**acute pericarditis**

**defn** - inflammation of the pericardium characterised by chest pain, pericardial friction rub & ECG changes

**symptoms**

1. chest pain - sharp, dull, aching, burning or pressing - intensity varies from barely perceptible to severe - pain is usually precordial with referral to the trapezius ridge - worse with inspiration, lying flat, with swallowing & movement
2. dyspnoea - may be present (especially with tamponade) 3. fever may be present 4. abdominal pain occurs infrequently in children

**signs**

1. pericardial friction rub - pathognomonic of acute pericarditis - may only be detected transiently - more than 50% are triphasic composed of:
   (i) atrial systolic rub preceding S1
   (ii) ventricular systolic rub (between S1 & S2)
   (iii) early diastolic rub (after S2)
2. tachypnoea may be present
3. tachycardia may be present
4. patient may have fever

**aetiology**

2. infectious - viruses including coxsackie B, echo virus, adenovirus, influenza, enterovirus, mumps virus, HSV, VSV, RSV. Viral pericarditis is usually short & self limiting after 1-2 weeks - bacterial develops from direct pulmonary extension, haematogenous, myocardial abscess or endocarditis, penetrating chest injury or subdiaphragmatic collections. Commonly leads to constrictive pericarditis - Tb should be considered in all subacute cases. It has a mortality of 50%.

- other infectious causes include fungal & parasitic

**investigation**

1. Bloods: - FBE, ESR & CRP, Cr - cardiac enzymes are elevated in 32% with viral pericarditis & are related to extent of myocardial inflammation - in selected patients ASO titres, rheumatoid factor, ANA, anti-disDNA, thyroid function

2. imaging - CXR - echocardiography is performed if tamponade is suspected on radiological grounds or if the illness lasts for > one week; it is also useful to look for regionality where there is diagnostic doubt

3. ECG (i) stage 1: - accompanies onset of acute pain - is characterised by diffuse concave upward ST elevation except in aVR & V1 which are usually depressed - T waves are upright in all leads with ST elevation & PR segment deviates opposite to P-wave polarity (ii) stage 2: - occurs several days later - ST's return to baseline & there is flattening of T waves (iii) stage 3: - T waves become inverted (iv) stage 4: - ECG returns to baseline weeks after onset

**treatment**

1. oxygen & cardiac monitoring 2. rule out other life-threatening causes of chest pain 3. treat pain with NSAIDs 4. emergency treatment of tamponade (under USS guidance if required) 5. antibiotics or anti-Tb therapy if appropriate

**disposition**

- mortality varies depending on the aetiology - it is near 100% in untreated purulent pericarditis & virtually zero in viral or idiopathic pericarditis

- many patients can be managed as an outpatient - echo is needed if symptoms persist longer than one week - the following risk factors indicate poor prognosis & suggest inpatient management should be considered:
  (i) fever >38
  (ii) immunosupression
  (iii) trauma
  (iv) oral anticoagulants
  (v) myocarditis
  (vi) pericardial effusion or tamponade

- refer patients with viral pericarditis to cardiologist - complicated causes such as Tb, purulent & uraemic aetiologies require multidisciplinary involvement

- patient has been discharged but returns with persistent or recurrent pericarditis & evidence of myopericarditis or congestive heart failure - consider pericardial biopsy or thoracoscopic biopsy

**Disposition**

1. Congenital - familial Mediterranean fever
2. Infectious
   - viruses including coxsackie B, echo virus, adenovirus, influenza, enterovirus, mumps virus, HSV, VSV, RSV. Viral pericarditis is usually short & self limiting after 1-2 weeks - bacterial develops from direct pulmonary extension, haematogenous, myocardial abscess or endocarditis, penetrating chest injury or subdiaphragmatic collections. Commonly leads to constrictive pericarditis - Tb should be considered in all subacute cases. It has a mortality of 50%.

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3. Non-infectious
   - RA is associated with pericardial effusion in 50% with nodules and only 15% without.
   - Symptomatic disease tends to occur in those with other extra-articular manifestations
   - SLE flare-ups may be associated with pericarditis
   - sarcoidosis, Siogrens, Reiter, ankloising spondylitis, immunotubulitic bowel disease, Wegener's, vasculitis, polymyositis, Bechets, Whipple disease & serum sickness.
   - after myocardial infarction fibrinous pericardial exudate appears within 24hrs & may lead to a rub within 24hrs or as late as 10 days. Pericardial pain occurs less frequently than the rub. Dressler's syndrome is usually observed 2-3 weeks after a myocardial infarction & may occur after pulmonary embolism - it may be autoimmune in nature.

4. Post-infectious
   - rheumatic fever (consider in all children with percarditis). Pericarditis usually appears 7-10 days after the onset of fever & arthritis

5. Neoplastic
   - most commonly metastatic (lung>breast>leukaemia & lymphoma>melanoma)

6. Metabolic
   - renal failure leads to uraemic pericarditis

7. Drugs, Poisons, Toxins
   - penicillin hypersensitivity
   - doxorubicin & cyclophosphamide have direct cellular toxicity
   - drug-induced SLE caused by procainamide, hydralazine, methyldopa, isoniazid & reserpine can cause pericarditis
   - radiotherapy can cause pericarditis

8. Iatrogenic:
   - similar to Dressler syndrome except that it occurs after cardiac surgery

9. Idiopathic:
   - common but difficult to distinguish from viral

**aetiology**

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look for signs of tamponade:

1. Beck's triad of elevated JVP, hypotension & muffled heart sounds
2. pulsus paradoxus is defined as 10mmHg decrease in arterial systolic pressure with inspiration

**Disposition**

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