **Valvular Heart Disease**:Disorders affecting the heart valves, impairing their function and leading to issues like regurgitation or stenosis.
7. **Cardiomyopathy**: Diseases of the heart muscle, resulting in impaired pumping ability.
8. **Peripheral Artery Disease (PAD)**:A condition where narrowed arteries reduce blood flow to limbs, often causing pain and mobility issues.
9. **Aortic Aneurysm**: A bulge in the aorta that can rupture, posing a life-threatening risk.
10. **Rheumatic Heart Disease:** Caused by rheumatic fever, it can lead to damage and scarring of the heart valves.
11. **Deep Vein Thrombosis (DVT) and Pulmonary Embolism (PE):** Conditions related to blood clots in veins, with DVT in the legs and PE in the lungs.
12. **Peripheral Vascular Disease (PVD):** Conditions affecting blood vessels outside the heart and brain.

13. **Endocarditis**:Inflammation of the inner lining of the heart chambers and valves, often due to infection.
14. **Congenital Heart Diseases**:Structural abnormalities present at birth affecting the heart's function.
15. **Cardiac Arrest**:Sudden loss of heart function, requiring immediate intervention.
16. **Hyperlipidemia**:Elevated levels of lipids (cholesterol and triglycerides) in the blood, contributing to atherosclerosis.
17. **Pulmonary Hypertension**:High blood pressure in the pulmonary arteries, affecting the lungs.
18. **Cardiac Arrhythmias**:Disorders in the heart's electrical system leading to irregular heartbeats.
19. **Mitral Valve Prolapse (MVP):**A common heart valve disorder where the valve doesn't close properly.
20. **Pericarditis**: Inflammation of the pericardium, the sac surrounding the heart.



21. **Ventricular Septal Defect (VSD):** A congenital heart defect involving a hole in the wall between the heart's lower chambers.
22. **Patent Ductus Arteriosus (PDA):** A congenital condition where a fetal blood vessel, the ductus arteriosus, fails to close after birth.
23. **Cardiac Tamponade**: Compression of the heart due to the accumulation of fluid in the pericardial sac.
24. **Aortic Valve Stenosis**: Narrowing of the aortic valve, impeding blood flow from the heart to the aorta.
25. **Brugada Syndrome**:An inherited cardiac arrhythmia disorder associated with an increased risk of sudden cardiac death.
26. **Bundle Branch Block**: A conduction disorder in the heart's electrical system, affecting the bundle branches.
27. **Atrial Septal Defect (ASD):** A congenital heart defect involving a hole in the wall between the heart's upper chambers.
28. **Leriche Syndrome**: A type of peripheral arterial disease affecting the lower limbs.
29. **Tetralogy of Fallot**: A congenital heart condition characterized by four specific defects.
30. **Takotsubo Cardiomyopathy** (Broken Heart Syndrome):A temporary heart condition often triggered by extreme stress.
31. **Marfan Syndrome**: A genetic disorder affecting the connective tissues, including the heart's valves and aorta.

32. **Raynaud's Disease** :A condition causing narrowing of blood vessels, usually in fingers and toes, in response to cold or stress.
33. **Wolff-Parkinson-White Syndrome (WPW)**: An abnormality of the heart's electrical pathways leading to episodes of rapid heart rate.
34. **Hypertrophic Cardiomyopathy**: Thickening of the heart muscle, making it harder for the heart to pump blood.
35. **Cardiogenic Shock**: A critical condition where the heart cannot pump enough blood to meet the body's needs.
36. **Ischemic Cardiomyopathy**: Weakening of the heart muscle due to reduced blood flow and oxygen delivery.
37. **Postural Orthostatic Tachycardia Syndrome (POTS)**: A disorder characterized by an abnormally increased heart rate when standing up.
38. **Arteriovenous Fistula (AVF):**An abnormal connection between an artery and a vein.
39. **Infective Endocarditis**:Infection of the inner lining of the heart, usually involving heart valves.
40. **Kawasaki Disease**:A childhood illness that affects the blood vessels, including the coronary arteries.
41. **Wegener's Granulomatosis (Granulomatosis with Polyangiitis):**An autoimmune disorder that can involve inflammation of blood vessels.
42. **Coarctation of the Aorta**: A congenital narrowing of the aorta, restricting blood flow to the lower part of the body.
43. **Superior Vena Cava Syndrome**: Obstruction of the superior vena cava, often due to a tumor or blood clot.
44. **Tricuspid Valve Stenosis**: Narrowing of the tricuspid valve, affecting blood flow between the right atrium and ventricle.
45. **Primary Pulmonary Hypertension**: Elevated blood pressure in the pulmonary arteries without an apparent cause.
46. **Bicuspid Aortic Valve:** A congenital condition where the aortic valve has two cusps instead of three.
47. **Peripartum Cardiomyopathy**:Heart failure that occurs during the last month of pregnancy or within five months after giving birth.
48. **Post-Myocardial Infarction Syndrome:**Inflammatory condition following a heart attack, involving chest pain and fever.
49. **Isolated Systolic Hypertension:** Elevated systolic blood pressure without a corresponding increase in diastolic pressure.
50. **Aortic Dissection:** A tear in the inner layer of the aorta, requiring immediate medical attention.
51. **Bradyarrhythmia:** Abnormally slow heart rhythms, including conditions like sinus bradycardia.
52. **Trikuspidalatresie:** A congenital heart defect where the tricuspid valve is missing or abnormally developed.
53. **Prinzmetal's Angina (Variant Angina):** Chest pain caused by coronary artery spasm, often occurring at rest.

54. **Alcoholic Cardiomyopathy:**Weakening of the heart muscle due to long-term alcohol abuse.
55. **Post pericardiotomy Syndrome:** Inflammation of the pericardium following cardiac surgery.
56. **Carotid Artery Disease**: Narrowing or blockage of the carotid arteries, increasing the risk of stroke.
57. **High-Output Heart Failure**:Heart failure resulting from increased cardiac output, often seen in conditions like anemia or hyperthyroidism.
58. **Restrictive Cardiomyopathy:**Stiffening of the heart muscle, restricting its ability to stretch and fill with blood.
59. **Noncompaction Cardiomyopathy**:A rare congenital disorder characterized by prominent trabeculations in the myocardium.
60. **Cardiac Cirrhosis**: Liver damage caused by long-term heart failure, leading to impaired blood flow to the liver.

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Cardiovascular Anatomy

I am Writing articles on cardiovascular anatomy to allows you to share knowledge and educate others about the structure and function of the cardiovascular system. You can explain complex concepts in a simplified way, making it easier for readers to understand and learn.

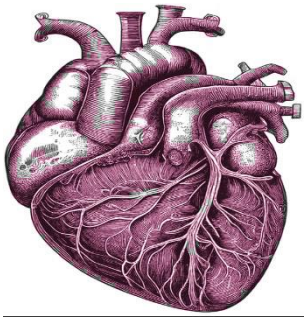
Cardiovascular system

Heart

The hollow, muscular organ that pumps blood through the body of a vertebrate animal by contracting and relaxing. In humans and other mammals, it has four chambers, consisting of two atria and two ventricles.

FUNCTION of CVS

Your heart's main function is to move blood throughout your body. Your heart also:
Controls the rhythm and speed of your heart rate.
Maintains your blood pressure.



Heart location

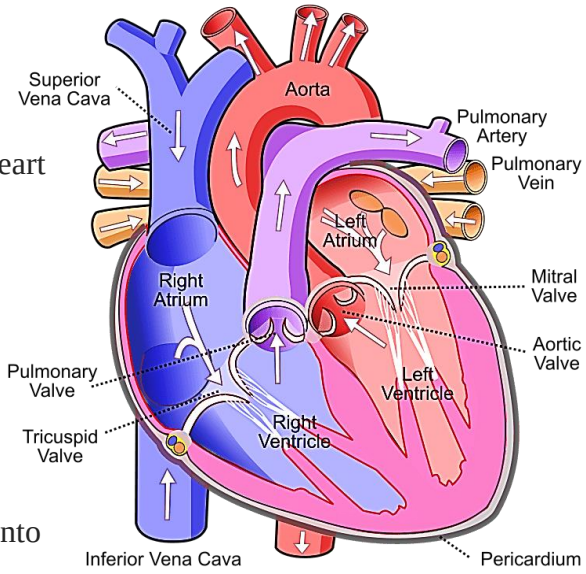
Your heart is located in the front of your chest. It sits slightly behind and to the left of your sternum (breastbone). Your ribcage protects your heart.

Heart WEIGHT

On average, an adult's heart weighs about 10 ounces. Your heart may weigh a little more or a little less, depending on your body size and sex.

How does a heart diagram look like?

The inside and outside of your heart contain components



Parts of the heart

The parts of your heart are like the parts of a house. Your heart has:
Walls.
Chambers (rooms).
Valves (doors).
Blood vessels (plumbing).
Electrical conduction system (electricity).

Heart walls

Your heart walls are the muscles that contract (squeeze) and relax to send blood throughout your body. A layer of muscular tissue called the septum divides your heart walls into the left and right sides.
Your heart walls have three layers:
Endocardium: Inner layer.
Myocardium: Muscular middle layer.

Epicardium: Protective outer layer.
The epicardium is one layer of your pericardium. The pericardium is a protective sac that covers your entire heart. It produces fluid to lubricate your heart and keep it from rubbing against other organs.
Heart chambers Your heart is divided into four chambers. You have two chambers on the top (atrium, plural atria) and two on the bottom (ventricles), one on each side of the heart.
Right atrium: Two large veins deliver oxygen-poor blood to your right atrium. The superior vena cava carries blood from your upper body. The inferior vena cava brings blood from the lower body. Then the right atrium pumps the blood to your right ventricle.
Right ventricle: The lower right chamber pumps the oxygen-poor blood to your lungs through the pulmonary artery. The lungs reload blood with oxygen.
Left atrium: After the lungs fill blood with oxygen, the pulmonary veins carry the blood to the left atrium. This upper chamber pumps the blood to your left ventricle.
Left ventricle: The left ventricle is slightly larger than the right. It pumps oxygen-rich blood to the rest of your body.
Heart valves
Your heart valves are like doors between your heart chambers. They open and close to allow blood to flow through.
The atrioventricular (AV) valves open between your upper and lower heart chambers. They include:
Tricuspid valve: Door between your right atrium and right ventricle.
Mitral valve: Door between your left atrium and left ventricle.
Semilunar (SL) valves open when blood flows out of your ventricles. They include:
Aortic valve: Opens when blood flows out of your left ventricle to your aorta.
Pulmonary valve: Opens when blood flows from your right ventricle to your pulmonary arteries
Blood vessels
Your heart pumps blood through three types of blood vessels:
Arteries carry oxygen-rich blood from your heart to your body's tissues. The exception is your pulmonary arteries, which go to your lungs.
Veins carry oxygen-poor blood back to your heart.
Capillaries are small blood vessels where your body exchanges oxygen-rich and oxygen-poor blood.
Your heart receives nutrients through a network of coronary arteries. These arteries run along your heart's surface. They serve the heart itself.
Left coronary artery: Divides into two branches (the circumflex artery and the left anterior descending.
Circumflex artery: Supplies blood to the left atrium and the side and back of the left ventricle.
Left anterior descending artery (LAD): Supplies blood to the front and bottom of the left ventricle.
Right coronary artery (RCA): Supplies blood to the right atrium, right ventricle, bottom portion of the left ventricle.
Electrical conduction system
Your heart's conduction system is like the electrical wiring of a house. It controls the rhythm and pace of your heartbeat. It includes:
Sinoatrial node: Sends the signals that make your heart beat.
Atrioventricular (AV) node: Carries electrical signals from your heart's upper chambers to its lower ones.
Your heart also has a network of electrical bundles and fibers. This network includes:

Left bundle branch: Sends electric impulses to your left ventricle.
Right bundle branch: Sends electric impulses to your right ventricle.
Bundle of His: Sends impulses from your AV node to the Purkinje fibers.
Purkinje fibers: Make your heart ventricles contract and pump out blood.

Care

You can also make lifestyle changes to keep your heart healthy. You may:
Achieve and maintain a healthy weight for your sex and age.
Drink alcohol in moderation.
Eat a heart-healthy diet with plenty of fruits, vegetables and whole grains.
Exercise moderately for at least 150 minutes per week.

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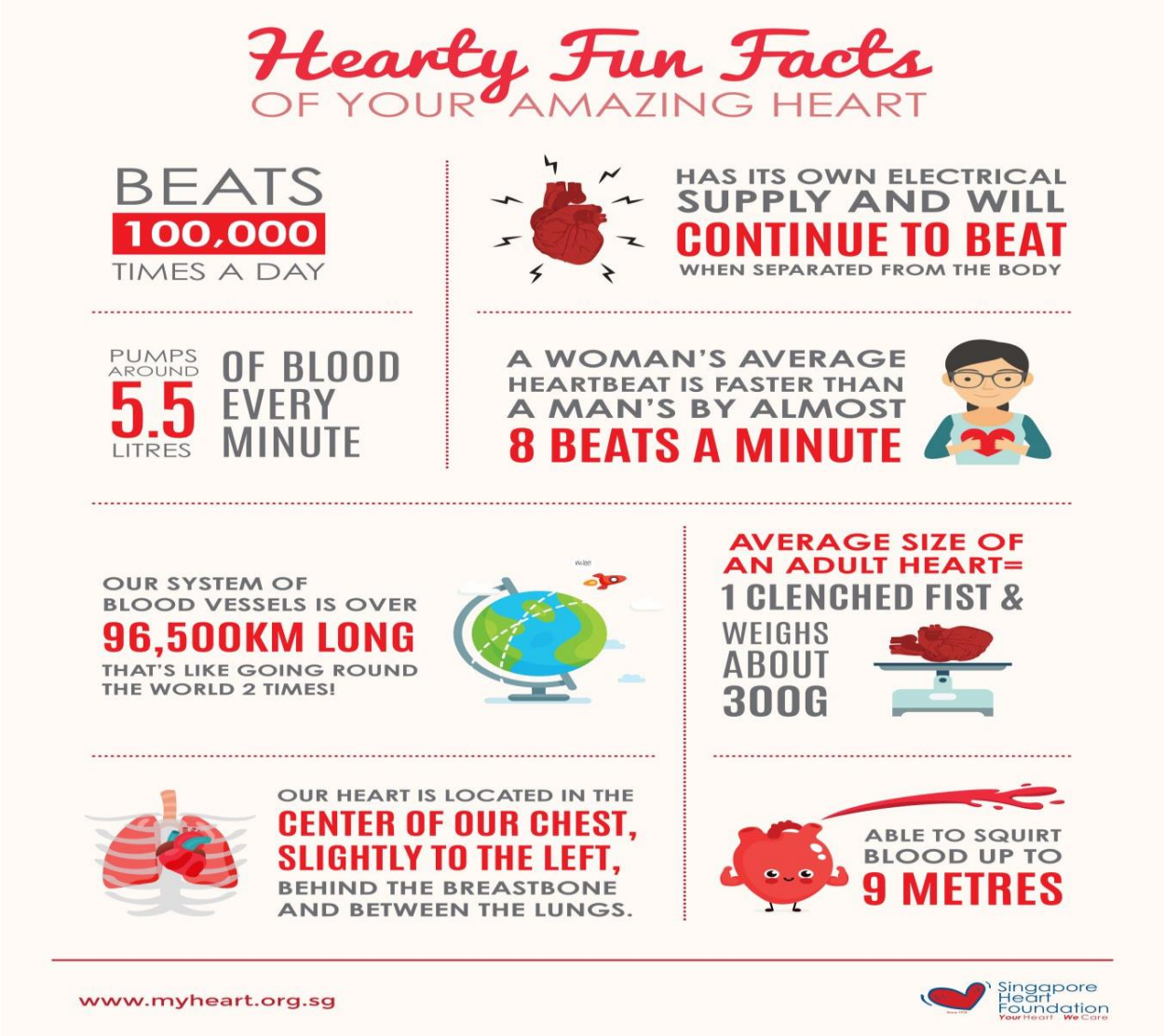

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Session 2018-2024

Rheumatic Fever

INTRODUCTION

I choose this RHEUMATIC FEVER topic to prevent and spread awareness about it. Because many of patients neglect the sore throat and in future suffer with rheumatic fever and rheumatic arthritis.

EPIDEMIOLOGY

Acute Rheumatic Fever can occur at any age, although most case occurs in children 5 to 15 years of age. Over 15 million cases of RHD world wide, with 282000 new cases and 233000 death annually.

RISK FACTOR

Family history
Certain strain of streptococcus bacteria
Environment factor like over crowding ,poor sanitation.

PHATOGENISIS

Upper respiratory infections
↓
Group A beta hemolytic streptococcus bacteria
↓
Molecular mimicry immune response against M proteins/TYPE 2 HYPERSENSITIVITY
↓
Ab production
↓
CD4 TCELLS
↓
Cross react with Cardiac protein
↓
Ab+ complement
↓
Recruit macrophage(activate macrophage)
↓
Inflammation of all 3 layer of heart

MODIFIEDJONES CRITERIA (2015UPDATE)

MAJORE CRITERIA

1. CARDITITIS

*Subclinical
* Echo with Doppler must be done in all cases of confirmed and suspected acute Rheumatic fever as auscultatory findings take time to develop .

*Subclinical carditis seen on echo is now considered a Major diagnostic criteria

PERICARDITIS(M/C)
*Echo -Valvulitis
*5-15years, Recurrent Sore throat
*Chestpain at rest, pericardial friction rub
* ECG-ST 1, concave upwards present in all leads except aVR.
• MANAGEMENT: Steroids

MYOCARDITIS: acute CHF
* Dilation of valve annulus leading to
*MRfunctional
* TRfunctional
* Pansystolic murmur
* Manifestation- Acute Pulmonary edema

• MANAGEMENT
* L-LASIX
* M-Morphine
* NO -NTG,
*P-Head high Position

ENDOCARDITIS
Aschoff nodules involve commissural ends (tips of valve where cusps touch each other)
* due to Valvulitis. Blood leaking across the valve (LV -LA)
*As this extra blood accumulate in left atria and when Come back to LV results in murmur called as Carey Coomb Murmur

MANAGEMENT
• Calcified MS: prosthetic vave
• Severe MR:Valvuloplasty

2. Arthritis
* Earliest manifestation within 2 weeks with history of sorethroat
*Ankle swelling
*Migratory polyarthritis
• Kneeswelling, Elbow swelling
• All joints do not affect simultaneously, they affect one after the other
*When swelling subsides, no residual deformity: Non- erosive arthritis
*Sometime can cause erosive arthritis called as Jacoud's Arthritis

3. Syndenham Chorea (Late neurological feature)
* Can develop as late as 90 days or later
* Damage to caudate nucleus
* Fast, purposeless, involuntary distal movements • Poor handwriting •School grades /se

Difference from Chorea
*Athetosis: Slow, writhing distal involuntary movement D/t lesion: Globus pallidus
* Hemibalismus: wide flinging movements proximal & Distal Due to :Subthalamic nucleus

ON EXAMINATION
* Darting Tongue (Inability to keep tongue in stable position)
* Milk-maid grip (Inability to generate persistent hand grip)
*Hung up reflexes (Hung up anklejerk- Myxedema)
*Dysarthria (unclear speech)

MANAGEMENT

• Self limiting condition: resolve in < 6 months- 12 months
• Valproate
• Haloperidol
• Phenobarbitone(if fails start steroids)
• Steroids(if fails start IV immunoglobulin)
• Medically refractory chorea: IVIg

4. Subcutaneous nodules

* Extensor in distribution
* Sites: Olecranon, occiput, spinous process of Vertebrae
* Rheumatoid nodules: - M/C
* Extra-articular manifestation of rheumatoid arthritis
* Extensor, non-tender
* Involve lungs, heart., mononeuritis multiplex

5. Erythema Marginatum (Not seen in Indian population)

• MINOR CRITERIA

* Fever (>38.5°C), Low endemicity (>38°C), High endemicity
* Arthralagia, Poly, Mono
*increased CRP OR ESR
*prolonged PR interval

DIAGNOSTIC ALGORITHM

• Major criteria + essential criteria (evidence of preceding Grp A Streptococcal infection)
• l major+ 2 minor criteria + essential criteria (evidence of preceding Grp A Streptococcal infection)
• Recurrence ARF:3 minor criteria

ESSENTIAL CRITERIA, UNIVERSAL CRITERIA, RECOMMENDED TESTS (EVIDENCE OF RECENTSTREPT. INFECTION <45 DAYS)

1. Elevated ASO titres
2. Anti DNASE B ab
3. Rapid gp A Strept carbohydrate test
4. Throat swab

MANAGEMENT

1. Aspirin: Arthritis
2.Steroids: Pericarditis/severe chorea
3. Valproate: chorea
4.IV lg:medically refractive chorea
5. Injection Benzathine penicillin I.M. every 3 weeks
*RHD with valvular lesion injection given till 40years
*RHD without valvular lesion injection given till 21 years
*For RF injection given till 21 years

PROPHYLAXIS

*Penicillin if resistance present Macrolides- Azithromycin

REFERENCE

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Pericarditis

DEFINITION The double wall, fluid filled sac that encloses the heart. The pericardial cavity contains 15-50 ml of fluid which serves as lubricant and allows the heart to contract and relax with minimum friction, it also protects the heart from infections and over distress.

OCCURRENCE

Pericarditis commonly occurs in the late summer and fall concurrent with epidemics of enteroviruses. Pericarditis occurs in people of all ages, although viral pericarditis is more common in adults than in children.

TYPES

m/c- Acute pericarditis-lasts <4-6weeks
sub-acute pericarditis-lasts >4-6weeks but <3 months
chronic pericarditis-lasts >3 months
recurrent pericarditis- symptom free intervals of 4-6 weeks.

SYMPTOMS

Chest pain (central/left)-sharp (more often) or dull
Pain may radiates to left shoulder or arm
Pain worsened with deep breathing, coughing, swallowing or lying flat & improved sitting up or leaning forward.

RISK FACTORS

Flu, Covid-19, Radiation, SLE autoimmune, Recent invasive heart procedure

CAUSES

Viral infection, most often gastrointestinal viruses
Bacterial – TB, Injury to chest, inflammatory disorders, kidney failure, tumor, genetic diseases, some medications following a heart attack or cardiac surgery.

PATHOPHYSIOLOGY

Any cause- inflammation- can cause fluid accumulation in pericardial cavity and cause effusion ----due to increased fluid volume limit cardiac filling and cause low cardiac output-----sometimes life threatening circulatory shock , while coming to chronic inflammation may result thickened or stiffened pericarditis as this condition reduces cardiac filling , blood backs up in body veins and lungs ----a peripheral venous congestion leads to swelling of legs &abdominal organs------- elevates pulmonary pressure so difficulty in breathing .

DIAGNOSIS

Pericardial rub , ECG shows in 4 stages—ST elevation, T wave flattering , T wave inversion , ECG returns to normal Chest X-ray ---- can see pericardial effusion

TREATMENT

Depend on severity , Anti-inflammatory – ibuprofen, aspirin/colchicine
Corticosteroids for noninfectious
Bacterial infections- antibiotics , possibly drainage
If Cardiac tamponade - Pericardiocentesis to remove excess
If severe constrictive pericarditis- Surgery - Pericardiectomy

COMPLICATIONS

Pericardial effusion, low cardiac output, Cardiac tamponade, Constrictive pericarditis, Hepatomegaly

REFERENCES:

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FELICIA ANTONY
6th semester 3rd year MBBS
Session 2021-2026

Heart Murmurs

Heart murmurs are sounds — such as whooshing or swishing — made by rapid, choppy (turbulent) blood flow through the heart. the sounds can be heard with a device called a stethoscope. a typical heartbeat makes two sounds like "lubb-dupp" (sometimes described as "lub-dup") when the heart valves are closing

SYMPTOMS:

Blue or gray fingernails or lips,chest pain,cough that doesn't go away, dizziness, swollen liver, swollen neck veins, fainting,heavy sweating with little or no activity, in infants, poor appetite and lack of growth,shortness of breath,swelling or sudden weight gain

CAUSES

Heart is filling with blood (diastolic murmur)
When the heart is emptying (systolic murmur)
Throughout the heartbeat (continuous murmur)

HARMLESS (INNOCENT) HEART MURMURS

Lack of healthy red blood cells that carry oxygen to body tissues (anemia)
Overactive thyroid (hyperthyroidism)
Phases of rapid growth, such as adolescence

Worrisome heart murmurs: Holes in the heart, Cardiac shunts.
Calciumdeposits.endocarditis.Rheumatic fever..

- **Risk factors**

• 1. In babies: Family history of heart problems linked to murmurs, Uncontrolled diabetes in the mother during pregnancy, German measles (rubella) in the mother
2. adults:A weakened heart muscle (cardiomyopathy),An infection of the lining of the heart (endocarditis).Anemia. Certain autoimmune disorders such as lupus and rheumatoid arthritis, Heart valve disease,High
blood pressure in the lungs (pulmonary hypertension), History of rheumatic fever

Prevention

There's no known prevention for heart murmurs. But healthy lifestyle changes can improve heart health and prevent some conditions linked to murmurs in adults. Many childhood heart murmurs go away on their own as children grow.

Diagnosis

Echocardiogram:It shows how blood flows through the heart and heart valvesAn echocardiogram uses sound waves to create pictures of the beating heart.
Chest X-ray.:It can tell whether the heart is enlarged

Electrocardiogram (ECG or EKG):This quick and painless test measures the electrical activity of the heart.

Cardiac catheterization. This test may be done when other tests haven't found a cause for the heart murmur. A heart doctor (cardiologist) inserts a flexible tube (catheter) into a blood vessel, usually in the groin or wrist. The catheter is gently guided to the heart. Dye may be injected through the catheter. The dye helps blood vessels show up better on the images made during the test.

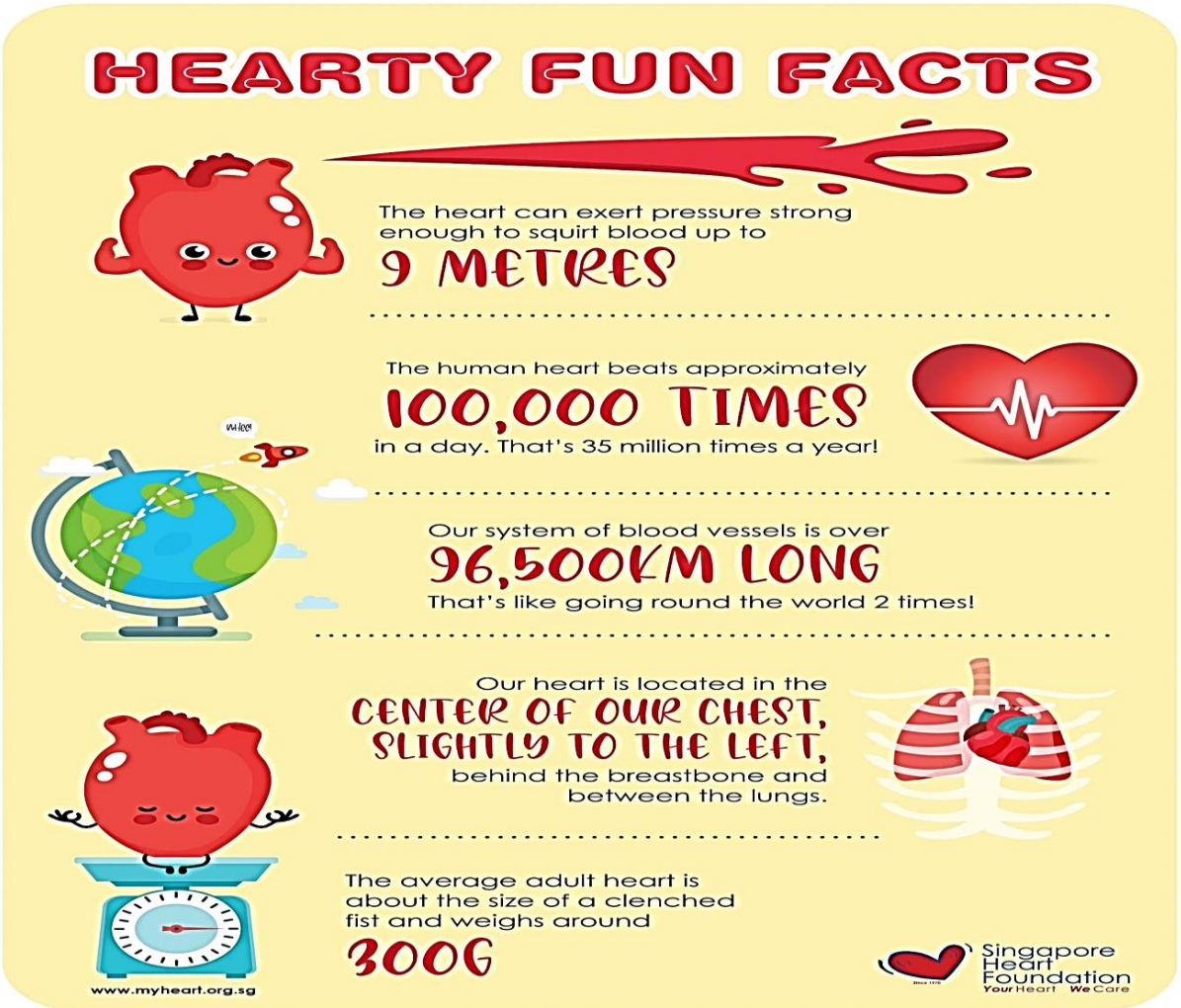
Treatment

Blood thinners (anticoagulants),Water pills (diuretics),Angiotensin-converting enzyme (ACE) inhibitors. Beta blockers

• Surgery or other procedures:

Heart valve surgery may be done as:

• Open-heart surgery
• Minimally invasive heart surgery
• Robotic heart surgery
• A procedure using flexible tubes (catheter procedure)



SYEDA MAHNOOR KAZMI
6th semester 3rd year MBBS
Session 2021-2026

Electrocardiogram

PURPOSE OF WRITING

I picked this topic because ECG is like a heart's storyteller—it narrates the tales of common heart issues. For doctors and med students, understanding its basics is like learning the language of hope. Early diagnosis is crucial in saving someone's life .

DEFINITION

The term "ECG" comes from the German word "elektro-kardiographie." The electrocardiogram, often shortened as ECG or EKG, is a non-invasive recording of the heart's electrical activity taken from the body's surface.

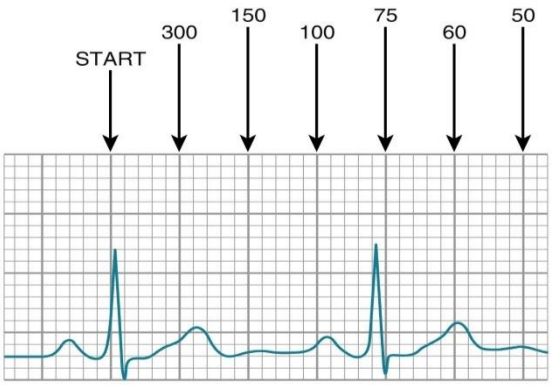
HISTORY

This vital medical tool was invented in 1902 by the Dutch physician Einthovan. Over the course of approximately ten years, his significant contributions to clinical studies played a pivotal role in fully recognizing the clinical potential of this technique.

NOMENCLATURE OF ECG

P Wave: Illustrates atrial depolarization. Following the P wave, the PR segment signifies a delay at the AV node, allowing the ventricles to adequately fill before contraction (usually 0.12 to 0.20 seconds). Diseases affecting the AV node often result in PR-segment prolongation or abnormalities. *PR interval* is the interval from the beginning of the P wave to the beginning of the Q wave (initial depolarization of the ventricle)

QRS Complex: Represents ventricular depolarization. Understanding QRS terminology can be complex, but essentially, a downward first deflection is a Q wave; if it's upward, there is no Q wave. The initial upward deflection is always an R wave. Any downward deflection after the R wave is an S wave. Although the atria repolarize during the QRS complex, it doesn't appear on the tracing due to the dominance of ventricular activity. Normally, QRS duration is under 0.12 seconds, but malfunctioning of the fast His-Purkinje conduction system can lead to abnormally widened QRS complexes, as seen in bundle branch blocks.

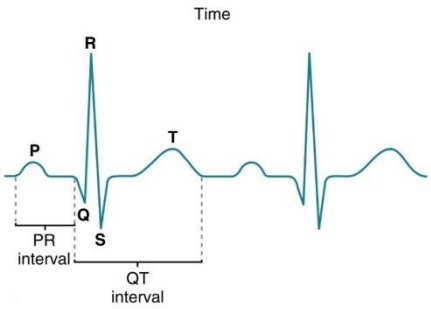


*QT Interval:*Spans from the onset of the Q wave to the conclusion of the T wave.Encompasses the entire duration of ventricular depolarization and repolarization.

ST segment is the segment from the end of the S wave to the beginning of the T wave. Maintains an isoelectric state. Signifies the phase when

the ventricles undergo depolarization.

T Wave: Occurs after ventricular depolarization in the QRS complex. Following a brief delay (ST segment), repolarization happens, represented by the T wave. Occasionally, a U wave may follow the T wave, which can be normal but is uncommon. It might also be observed in conditions like hypokalemia or bradycardia.



ECG REPORT

An ECG report consists of two primary elements: the 12-lead ECG, examining the heart from various perspectives, and the rhythm strip, valuable for detecting irregularities in rhythm (arrhythmias) or heart rate**.**

To find the heart rate using an ECG strip, consider that each small box represents 0.04 seconds (40 milliseconds), and every group of five small boxes forms a larger box, totaling 0.20 seconds (200 milliseconds). Count the number of large boxes between QRS complexes and use the guide:

300-150-100-75-60-50

If there is one big box between beats, the rate is 300; two is 150, and so on. This helps estimate the heart rate.

12-LEAD ECG

It involves placing electrodes on specific locations on the chest and limbs to create 12 different "views" of the heart's electrical signals. Each lead records the electrical activity between two electrodes, allowing for a comprehensive assessment of the heart's function.

The 12 leads are divided into three groups:

Limb Leads (I, II, III, aVR, aVL, aVF): These leads are placed on the arms and legs and provide information about the heart's activity in the frontal plane.

Precordial or Chest Leads (V1 to V6): These leads are positioned on the chest and give information about the heart's activity in the horizontal plane, focusing on the front of the heart.

12-lead Precordial lead placement:

V1: 4th intercostal space (ICS), RIGHT margin of the sternum

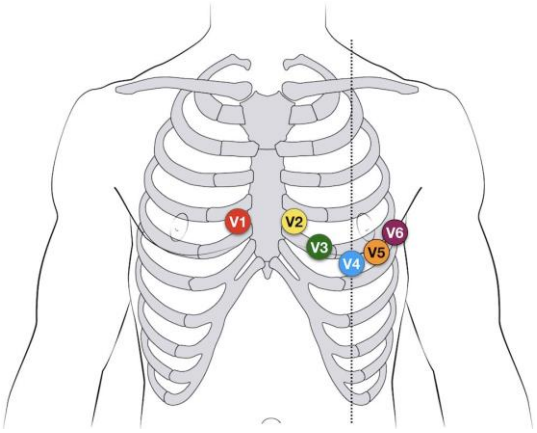
V2: 4th ICS along the LEFT margin of the sternum

V4: 5th ICS, mid-clavicular line

V3: midway between V2 and V4

V5: 5th ICS, anterior axillary line (same level as V4)

V6: 5th ICS, mid-axillary line (same level as V4)



- **Augmented Leads** (aVR, aVL, aVF): These leads are derived from the limb leads and provide additional perspectives on the heart's electrical activity.

INDICATIONS

Due to its extensive application in medicine, the ECG serves various purposes outlined as follows:

- ✓ Primary indications involve symptoms such as palpitations, dizziness, cyanosis, chest pain, syncope, seizures, and poisoning.
- ✓ It is used for symptoms or signs linked to heart conditions like tachycardia, bradycardia, and clinical situations including hypothermia, murmurs, shock, hypotension, and hypertension.
- ✓ To detect myocardial injury, ischemia, and the presence of prior infarction
- ✓ Evaluation of metabolic disorders
- ✓ Valuable aid in the study and differential diagnosis of congenital heart disease
- ✓ Cardiopulmonary resuscitation
- ✓ Screening tool in a sports physical exam to rule out cardiomyopathyKrikler DM.

CONTRAINDICATIONS

There are no definite restrictions against an electrocardiogram. However, relative limitations to its application encompass:

- ✓ Patient refusal
- ✓ Allergy to the adhesive used for lead attachment

CLINICAL SIGNIFICANCE

The objective of interpreting an electrocardiogram (ECG) is to discern whether the observed waves and intervals are within the normal range or indicative of pathology. Analyzing the electrical signals provides a valuable approximation of potential heart pathology.

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MAAZ AKRAM
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Session 2021-2026

Cardiomyopathy

I am Maaz Akram From 5th semester 3rd year . The reason of this topic because in our country so many patients in this year That way I wanted to explore this multifaceted treatment approaches ..Better management and improved quality of life for patients

INTRODUCTION DISEASE

1:- RESTRICTIVE CARDIOMYOPATHY

A. Decreased compliance of the ventricular endomyocardium that restricts filling During diastole.
B. Causes include amyloidosis, sarcoidosis, endocardial fibroclastosis , and Loeffler syndrome (endomyocardial fibrosis with an eosinophilic infiltrate And eosinophilia).
C. Presents as congestive heart failure, classic finding is low voltage EKG with Aminished QRS amplitude.

2:- HYPERTROPHIC CARDIOMYOPATHY

A. Massive hypertrophy of the left ventricle
B. Umally due to genetic mutations in sarcomere proteins, most conummon form is autosomal dominant C Clinical features include

- ✓ .Decreased cardiac output Left ventricular hypertrophy leads to diastolic dysfunction (ventricle cannot fil)。
- ✓ .Sudden death due to ventricular arrhythmias, hypertrophic cardiomyopathy is a common cause of sudden death in young athletes.
- ✓ .Syncope with exenise-Subaortic hypertrophy of the ventricular septum results in functional aortic stenosis.

C. Biopsy shows myofiber hypertrophy with disarray

3:- DILATED CARDIOMYOPATHY

A Dilation of all four chambers of the heart ; most common form of cardiomyopathy
B. Results in systolic dysfunction (ventricles cannot pamp), leading to biventricular CHF: complications include mitral and tricuspid valve regurgitation and arrhythmia.
C.Most commonly idiopathic, other causes include
1 Genetic mutation (usually autosomal dominant)
2 Myocarditis (usually due to coxsackie A or B) characterized by a lymphocytic infiltrate in the myocardium results in chest pain, arrhythmia with sudden death, or heart failure. Dilated cardiomyopathy is a late complication.
3.Alcohol abuse
4.Drugs (eg, dexorubicin)

5.Pregnancy seen during late pregnancy or soon (weeks to mantha) after childbirth
6.Hemochromatosis
D.Treatment is heart transplant.

EPIDEMIOLOGY

Cardiomyopathy often goes undiagnosed, so the numbers can vary. As many as 1 of 500 adults may have this condition. Males and females of all ages and races can have cardiomyopathy.

MORTALITY RATE

Among males with HCM, one-, five-, and 10-year mortality rates after diagnosis were 3.1% (95% CI 2.0-4.7 %), 14.4% (95% CI 11.4-18.0%) and 31.8% (95% CI 24.3-40.8%), while the corresponding rates for females with HCM were 2.8% (1.7-4.8%), 19.4 % (15.4-24.3%) and 42.5% (95% CI 32.6-54.1%).

ETIOLOGY

Viral infections in the heart are a major cause of cardiomyopathy. In some cases, another disease or its treatment causes cardiomyopathy. This might include complex congenital (present at birth) heart disease, nutritional deficiencies, uncontrollable, fast heart rhythms, or certain types of chemotherapy for cancer..

RISK FACTORS

• Family history of cardiomyopathy, heart failure and sudden cardiac arrest.
• Long-term high blood pressure.
• Conditions that affect the heart, including a past heart attack, coronary artery disease or an infection in the heart (ischemic cardiomyopathy)
• Obesity, which makes the heart work harder.

SYMPTOMS

• Shortness of breath or trouble breathing, especially with physical exertion.
• Fatigue.
Swelling in the ankles, feet, legs, abdomen and veins in the neck.
• Dizziness.
• Lightheadedness.
• Fainting during physical activity.
• Arrhythmias (abnormal heartbeats)

PATHOGENESIS

- Complex and multifactorial, involving genetic, environmental, and lifestyle factors that can interact and trigger a cascade of events leading to the development of the disease.
- Genetic mutations are known to play a significant role in the pathogenesis of cardiomyopathy.

DIAGNOSIS

Be recommended to diagnose cardiomyopathy: Blood tests: A small amount of blood is usually drawn from a vein in your arm using a needle. Chest X-ray: A chest X-ray takes pictures of the organs and structures inside your chest, and can show whether your heart is enlarged.

TREATMENT

a. Beta-blockers.
b. ACE inhibitors.
c. Aldosterone receptor blockers (ARB).

d. Angiotensin receptor-neprilysin inhibitors (ARNi).
e. Diuretics.
f. Anti-arrhythmic, drugs that treat abnormal heart rhythms.
g. Blood thinners (anticoagulants).

RATE OF CURE

A prevalence of 1:2500 is commonly cited to estimate the prevalence of idiopathic DCM. This estimate relies on a population-based epidemiologic study conducted in Olmstead County, Minnesota, between 1975 and 1984 and is based on a mere 45 DCM cases in a limited demographic band.

COMPLICATIONS

Dilated cardiomyopathy can lead to heart valve problems, arrhythmia, blood clots in the heart, heart failure and even sudden cardiac death. About a third of the people with dilated cardiomyopathy inherit it from their parents. Other causes of DCM include: Autoimmune disease.

PREVENTIONS

Avoid excess alcohol use.
• Treat high blood pressure.
• Live a healthy lifestyle and maintaining a healthy weight.
• Avoid illegal drug use.

Get prompt treatment for potential causes, like thyroid disease.
• Reduce your risk factors for coronary artery disease.

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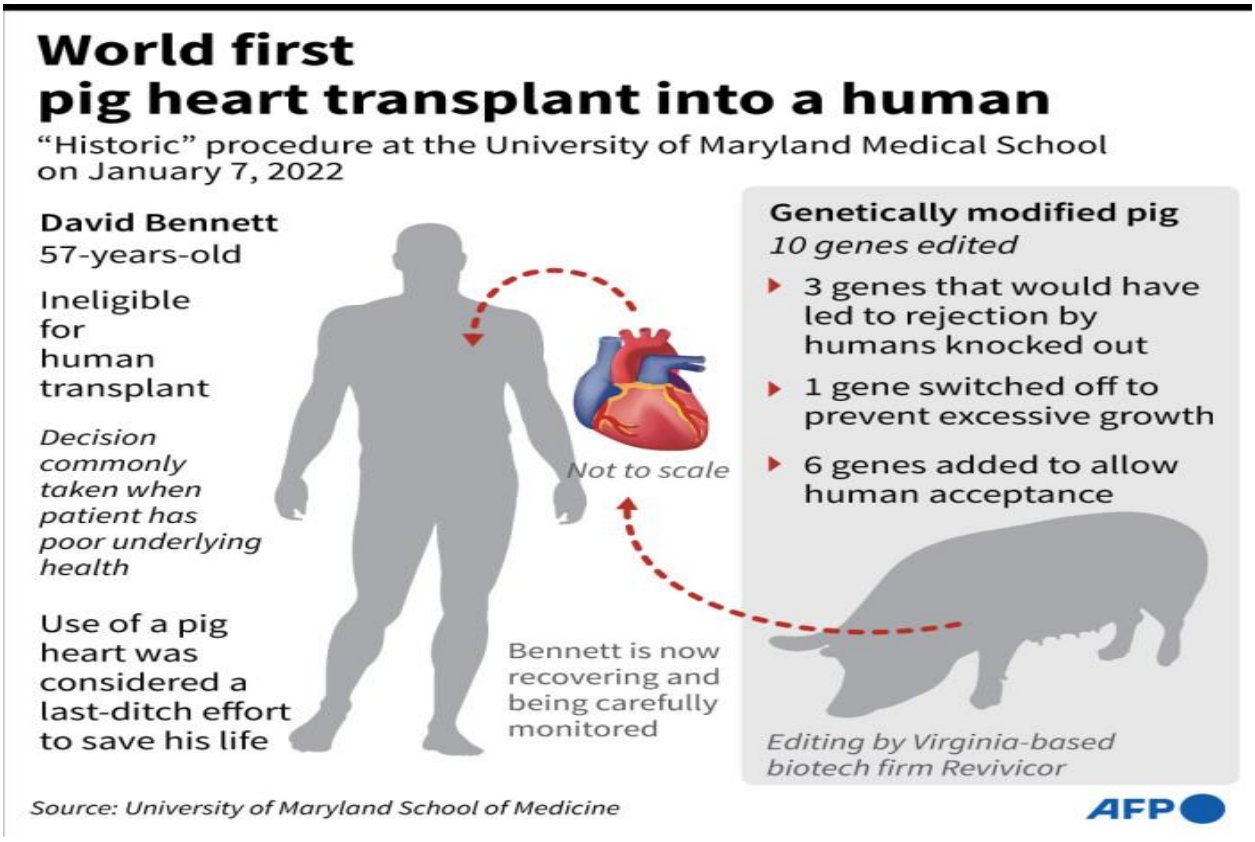

ABDUL REHMAN
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Session 2021-2026

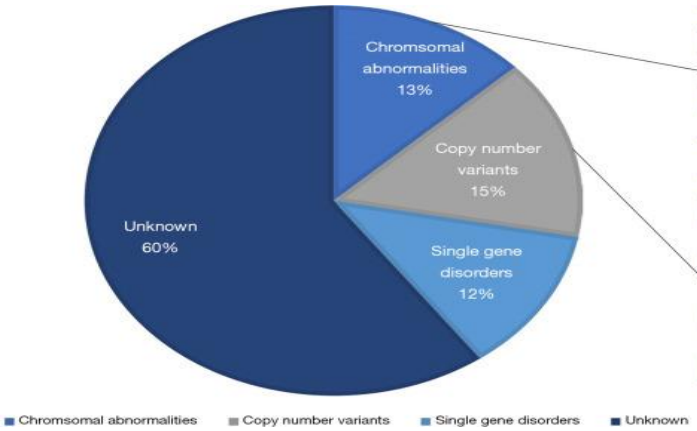
Congenital Heart Disease

Introduction

Congenital heart disease (CHD) is the most common human birth defect and remains a leading cause of mortality in childhood. Although advances in clinical management have improved the survival of children with CHD, adult survivors commonly experience cardiac and non-cardiac comorbidities, which affect quality of life and prognosis. Therefore, the elucidation of genetic etiologies of CHD not only has important clinical implications for genetic counseling of patients and families but may also impact clinical outcomes by identifying at-risk patients. Recent advancements in genetic technologies, including massively parallel sequencing, have allowed for the discovery of new genetic etiologies for CHD. Although variant prioritization and interpretation of pathogenicity remain challenges in the field of CHD genomics, advances in single-cell genomics and functional genomics using cellular and animal models of CHD have the potential to provide novel insights into the underlying mechanisms of CHD and its associated morbidities.

Established etiologic contributors to CHD

The etiology of CHD is multifactorial as both genetic and environmental factors have been implicated in its etiology (20). Specific genetic causes can be detected in an estimated 40% of CHD cases (Figure 1). Genetic causes of CHD are extremely heterogeneous, including chromosomal anomalies or aneuploidies (estimated 13%, range from 9% to 18%) (21), copy number variants (CNVs) (estimated 10–15%: range from 3% to 25% in syndromic CHD and 3% to 10% in non-syndromic CHD) (22-24), and single gene disorders (12%) (13,25-27). The genetic basis of CHD can be divided into syndromic CHD and non-syndromic CHD, where congenital abnormalities are isolated to the heart.



Aneuploidies	Proportion
Down syndrome	75%
Trisomy 18	16%
Turner syndrome	5%
Trisomy 13	4%

Copy number variants	Proportion
22q11.2 deletion syndrome	10%
1p36 deletion	6%
Williams-Beuren syndrome	4%
Jacobsen syndrome	0.1%
8p23.1 deletion	0.1%
1q21.1 deletion/duplication	NR
Other	79.8%

Genetic testing Over the past 20 years, the advanced genetic testing methodologies (e.g., CMA and exome sequencing) are increasingly being incorporated into the genetic evaluation of patients with CHD, and the results of this testing has important clinical implications. The clinical benefits of genetic testing for patients with CHD include establishing a genetic diagnosis, anticipatory management of CHD and associated extra-cardiac conditions, and clinical screening of at-risk family members.

Mortality Rate

Preterm infants with low birth weight and comorbidities presented a higher risk of mortality related to congenital heart diseases. This cohort was extinguished very quickly, signaling the need for greater investment in assistance technology in populations with this profile. The incidence rate of mortality from congenital heart disease was 81 cases per 100,000 live births. The lethality attributed to critical congenital heart diseases was 64.7%, with proportional mortality of 12.0%. The survival rate at 28 days of life decreased by almost 70% in newborns with congenital heart disease. The main cause of death is cardiogenic shock.

Pharmacological Therapeutics

Angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, beta-blockers, and potassium-sparing diuretics have shown a mortality benefit in most patients. Other therapies, such as endothelin receptor antagonists, phosphodiesterase-5 inhibitors, prostaglandins, and soluble guanylyl cyclase stimulators, have benefited patients with pulmonary artery hypertension. Likewise, the adjunctive symptomatic treatment of these patients has further improved the outcomes, since antiarrhythmics, digoxin, and non-steroidal anti-inflammatory drugs have shown their benefits in these cases.

Conclusion

Prenatal diagnosis of severe CHD had an impact on the decision regarding termination of pregnancy, but not on the 1-year prognosis among live births. We should now use large multicentre CHD registries to determine the impact of prenatal diagnosis on postnatal management, neurological. CHD depends on various therapeutic interventions, which ultimately lead to definitive surgical correction. Although many patients are reaching adulthood, additional treatment options will improve their quality of life, especially in developing countries. Likewise, innovation should be sought for new drug candidates that address the physiological nature of CHD defects and eventual increase in survival

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Session 2021-2026

Angina Pectoris

Angina pectoris is of interest as a cause of disability, and also because it is a marker for potential severe manifestations of coronary heart disease like cardiac infarction, stroke, or sudden death. It is the most common symptom of ischemic heart disease, which is the major cause of morbidity and mortality worldwide.

Definition

Angina is defined as the chest pain or discomfort, caused by an imbalance between oxygen supply and demand, as a result of reduced blood flow to the heart muscle. It exists in 3 forms: 1)Stable, in which the symptoms present upon exertion only; 2)Unstable, occurring even at rest; & 3)Prinzmetal, also known as vasospastic.

Epidemiology

Angina is estimated to have affected 1.6% of the population (approximately 112 million people). It is slightly more common in men than women. It's prevalence also increases with age (over 50 years).

Mortality rate

The average annual mortality in unselected patients with chronic stable angina is 4%. Mortality is increased in male patients and in patients who have risk factors.

Etiology

The most common cause of stable and unstable angina is coronary artery disease. This is when these arteries are narrowed by the build-up of a fatty substance called plaque (atherosclerosis). In the case of Prinz metal angina, vasospasm results in the narrowing of the artery.

Risk factors

The unmodifiable factors include a person's age, sex, genetics and ethnicity. People with lifestyle habits like alcohol use, smoking, unhealthy diet, as well as underlying medical conditions like hyperlipidemia, diabetes, hypertension, obesity, cardiomyopathy, etc are also at risk.

Pathogenesis

In classic angina, atherosclerotic narrowing limit the ability of the coronary arteries to augment myocardial blood flow in response to increases in demand; and in variant angina, a primary reduction in coronary blood flow occurs, unrelated to changes in demand.

Diagnosis

The most widely used method is ECG, followed by stress test, chest X-ray, coronary angiography, CT and cardiac MRI.

Treatment

*Treatment of chronic stable angina is aimed at managing symptoms as well as slowing the progression to cardiac events. To provide symptomatic relief, Nitrates (sub-lingual) are the drugs of choice.

*Treatment for unstable angina is aimed at pain reduction, limiting damage to the myocardium, and decreasing morbidity and mortality. The first-line drugs are Nitrates; in addition, Morphine, Oxygen therapy, & Aspirin are administered. Other medications include Beta blockers, Calcium channel blockers, Sodium channel blockers, & Anti-coagulants.

*Vasospastic angina is managed using Nitrates, Calcium channel blockers, & Statins.

Complications

If left untreated, it could lead to myocardial infarction, commonly known as a heart attack. Other possible complications include arrhythmias, heart failure, stroke or even death.

Conclusion

Preventive measures include modifying our diet, avoiding alcohol and smoking, doing exercises, and managing stress as well.

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AMBREEN ASLAM
6th semester 3rd year MBBS
Session 2021-2026

Hyperlipidemia

My choice of exploring hyperlipidemia stems from a personal connection to the disease. I seek advancements for enhanced patient management and improved quality of life.

INTRODUCTION

Hyperlipidemia is a medical condition characterized by elevated levels of lipids (fats) in the blood. It plays a significant role in cardiovascular diseases. It poses a considerable threat to heart health. It is immediate requirement to change the life style after a person is diagnosed with this condition as, if untreated, it can lead to serious health hazards and even mortality of a patient.

EPIDEMIOLOGY

The global prevalence of hyperlipidemia is substantial. It is affecting millions of individuals. Its prevalence was 33.8% in 2020, in the world. More than 34 M people are being affected by hyperlipidemia in a year. The condition is often associated with lifestyle factors.

MORTALITY RATE

While hyperlipidemia itself may not directly cause mortality, its impact on cardiovascular health can lead to severe complications such as heart attacks and strokes. To understand the mortality rate that is associated with these complications, it is crucial to assess the overall risk. For instance, in diabetes patients, hyperlipidemia is a risk of mortality and its hazard value is 1.02 with confidence interval of 95% between 0.73 to 1.43.

ETIOLOGY

- genetic factors,
- poor dietary choices,
- sedentary lifestyles, or
- underlying medical conditions.

RISK FACTORS

Various factors contribute to the development of hyperlipidemia. They include

- genetics,
- diet,
- physical inactivity, and
- certain medical conditions.

It is essential to Identify these risk factors for preventive measures and early intervention.

SYMPTOMS

- ✧ Xanthomas
- ✧ Xanthelasma
- ✧ Arcus senilis
- ✧ Corneal arcus

DIAGNOSIS

- ❖ Lipid Panel: It measures levels of total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and triglycerides in the blood.
- ❖ Non-HDL Cholesterol: It calculates the total cholesterol minus the HDL cholesterol. It provides a more comprehensive assessment of atherogenic lipoproteins.
- ❖ Apolipoprotein B (ApoB) Test: It measures the number of ApoB-containing lipoproteins, and provides information about the total number of atherogenic particles.
- ❖ High-Sensitivity C-Reactive Protein (hs-CRP) Test: It assesses inflammation levels in the body, which can be associated with cardiovascular risk in conjunction with lipid levels.
- ❖ Lipoprotein(a) Test: It measures the level of lipoprotein(a), a type of LDL particle, which may be a risk factor for cardiovascular disease.
- ❖ Electrophoresis: It separates lipoproteins based on their electrical charge, and helps to identify abnormalities in lipoprotein patterns.

TREATMENT

- ■ Lifestyle Modifications:
- ✓ Dietary Changes:

a. Reduce saturated and trans fats in the diet.
b. Increase intake of fruits, vegetables, whole grains, and fiber.
c. Choose lean proteins, such as fish and poultry.
d. Limit cholesterol intake from animal sources.

- ✓ Regular Exercise:

e. Engage in aerobic exercise (e.g., brisk walking, jogging) for at least 150 minutes per week.
f. Include strength training exercises.

- ■ Medications:
- ✓ Statins:
  - Atorvastatin
  - Pravastatin
  - Lovastatin
- ✓ Bile Acid Resins:
  - Cholestyramine
  - Colesevelam
  - Colestipol

COMPLICATIONS

Hyperlipidemia is a significant risk factor for cardiovascular diseases, It includes

- ➢ atherosclerosis,
- ➢ coronary artery disease
- ➢ stroke.

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BADAR ALI
9th semester 5th year MBBS
Session 2020-2024

Right Sided Heart Failure

In right –sided heart failure, the hearts right ventricle is too weak to pump enough blood to the lungs. As blood builds up in the veins, fluids gets pushed out into the tissues in the body.

Right sided heart failure symptoms include swelling and shortness of breath

Blood builds up in the veins, increase pressure in veins, this pressure let the fluid leakage from the capillaries = causes edema or swelling of body.

It can cause the portal hypertension and many more complications related to this one complication like esophageal varices with symptoms of hemoptysis, anorectal varices, caput medusa.

INCIDENCE OF RHF

The prevalence rate of heart failure among US adults is approximately 1.9% to2.6%for the overall population and is higher among older patients.

3% to 9% of acute heart failure admission, and the in hospital mortality of patients with acute RV failure ranges from 5% to 17%.

RISK FACTOR

Left heart failure
Chronic lung disease
Cor pulmonale
Valvular heart disease
Cardio myopathy
Obstructive sleep apnea
Hemodynamic overload

PATHOGENESIS

Pathogenesis of RHF includes the incompetence of the right heart to maintain systemic venous pressure sufficiently low to guarantee an optimal venous return and to preserve renal function. Virtually, all myocardial disease involving the left heart may be responsible for RHF This may result from coronary artery disease, hypertension, valvar heart disease. In case of dilated cardiomyopathies, the heart is unable to contract properly to push blood to the other body parts. It’s important to recognize that right heart failure often occurs secondary to left heart failure but it can also result from primary conditions affecting the right side of the heart

SYMPTOMS

Swelling
Enlarged liver
Distended neck veins
Fatigue and weakness
Cyanosis

DIAGNOSIS

Electrocardiogram

Echocardiogram
Blood test, especially to measure substances called natriuretic peptide

TREATMENT

The treatment of right heart failure involves addressing the underlying causes and managing symptoms
Address underlying cause
Medication like diuretics, vasodilators, inotropic agent
Oxygen therapy
Surgical and interventional options, (Heart valve repair, implantation of device like pacemaker
Heart transplant
Pulmonary rehabilitation

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Pregnancy Associated Anemia

I chose to explore this topic of pregnancy associated Anemia for my article due to it's significant impact on maternal health and development of the child in mother's body. The prevalence of anemia among young mothers during pregnancy has a huge affect on the fetal development.

INTRODUCTION:

Anemia is when your blood has too few red blood cells. Having too few red blood cells makes it harder for your blood to carry oxygen or iron. This can affect how cells work in nerves and muscles. During pregnancy, your baby also needs your blood and the most common type of Anemia in pregnancy include " Iron-deficiency anemia ". which arises when the body lacks sufficient iron to produce an adequate amount of hemoglobin. " Folate deficiency Anemia" due to folic acid deficiency. " Vitamin B12 deficiency Anemia " due to poor absorption of B12 vitamin.

EPIDEMIOLOGY:

WHO estimates that 40% of children 6–59 months of age, 37% of pregnant women, and 30% of women 15–49 years of age worldwide are anemic.

Iron-deficiency anemia is the most prevalent form of anemia in pregnancy, affecting a large number of women globally. In various regions, the occurrence of iron-deficiency anemia during pregnancy can range from 15% to 70%, emphasizing its widespread impact.

Folate deficiency anemia and vitamin B12 deficiency anemia also contribute to the overall burden of pregnancy-related anemia, with varying prevalence rates across different populations.

MORTALITY RATE:

The mortality rate directly attributed to pregnancy-related anemia is relatively low in developed countries where access to healthcare and proper antenatal care is widespread. However, in resource-limited settings, rural areas , particularly 3rd world countries or underdeveloped counting, severe anemia during pregnancy can contribute to maternal mortality. Complications such as hemorrhage during childbirth, can lead to shock affecting the mortality.

SIGNS AND SYMPTOMS:

Pregnancy-associated anemia can manifest through various signs and symptoms, including:

•Fatigue: Persistent tiredness and weakness are common due to a decrease in red blood cells and hemoglobin.

•Shortness of Breath: Reduced oxygen-carrying capacity of the blood can lead to increased breathlessness, especially during physical activity.

•Loss of consciousness(vertigo) and dizziness: due to reduced blood flow

•Paleness of the skin, lips, nails, palms of hands, or underside of the eyelids: due to the lack of blood transport.

•Labored breathing
•Rapid heartbeat (tachycardia)
•Trouble concentrating or focusing

AETIOLOGY:

The most common nutritional cause of anemia is iron deficiency, although deficiencies in folate, vitamins B12 and A are also important causes.

Anemia is a serious global public health problem that particularly affects young children, menstruating adolescent girls and women, and pregnant and postpartum women.

•Iron-deficiency anemia: during pregnancy, baby uses your red blood cells for growth and development, especially in the last 3 months of pregnancy. If you have extra red blood cells stored in your bone marrow before you get pregnant, your body can use those stores during pregnancy. Women who don't have enough iron stores can get iron-deficiency anemia. This is the most common type of anemia in pregnancy.

•Vitamin B-12 deficiency: Vitamin B-12 is important in making red blood cells and protein. Eating food that comes from animals, such as milk, eggs, meats, and poultry, can prevent vitamin B-12 deficiency.

•Folate deficiency: Folate (folic acid) is a B vitamin that works with iron to help with cell growth. If you don't get enough folate during pregnancy, you could get iron deficiency. Folic acid helps cut the risk of having a baby with certain birth defects of the brain and spinal cord if it's taken before getting pregnant and in early pregnancy.

RISK FACTORS FOR ANEMIA IN PREGNANCY:

All pregnant women are at risk for becoming anemic. That's because they need more iron and folic acid than usual. But the risk is higher if:

•Multiple pregnancies (more than one child).
•Have had two pregnancies close together (very less gap between conception after first pregnancy and second).
•Vomit a lot because of morning sickness (common in pregnancy).
•Teen pregnancies (no proper education).
•Don't eat enough foods that are rich in iron(malnutrition).
•Anemic before pregnancy.
• vegan / vegetarians

COMPLICATIONS:

Severe or untreated iron-deficiency anemia during pregnancy can increase your risk of having:

•Preterm or low-birth-weight baby.(premature)
•A blood transfusion (if you lose a significant amount of blood during delivery)
•Postpartum depression.
•Baby born with anemia.
•Child with developmental delays.
•Baby with a serious birth defect of the spine or brain (neural tube defects) in case of folate and Vitamin B12 deficiency anemia in pregnancy.
•Maternal infection (most often, pneumonia, urinary tract infections [UTIs], and endometritis)
•Pregnancy-induced hypertension
•Heart failure

•Pulmonary infarction
•Fetal growth restriction
•Preterm delivery
•Low birth weight

PREVENTION:

To prevent anemia during pregnancy, make sure you get enough iron. Eat well-balanced meals and add more foods that are high in iron to your diet.

•Lean red meat, poultry, and fish
•Leafy, dark green vegetables (such as spinach, broccoli, and kale)
•Iron-enriched cereals and grains
•Beans, lentils, and tofu
•Nuts and seeds
•Eggs

Foods that are high in vitamin C can help your body absorb more iron. These include:

•Citrus fruits and juices
•Strawberries
•Kiwis

CONCLUSION:

Prevalence of Anemia among pregnant women is one of the major risks to a Childs health and it threatens women's mortality During pregnancy and after complications. Awareness among women related to properly nutrition plays an important role in decreasing the risks, and an early detection, prevention and proper set of measures to help the pregnant women during the process of pregnancy reducing my other risk factors and specific signs and symptoms that affects her and fetal health.

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Aortic Aneurysm

Aortic Aneurysm is a condition in which there is a permanent localized bulge or dilation of the wall of aorta greater than 1.5 times of the expected normal diameter of aorta.

EPIDEMIOLOGY:

- 7 out of 100,000 people die with aortic aneurysm.

TYPES:

On the basis of localization

- Thoracic Aortic Aneurysm
  - Ascending Thoracic Aorta
  - Aortic Arch Aneurysm
  - Descending Thoracic Aorta
- Abdominal Aortic Aneurysm

On the basis of shape

- **Fusiform Aneurysm:** Bulge or Balloon out on all side of the aorta (More Common).
- **Saccular Aneurysm:** Bulge or Balloon out on only one side of aorta.

ABDOMINAL AORTIC ANEURYSM:

Abdominal aneurysm is a condition in which the diameter of abdominal aorta becomes greater than 3cm. It is the most common type of aortic aneurysm. Usually occurs between renal arteries and iliac arteries. It is more common in old people.

Cause:

- Atherosclerosis

Risk Factos:

- Hypertension
- Tobacco Use
- Old Age
- Male Gender
- Family History

Clinical Feature:

Asymptomatic. May present as palpable pulsatile Abdominal Mass.
In case of rupture: Acute abdominal and back pain, rapid heartbeat and Hypotension. Patients usually die before reaching the hospital.

INVESTIGATION:

Abdominal Ultrasound.
Contrast CT scan.
Aortography.

TREATMENT:

Beta Blockers and Roxithromycin to reduce expansion rate in small aneurysm.
Surgery if aneurysm is more than 5cm.
Endovascular repair by aortic stenting can also be performed through femoral arteries.

THORACIC AORTIC ANEURYSM:

Thoracic Aortic Aneurysm occurs when the diameter of thoracic aorta becomes greater than 5cm. Thoracic aortic aneurysm accounts for only 10% of cases. Ascending aortic aneurysm is associated with Marfan Syndrome while descending and aortic arch is caused by Atherosclerosis.

Risk Factos:

- Hypertension
- Connective Tissue Disorder (e.g. Marfan Syndrome)
- 3^0 Syphilis

Clinical Feature:

- Pain in chest, back or neck.
- Dyspnea (due to pressure on trachea).
- Dysphagia (due to pressure on esophagus).
- Neck or arm edema (due to pressure on superior vena cava).

INVESTIGATION:

Chest X-ray.
CT scan.
Aortography.

TREATMENT:

Control of Hypertension by using beta blockers.
Surgery in case of rapid expansion or size is more than 6cm.

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1-First Aid
2-Dr Inam Danish.

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Sudden Death Due to Cardiac Diseases

Introduction:

The death occurs suddenly within 1 hour of the outbreak of symptoms or when a person dies suddenly and without witnesses within 24 hours of being spotted and in a healthy state before the body's discovery is called Sudden death (SD) (1). Cardiac arrhythmias are strongly linked to an increased risk of cardiovascular problems and premature death, which lowers the quality of life, increases mortality and raises healthcare costs. (2).

Epidemiology:

In the modern world, the number of deaths has decreased several times during the past two decades but still, nearly 20% of deaths occur so unexpectedly and suddenly, (3). According to the World health organization (WHO), data among the top ten leading causes of death Ischemic heart disease is at first number most leading cause of death in the world(4). Sudden cardiac death is a very destructive event for any family and community

Causes:

There may be several cardiac anomalies reasons for sudden deaths like Hypertrophic cardiomyopathy (HCM) which is the most common inherited disorder in young adults (6), or there may be Arrhythmogenic Right Ventricular Cardiomyopathy (7) or Those who have valvular heart disease (VHD) are susceptible to Brady- and tachyarrhythmias, which can result in SCD.(8) . The most common cause is coronary artery disease in older patients but in the case of young adults, the most common cause is structural heart disease (5). However Sudden death can also occur in non-cardiac conditions like septic shock due to respiratory viruses (9), pulmonary aspergillosis (10), acute pulmonary embolism with Covid-19 (11), and brainstem malformation (12). SCD though is very rare in athletes but it gets much public attention and an underrated cause of sudden death in male athletes is hypertrophic cardiomyopathy (13).

Mortality Rate:

In the United States and Europe, an estimated 0.1% of the population has an out-of-hospital cardiac arrest each year. Males and those between the ages of 66 and 68 are more likely than females to experience sudden cardiac arrest (SCA) (14). Despite the general decline in cardiovascular mortality over the past few decades, around 50% of all cardiac fatalities are sudden. This percentage has not changed over time. SCD accounts for 13% of all natural fatalities; if the criteria is expanded to include the first 24 hours of symptoms, that percentage rises to 18.5% (15). The key issue with SCD is that most out-of-hospital sudden cardiac arrests happen to people in whom cardiac arrest is the first clinical manifestation of the underlying illness or in whom the disease has already been recognized but was deemed to be low risk. There is an inverse connection between incidence and absolute number of events, indicating that a major fraction of the overall population burden arises from subgroups with lower risk scores, making detection and prevention of future occurrences particularly challenging. (15)

Conclusion:

In the story of sudden heart problems, the mix of issues with the heart and unexpected things happening creates a complicated tale. Even though we have better healthcare now, sudden heart problems are still a big challenge, especially for those with hidden heart issues or seen as low risk. Technology, like wearable devices and genetic screening, steps in to help find those at risk early. Other than heart issues, infections and unexpected problems add more layers to how sudden heart problems happen. Community awareness plays a big role in teaching people how to help when someone suddenly has heart trouble. In this ongoing story, our working together in research and collaboration gives hope for a future where the sad ending of sudden heart problems becomes rarer. With each heartbeat, we move towards a story where life wins over unexpected challenges

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