Griffith University Medicine Society
Gold Coast, Australia
GUMS

Year 1 Peer Based Learning 2020
Systems - Cardiovascular

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Case 1

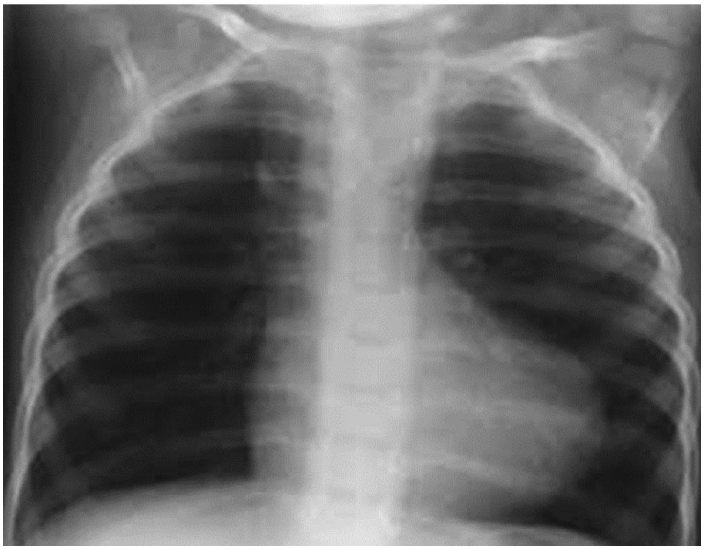
38 year old Terra presents to the GP with her 3 month old baby boy, Fallon, after noticing he is turning blue more frequently. The first few incidents were when Fallon was crying but recently he has also turned blue whilst feeding. Terra also mentions when she took him to his previous check up he was underweight and was below average height for his age. As the GP, you listen to Fallon's heart and notice a murmur.

1. **List the possible causes of Fallon's symptoms**
    - Tetralogy of Fallot's
    - Transposition of great vessels
    - Persistent truncus arteriosus
    - Tricuspid Atresia
    - Total Anomalous pulmonary venous return
    - Ebstein's Anomaly

2. **What further tests would you order?**
    - ECG
    - Echocardiogram
    - Chest X Ray

You refer Fallon to a pediatric cardiologist who orders the above tests. The ECG shows right ventricular hypertrophy and on the chest X Ray the heart is "boot" shaped. There is an ejection systolic murmur in the pulmonary area and no signs of systemic congestion

3. What is the most likely diagnosis considering the new information and what other signs would you see
    - **Tetralogy of Fallot's**
    - **Echocardiography:**
        - detection of the main features of TOF,
        - quantification of right ventricular outflow tract pressure gradient
    - **Chest X-ray:**
        - "Boot"-shaped heart
        - Decreased pulmonary vascularity (Pulmonary Oligaemia)
        - Right aortic arch
        - Cardiac apex is upturned
    - Tet spells



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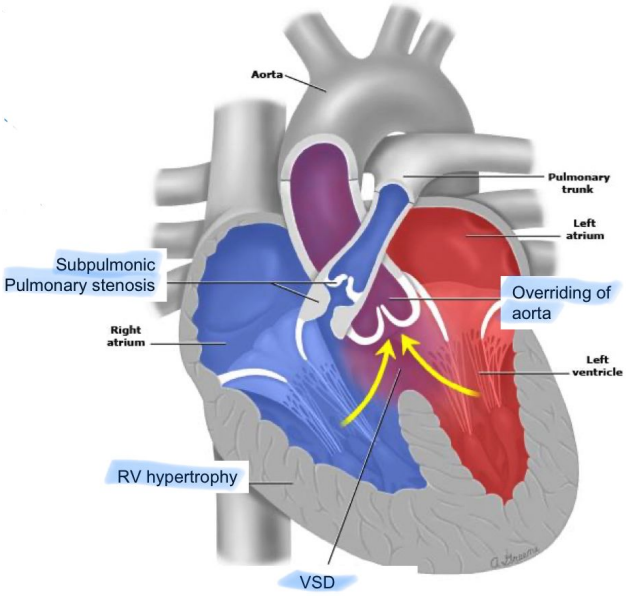
4. **What is Tetralogy of Fallot and what are the 4 main features**
    - Most common cyanotic congenital heart disease
    - Associated with the defective development of the IV septum and Spiral septum - unequal division of TA and VSD
    - 4 features are
        - Pulmonary Stenosis (subpulmonic - below the valve)
        - VSD
        - Overriding of aorta
        - Right ventricular hypertrophy, secondary to increased afterload



5. **What are Tet spells in Tetralogy of Fallot**
    - Episodes of intense cyanosis, sudden onset SOB and irritability
    - Associated with psychological and physical stress (e.g. crying, feeding, defecation)
    - Children tend to squat - may help to increase systemic vascular pressure reducing right to left shunt

6. **Which of the following cause a left to right shunt**
    - Aortic stenosis - no shunt
    - Atrial septal defect (ASD) - correct
    - Tricuspid Atresia - causes a right to left shunt
    - Ventricular septal defect (VSD) - correct
    - Patent Ductus Arteriosus - correct
    - Coarctation of aorta - no shunt
    - Tetralogy of Fallot - causes a right to left shunt

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Case 2

56 year old, Ronald Mcdonald presents to the emergency department complaining of severe chest pain at rest and it radiates down his left arm

7. **What immediate differential do you have? Name two other signs or symptoms you would ask/look for?**
    - Myocardial infarction
    - Other Sx:
        - Dyspnoea
        - Diaphoresis
        - Nausea, vomiting
        - Fatigue
        - Palpitations

8. **What are the tests you would do to confirm your diagnosis**
    - **ECG and cardiac biomarkers**
        - ECG should be performed immediately once acute coronary syndrome is suspected, followed by measurement of cardiac biomarkers
    - **ECG**
        - A 12 lead ECG is the best initial test
    - **Cardiac biomarkers**
        - Serum troponin T is the most important cardiac-specific marker and may be measured 3–4 hours after the onset of myocardial infarction.
        - CK-MB values correlate with the size of the infarct, reach a maximum after approximately 12–24 hours, and normalize after only 2–3 days, making CK-MB a good marker for evaluating reinfarction.
        - Additional findings may also include elevated inflammatory markers (↑ WBC, CRP)

Biomarker/enzyme	Rise*	Maximum*	Normalization*	Characteristics
Troponin T/I	6-8 h	12–24 h	7–10 days	• Cardiac-specific with **high sensitivity for myocardial ischemia** • The degree of elevation often correlates with the size of the infarct. • High sensitivity troponin assays (HscTn) may detect an increase in serum troponin level as early as 90 to 180 minutes after myocardial ischemia has occurred [21] • Can also be elevated in other cardiac and noncardiac conditions: See differential diagnosis of increased troponin below.
Myoglobin	~ 1 h	4–12 h	24 h	• Nonspecific marker that is no longer commonly used
CK-MB	~ 4–9 h	12–24 h	2–3 days	• CK-MB is more specific to cardiac tissue than total CK. • Can be helpful for **evaluating reinfarction** because of its short half-life but is no longer commonly used • The degree of elevation often correlates with the size of the infarct.

* The values rise, reach a certain maximum, and normalize in the span of hours or days following the onset of myocardial infarction or its symptoms. Values and time references may vary based on the precise laboratory methods employed.

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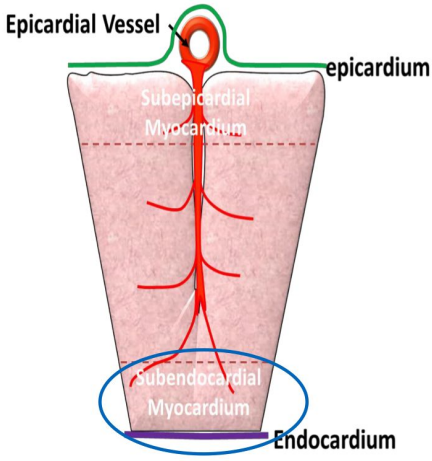
A ECG is performed on Ronald and it shows ST depression and T wave inversion.

9. **Based on the ECG findings name to differentials that can cause this and what is common to both of these differentials in terms of involvement of the myocardium**
    - **Unstable angina and NSTEMI**
    - Both usually affect the inner layer of the myocardium just below the endocardium (subendocardial infarction) > ST depression
    - The subendocardial myocardium is more vulnerable to ischemia since the coronary arteries are in the epicardium and they penetrate into the myocardium
        - The terminal branches that supply the subendocardial myocardium are subject to compression by high ventricular luminal pressure during systole
    - Ruling out….
        - Stable angina - can also present with ST depression however in the initial stem it states he has chest pain at REST
        - STEMI - causes a transmural infarction which leads the ST ELEVATION
    - However, **this may still be a STEMI! If this patient had ST depression in the anterior leads, you should put a set of ECG leads on their back (V7, V8, V9) - these may reveal ST elevation! And therefore you have a posterior STEMI instead of an anterior NSTEMI!**



10. **Complete the table below comparing the types of Acute Coronary Syndrome (ACS)**

	Unstable Angina (UA)	**NSTEMI**	**STEMI**
Description	patients with ischemic symptoms suggestive of an ACS and no elevation in troponin, with or without ECG changes indicative of ischemia (eg, ST-segment depression or transient elevation or new T wave inversion	Acute myocardial ischemia that is severe enough to cause detectable quantities of myocardial injury biomarkers but without ST-segment elevations on ECG	Acute myocardial ischemia that is severe enough to cause ST-segment elevations on ECG

Clinical presentation	**Classic presentation**
	• Acute retrosternal chest pain
	  ○ dull , squeezing
	  ○ Commonly radiates to left chest, arm shoulder, neck, jaw and or epigastrium
	  ○ Precipitated by exertion or stress
	
	Autonomic symptoms may be present:
	• diaphoresis, anxiety
	• syncope, dizziness, lightheadedness
	• palpitations,
	• nausea, and/or vomiting		

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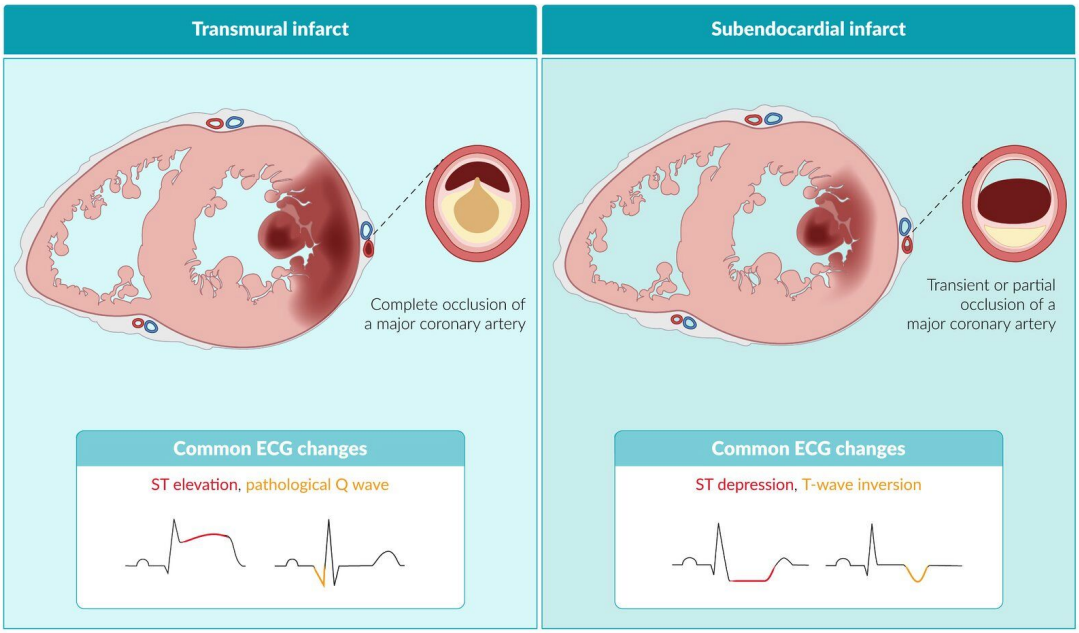
	• Dyspnoea • Pallor **Other findings** • Tachycardia, arrhythmias • Congestive heart failure (e.g., orthopnea, pulmonary edema) or cardiogenic shock (e.g., hypotension, tachycardia, cold extremities) • New heart murmur on auscultation (e.g., new S4) **More common in inferior wall infarction** • Epigastric pain • Bradycardia **STEMI classically manifests acutely with more severe symptoms, while unstable angina/NSTEMI has a continuous course with milder symptoms.**		
Pathophysiology	Partial coronary artery occlusion • Decreased myocardial blood flow → supply-demand mismatch → myocardial ischemia • Usually affects the inner layer of the myocardium (subendocardial infarction)		Classically due to complete occlusion of a coronary artery Affects full thickness of the myocardium (transmural infarction)
Cardiac biomarkers	No elevated cardiac biomarkers	Elevated cardiac biomarkers (e.g., troponin)	Cardiac biomarkers elevated (e.g., troponin)
ECG findings	Normal or may get ST depression, loss of R wave or T wave inversion ST-segment and/or T wave electrocardiographic changes are often persistent in NSTEMI, while, if they occur in UA, they are usually transient. May see reciprocal depressions		**ST elevations** (in two contiguous leads) or new **left bundle branch block, hyperacute T waves** Sign of **old** infarction = pathological Q waves, poor R wave progression



Picture from Amboss - Acute coronary syndrome

11. What type of patients are likely to present with Atypical signs of MI

- Elderly, diabetic individuals and females
- Minimal to no chest pain and Autonomic symptoms are often the chief complaint
- In patients with diabetes - chest pain may be completely absent (e.g. silent MI) due to polyneuropathy

12. Explain the rationale for giving nitrates in these conditions

- More marked dilation of veins than arteries
- Rapid dilation of peripheral venous vessels - causes pooling of blood in veins
    - Decreases ventricular blood volume (preload)
    - Decreases cardiac workload
    - Decrease myocardial oxygen demand
- Dilation of large muscular arteries - reduces peripheral resistance
    - Decreases cardiac workload
    - Decreases myocardial oxygen demand
- Produces dilation of coronary arteries - directly increases oxygen supply to myocardium

13. What is an important consideration in the use is nitrates

- Nitrate tolerance
    - Follows continuous use (>24 h)
    - Due to the exhaustion of catalyst (-SH/thiol groups) in smooth muscles of blood vessels
    - Endothelial catalyst is required for the conversion of nitrates to nitric oxide
    - Responsiveness can be restored by interrupting therapy for 8-12 hours each day (nitrate free period)

Ronald was also found to have elevated troponins (NSTEMI) and the doctors on rotation were from UQ and did not commence treatment straight after doing the initial ECG. A repeat ECG is taken and Ronald is found to have ST elevation now

14. Describe what happened here?

- The point of this question is to discuss the natural history of MIs.
- Initially, when the MI has just occurred, only their subendocardium was affected. Due to lack of treatment over this time span, they have now had a transmural infarction leading to ST elevation - this is a STEMI.

The ECG shows ST elevation in leads V5, V6, I, aVL, II, III and aVF. He also developed a systolic murmur best heard at the 5th intercostal space, left midclavicular line.

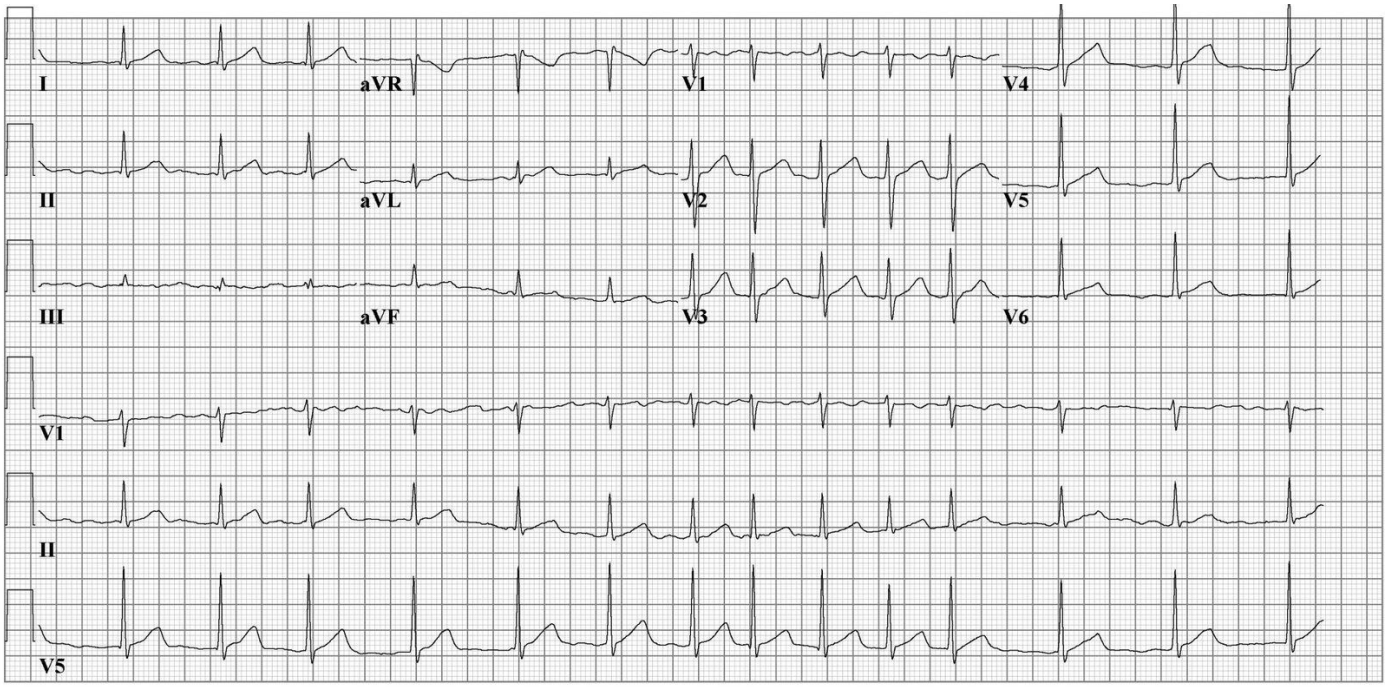
15. Account for the ECG changes by stating an infarction site in a single location

- V5, V6, I, aVL = lateral surface which is supplied by left circumflex artery (LCx)
- II, III, aVF = inferior surface which can be supplied by the right coronary artery (RCA) or the posterior descending artery (PDA)
- Therefore for a single site to cause ST elevation in all 7 of the above leads the patient must have a left dominant circulation (PDA branches from the LCx) and therefore a LCx infraction causes a infarction in both LCx and PDA territory

16. Why has Ronald developed a murmur

- Papillary muscle rupture leading to mitral regurgitation
- Usually occurs 2-7 days after MI
- In this case it's the rupture of the posteromedial papillary muscle due to occlusion of the posterior descending artery - this is the most common due to single blood supply by the PDA
- Clinical features
    - New holosystolic, blowing murmur over the 5th ICS on the midclavicular line
    - Signs of acute mitral regurgitation: dyspnoea, cough, bilateral crackles, hypotension
- the anterolateral papillary muscle is usually preserved since it has dual supply from the LCx and LAD

Ronald then goes on to develop the following ECG. (12 lead ECG (paper speed: 25mm/s)



ECG from Amboss - Acute coronary syndrome

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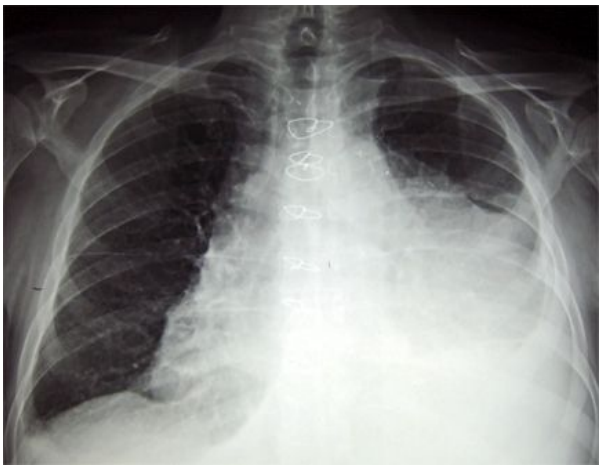
17. State the diagnosis and explain two mechanisms which could have caused this to arise

- Atrial fibrillation
  - Irregular ventricular rate approx. 66/min and 120/min
  - Normal cardiac axis
  - Irregular RR intervals with narrow QRS complexes
  - No discernable P waves
- Mechanisms
  I. Mitral regurgitation → blood enters left atrium during ventricular systole → leads to left atrial dilatation over time → distends cardiomyocytes → disrupts conduction of electrical signals between cells → atrial fibrillation
  II. MI → necrosis of cardiomyocytes disrupts conduction → atrial fibrillation

18. Following the MI and AF, what is the most important cardiac condition to monitor Ronald for and how can it arise?

- Congestive Heart failure
- Due to a combination of myocardial stunning, myocyte necrosis, decompensation of pre-existing heart failure or acute mitral regurgitation due to papillary muscle dysfunction and can be compounded by fluid or contrast overload, renal dysfunction, or complications such as ventricular septal defect or cardiac tamponade.
- Late heart failure reflects the consequences of cardiomyocyte death and scar formation occurring alongside ventricular remodelling.

19. Account for the following x-ray following the infarction. What is the diagnosis and state the key clinical signs and symptoms you would expect.



- **Cardiac tamponade due to left ventricular free wall rupture**
  - Ventricular free wall rupture is a complication of myocardial infarction in which the ventricular wall ruptures due to weakening of the infarcted region.
  - A free wall rupture typically occurs within 2 weeks of an anterior or lateral wall infarct and presents with new-onset chest pain and cardiogenic shock due to cardiac tamponade.
- On Xray, since there is more fluid in the pericardial cavity, which extends around the whole heart, all heart borders are enlarged - called **'water bottle sign'**

- **Signs and Symptoms**
  - Chest pain
  - Dyspnoea
  - Signs of cardiac tamponade
    - **Beck triad - hypotension, muffled heart sounds, distended neck veins**
    - Tachycardia, pulsus paradoxus
    - pallor , cold sweats
    - Left ventricular failure
    - Symptoms of right heart failure (e.g. fluid retention, peripheral pitting oedema)
    - Cardiogenic shock, cardiac arrest

Case 3

Penny G presents to the ED feeling generally unwell with aching joints, fever, cough and painful nodules on the hands and feet.

20. List the possible differential diagnoses and state which two are the most likely

- **Infective endocarditis**
- **Acute Rheumatic fever**

- Other possible casues
  - Atrial myxoma
  - Systemic lupus erythematosus
  - Renal cell carcinoma
  - Carcinoid syndrome

Because you are a Griffith student you undertake a physical examination and a history. You examine her heart and find a holosystolic murmur in the fifth intercostal space at the midclavicular line. You notice she has a fever of 38.5^{o}C and has poor dental hygiene. Penny also states she went to the dentist for a clean two days ago. You send her blood for two separate cultures which come positive for staphylococcus aureus and a Transthoracic echocardiography (TTE) is performed which detects vegetations on her mitral valve.

21. What is the diagnosis

- Infective endocarditis

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22. How would you differentiate between the two most likely diagnoses - Fill in the table below for both

	Infective endocarditis	Rheumatic fever
Signs & symptoms	**Constitutional symptoms** Fever and chills (~ 90% of cases), tachycardia, General malaise, weakness, night sweats, weight loss Dyspnoea; cough, pleuritic chest pain Arthralgias, myalgias **Extracardiac manifestations** These manifestations are mainly caused by bacterial microemboli and/or the precipitation of immune complexes **Petechiae;** especially splinter hemorrhages **Janeway lesions, Osler nodes** **Roth spots:** round retinal hemorrhages with pale centers **Signs of acute renal injury,** including hematuria and anuria; due to renal artery occlusion or glomerulonephritis **Neurological manifestations; septic embolic stroke** **Signs of pulmonary embolism** **Possible arthritis**	**Constitutional symptoms**: fever, malaise, fatigue **Joints:** migratory polyarthritis **Skin** **Subcutaneous nodules** **Erythema marginatum:** centrifugally expanding pink or light red rash with a well-defined outer border and central clearing - Painless and nonpruritic **CNS:** **Sydenham chorea** (involuntary, irregular, nonrepetitive movements of the limbs, neck, head, and/or face) - Occurs 1–8 months after the inciting infection - **Pathophysiology:** Streptococcal antigens lead to antibody production → antibodies cross-react with structures of the basal ganglia; and cortical structures

Cardiac manifestations	New heart murmur development or change to a preexisting one
	Mitral valve regurgitation → holosystolic murmur, loudest at the heart's apex, and radiates to the left axilla
	Tricuspid valve regurgitation → holosystolic;
	Aortic valve regurgitation → early diastolic murmur
	Signs of progressive heart failure (e.g., dyspnoea, oedema)
	Signs of acute cardiac decompensation (pulmonary oedema)
	Arrhythmias
		Pancarditis (endocarditis, myocarditis, and pericarditis)
		Valvular lesions: most commonly on high-pressure valves
		• **Mitral valve** (~ 65% of cases) -
		  ○ Early mitral regurgitation or prolapse
		  ○ Late mitral stenosis: Rheumatic fever is the most frequent cause of mitral stenosis.
		  ○ Mixed mitral stenosis/regurgitation
		• **Aortic valve** (~ 25% of cases)
		• **Tricuspid valve** (~ 10% of cases)
		Dilated cardiomyopathy due to severe valvular disease, **myocarditis**

Diagnostic criteria	**Dukes criteria** - To confirm the diagnosis, one of the following requirements must be met: • Two major criteria OR • One major and three minor criteria OR • Five minor criteria **Major diagnostic criteria** • Two separate blood cultures positive for typical pathogens • Evidence of endocardial involvement in echocardiography ○ A new valvular regurgitation **Minor diagnostic criteria**	**Jones criteria -** Two major criteria or one major plus two minor criteria or three minor criteria are required for diagnosis. **Please see amboss table image below this table for major and minor criteria**

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	• Predisposition: underlying heart disease or IV drug abuse • Fever ≥ 38°C (100.4F) • Vascular abnormalities • Immunologic disorder • Microbiology	
Epidemiology/risk factors	**Incidence increases with age (age > 60 years old)** **Male to female ratio of 2:1** **Pre Existing conditions** Pre Damaged or prosthetic heart valves Congenital heart defects **Bacteremia** Infected peripheral venous catheters, surgery, dental procedures Non-sterile venous injections (e.g., IV drug abuse)	Peak incidence: 5 - 15 years old More common in females than males more commonly in resource-limited countries More common in indigenous australian populations compared to general population - higher rates in northern territory
Pathogens	**Staphylococcus aureus** (45–65%) **Viridans streptococci** (30%): S. sanguinis, S. mutans, S. mitis **Staphylococcus epidermidis** **Enterococci,** especially Enterococcus faecalis (< 10%) **Streptococcus gallolyticus (S. bovis)** **Gram-negative HACEK group (H**aemophilus species**,** **A**ggregatibacter actinomycetemcomitans, **C**ardiobacterium hominis, **E**ikenella corrodens, **K**ingella kingae) **Candida species -** Aspergillus fumigatus **Coxiella burnetii -** Bartonella species	Previous infection with **group A β-hemolytic streptococcus (GAS)**, also referred to as **Streptococcus pyogenes** Usually acute tonsillitis or pharyngitis (“strep throat”)

	Low risk population	High risk population
Major criteria	• Arthritis (migratory polyarthritis involving primarily the large joints) • Carditis (pancarditis, including valvulitis) • Sydenham chorea (CNS involvement) • Subcutaneous nodules • Erythema marginatum	• Arthritis (monoarthrits or migratory polyarthritis) • Polyarthralgia • Carditis (pancarditis, including valvulitis) • Sydenham chorea (CNS involvement) • Subcutaneous nodules • Erythema marginatum
Minor criteria	• Polyarthralgia • Elevated body temperature (≥ 38.5°C) • ↑ Acute phase reactants (ESR ≥ 60 mm/h, CRP ≥ 3.0 mg/dl) • Prolonged PR interval on electrocardiogram	• Monoarthralgia • Elevated body temperature (≥ 38°C) • ↑ Acute phase reactants (ESR ≥ 30 mm/h, CRP ≥ 3.0 mg/dl) • Prolonged PR interval

Table from amboss- Jones Criteria for rheumatic fever

23. What is the pathogenesis of Penny’s condition

- **Pathogenesis:** localized infection or contamination → bacteremia → bacterial colonization of damaged valve areas → formation of fibrin clots encasing the vegetation → valve destruction with loss of function

- **Frequency of valve involvement:** mitral valve > aortic valve > tricuspid valve > pulmonary valve
- **Clinical consequences**
  - Bacterial thromboemboli from bacterial vegetation → vessel occlusion with infarctions
  - Formation of immune complexes and antibodies against tissue antigens → glomerulonephritis; Osler nodes

24. Why does this condition cause a heart murmur?

- Lesions develop on the valves, called vegetations, and are composed of fibrin, platelets, infecting organisms and are clumped ("agglutination") together by antibodies produced by the bacteria
- As inflammation continues, ulceration may result in erosion or perforation of the valve cusps, leading to valvular incompetence, damage to the conduction pathway (if in the septal area), or rupture of a sinus of Valsalva (if in the aortic area).

25. What is the most likely affected valve if IV drug users? What other patients groups are also likely to have this valve involved?

- The tricuspid valve is the most commonly affected valve in IV drug users (associated with Pseudomonas, S. aureus, and Candida).
- HIV infection; immunosuppressed patients,; patients with central venous catheters
- Possible explanations as to why tricuspid more likely to be affected in IV drug users include:
  - damage to right-side cardiac valves as a result of repeated bombardment by particulate matter in IV drugs
  - right-side valve vasospasm, intimal damage, and thrombus formation induced by injected drugs;
  - drug-induced pulmonary hypertension and increased right-side cardiac turbulence

Please provide feedback for this case at:
https://gums2020.typeform.com/to/e0h7US

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