Myocarditis (inflammatory disease of the heart muscle)

<table>
<tr><td>how bad is it?</td><td colspan="2">❖ it varies.. some patients present with few symptoms(flu-like symptoms) and some present with severe symptoms with progression to CHF & dilated cardiomyopathy</td></tr>
<tr><td rowspan="2">Etiology</td><td>infectious</td><td>non infectious</td></tr>
<tr><td>1. Viruses
◆ Coxsackie B(the most common cause)
◆ others: coxsackievirus A, HIV, ..etc
2. Bacterial (corynebacterium diphtheriae, syphilis) it could be a complication of bacterial endocarditis
3. Protozoan (Trypanosoma cruzi → Chagas disease)
4. Spirochete (Borrelia burgdorferi → Lyme disease)
“think outside the box :)”</td><td>1. Systemic diseases (SLE, Sarcoidosis, Vasculitides[Wegener’s], Celiac disease)
2. Neoplastic infiltration
3. Drugs & toxins (Ethanol, Cocaine, Radiation, Chemotherapeutic agents - Doxorubicin)</td></tr>
<tr><td>Clinical presentation</td><td colspan="2">● days to weeks after onset of acute febrile illness
● it may present with heart failure without any known antecedent symptoms
● Fever, headache, muscle aches, diarrhea, sore throat and rashes “similar to any viral infection”
● chest pain, arrhythmias, sweating, fatigue.</td></tr>
<tr><td>Diagnosis</td><td colspan="2">↑WBCs, ↑ESR, ↑Troponins and ↑CK-MB
ECG (nonspecific ST-T changes and conduction delays are common)
Blood cultures
Serology; Viral serology and other specific test for Lyme, diphtheria and Chagas disease.
Radiology: Chest X-rays show cardiomegaly + pulmonary edema, MRI and Echocardiogram
Heart muscle biopsy.</td></tr>
<tr><td>Management (supportive/ symptomatic)</td><td colspan="2">● Restricted physical activity in heart failure.
● If infecting agent is identified → specific antimicrobial therapy
● Treatment of heart failure arrhythmia
● Other drugs in special situations (anticoagulant, NSAID, steroid, immunosuppressive ..etc)
● Heart transplant (in progressive cases)</td></tr>
<tr><td>prognosis</td><td colspan="2">● Most cases of viral myocarditis are self limited.
● ⅓ of the patients are left with lifelong complications, ranging (mild conduction defects - severe HF) so patients should be followed regularly every 1-3 months.
● Sudden death may be the presentation of myocarditis in about 10% of cases.</td></tr>
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pericarditis (inflammation of pericardium usually of infectious etiology)

<table>
<tr><td>Etiology</td><td colspan="4">❖ Viruses Coxsackie A and B, echoviruses (the most common cause)
❖ Bacterial Pericarditis usually complication of pulmonary infections
(S. pneumonia, M. tuberculosis, S. aureus, H. influenzae, K. pneumoniae and legionella)
❖ HIV patients may develop pericardial effusions (tuberculosis, M.avium complex).
❖ Disseminated fungal infection (Histoplasma, Coccidioides)
❖ Parasitic infections (rare) (toxoplasma gondii, Entamoeba histolytica).</td></tr>
<tr><td>How?</td><td colspan="4">● Contiguous spread, hematogenous spread, lymphangitic spread, traumatic or irradiation</td></tr>
<tr><td>Pathophysiology</td><td colspan="4">fibrinous exudate with/without serous effusion + dull, opaque, “sandy” pericadium + adhesions and fibrosis</td></tr>
<tr><td rowspan="2">Types of pericarditis</td><td>Caseous pericarditis</td><td>Serous pericarditis</td><td colspan="2">Fibrous Pericarditis</td></tr>
<tr><td>TB in origin</td><td>Autoimmune diseases (RA, SLE)</td><td colspan="2">chronic Pericarditis encased in scar tissue</td></tr>
<tr><td rowspan="2">Types of Effusive Fluid</td><td>Serous</td><td>transudative - heart failure</td><td>Hemorrhagic</td><td>with any type but especially with infections & malignancies</td></tr>
<tr><td>Suppurative</td><td>pyogenic infection with cellular debris and ↑ leukocytes</td><td>Serosanguinous</td><td>with any type</td></tr>
<tr><td>Tuberculous Pericarditis (common here in KSA!)</td><td colspan="4">● Physical findings: fever, pericardial friction rub, hepatomegaly
● TB skin test usually positive, fluid smear for TB often negative
● Pericardial biopsy more definitive</td></tr>
<tr><td>Clinical presentation</td><td colspan="4">● Sudden pleuritic chest pain(very sharp), fever, dyspnea and friction rub (in auscultation).
● On examination exaggerated pulsus paradoxus JVP and tachycardia(↓venous return).
● As the pericardial pressure increases, palpitations presyncope or syncope may occur.</td></tr>
<tr><td>Diagnosis</td><td colspan="4">● ECG will show ST elevation, PR depression and T-wave inversion may occur later.
● Leukocytosis and ↑ ESR are typical
● Other routine testing urea and creatine.
● Blood culture
● Chest x-ray may show enlarged cardiac shadow or calcified pericardium
● CT scan show pericardial thickening >5mm.</td></tr>
<tr><td>Management</td><td colspan="4">● supportive for cases of idiopathic and viral Pericarditis including bed rest and NSAIDS, Colchicine.
● Corticosteroid is controversial and anticoagulants usually contraindicated.
● If infecting agent is identified → specific antimicrobial therapy
● Antiviral (acyclovir → herpes simplex or varicella) (ganciclovir → cytomegalovirus).
● Pericardiocentesis to relief tamponade.</td></tr>
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Infective endocarditis (Infection or colonization of endocardium, heart valves, congenital defects)

<table>
<tr><td>mortality</td><td colspan="3">30% with antibiotic :(</td></tr>
<tr><td rowspan="2">classification</td><td>acute IE</td><td colspan="2">subacute IE</td></tr>
<tr><td>• Affects normal heart valves
• Rapidly destructive
• Metastatic foci
• Commonly Staph. aureus, strept.pneumoniae
• If not treated, usually fatal within 6 weeks</td><td colspan="2">• Often affects damaged heart valves
• Indolent nature
• if not treated, usually fatal by one year
• can follow dental procedures in patients with: RHD, congenital heart disease.</td></tr>
<tr><td rowspan="2">Predisposing factors</td><td>cardiac lesions → blood flow stasis</td><td colspan="2">systemic factors</td></tr>
<tr><td>• Chronic rheumatic valvular disease
• Congenital heart disease and defects
• Atherosclerosis
• Prosthetic valves</td><td colspan="2">• Immunosuppressive treatment
• Immune defects (disease)
• Alcoholism
• IV Drug abuse</td></tr>
<tr><td>pathogenesis</td><td colspan="3">• Formation of vegetations (fibrin + platelets + bacteria colonies) attached to heart valves → break off → infected emboli → stroke, MI, ischemic limbs, hypoxia, splenic or renal infarction
• Immune complex formation causes glomerular damage → haematuria
• Valves infection → destruction → heart failure .
• Drug addicts → valves of right side of heart (T,P) →lung emboli → pneumonia</td></tr>
<tr><td rowspan="3">Factors affecting severity & outcome</td><td>bacterial factors</td><td colspan="2">virulence / bacteria in the blood</td></tr>
<tr><td rowspan="2">host factors</td><td>factors ↑ susceptibility</td><td>protective factors</td></tr>
<tr><td>• Local (congenital or rheumatic heart disease, prosthetic heart valves, other cardiovascular disease, heart surgery)
• General (underlying disease → e.g: diabetes.M)
• Drugs:
✗ Iatrogenic → Immunosuppressive , cytotoxic agents
✗ Self- inflicted → alcoholism, addiction (injected drugs)</td><td>antimicrobial chemotherapy</td></tr>
<tr><td>clinical presentation</td><td colspan="3">• Fever (mild and prolonged), malaise, weight loss, weakness
• Changing murmurs
• Anaemia , leucocytosis, ↓complement levels
• Microscopic haematuria
• Petechiae (red or purple spots due to bleeding) → extremities, mucous membranes
• Splenomegaly
• Splinter haemorrhage (fingernail hemorrhage)
• Hypergammaglobulinemia
• osler’s node (painful & erythematous nodules located on pulp of fingers and toes) (subacute IE)</td></tr>
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<table>
<tr><td>laboratory diagnosis</td><td colspan="2">

serial blood culture (2-3 sets before antibiotic therapy)
serological tests: CFT for (coxiella burniti)
sensitivity test
Chest x-ray: look for multiple focal infiltrates and calcification of heart valves
ECG: rarely diagnostic/ look for evidence of ischemia, conduction delay, and arrhythmias
Echocardiography (YOU CAN SEE THE VEGETATION!)

</td></tr>
<tr><td rowspan="2">spread of infection</td><td>local</td><td>metastatic</td></tr>
<tr><td>

Heart failure (extensive valvular damage)
Paravalvular abscess (30-40%):

✗ common in aortic valve/IVDA/S. aureus
✗ extend into adjacent conduction tissue “arrhythmia”
✗ ↑ rates of embolization and mortality

Pericarditis
Fistulous intracardiac connections

</td><td>

Metastatic abscess:
(Kidneys, spleen, brain, soft tissues)
Meningitis and/or encephalitis
Vertebral osteomyelitis
Septic arthritis

</td></tr>
<tr><td>treatment</td><td colspan="2">

Criteria of antibiotic

Bactericidal (β-lactam) + synergistic drug (aminoglycoside)
Parenteral
High dose
Prolonged (4 weeks at least)

</td></tr>
<tr><td>Antimicrobial prophylaxis (prevention)</td><td colspan="2">

For susceptible patients (rheumatic valvular or congenital heart disease)
before tooth extraction, deep scaling: benzyl-penicillin (I.M) 30 mins before penicillin (oral)
If patient is allergic to penicillin: Vancomycin or Erythromycin (I.V) 30 minutes befor operation.
Genitourinary procedures: gentamicin +ampicillin (I.V) 30 minutes before operation

</td></tr>
<tr><td>microorganism</td><td>notes</td><td>treatment (β-lactam + aminoglycoside)</td></tr>
<tr><td>strept. Viridans</td><td>

oral normal flora
patient with congenital heart disease or patient suffering from rheumatic heart disease who did dental procedure will develop subacute IE

</td><td>Benzyl-penicillin or
penicillin + gentamicin</td></tr>
<tr><td>Strept. Faecalis</td><td>

endocarditis after genitourinary or gut procedures

</td><td>ampicillin + gentamicin</td></tr>
<tr><td>S. aureus</td><td>

acute endocarditis in IV drug abuser
it usually affects tricuspid valve (from pathology)
poor prognosis

</td><td>cloxacillin + gentamicin</td></tr>
<tr><td>S. epidermidis</td><td>

endocarditis if the patient has prosthetic heart valves

</td><td>-</td></tr>
<tr><td>candida albicans</td><td>

endocarditis if the patient is immunocompromised
RARE
poor prognosis

</td><td>-</td></tr>
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* important notes:

1. always ask for blood culture if you suspect any infection, it may help
2. make sure that the specimen is formalin-free when you send it to microbiology lab (formalin kills microorganisms)
3. the parasite toxoplasma gondii causes toxoplasmosis "this disease can be passed on from cats to humans"
4. there are 3 types of bacteremia:
    - transient → lasts for minutes to hours
    - intermittent → occur with undrained abscesses
    - continuous or persistent → endocarditis - endarteritis - suppurative thrombophlebitis - infected aneurysm -in the first two weeks of typhoid fever and brucellosis.
5. in acute endocarditis a high virulent microorganism will damage the endocardium, while in subacute endocarditis the endocardium is already damaged and th will attract the microorganisms to infect that area.
6. in endocarditis the complement levels are decreased.