Infectious diseases of the heart

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Infectious diseases of the heart

- Infective endocarditis
- Myocarditis
- Pericarditis
Infective endocarditis
Epidemiology

- IE is a relatively rare but **serious disease** with **high mortality!!!** despite the improvement in dg. and th.
- Estimated annual incidence 3-10/100 000
- The profile of patients and pathogens has changed over time (rheumatic fever x PM/ICD)
- **Predisposing factors:**
  - prosthetic valves
  - elderly patients with degenerated valves
  - i.v. drug users
  - i.v. catheters, pacemaker electrodes
Classification

• (Acute x subacute/ lenta)

• NVE - native valve endocarditis
• PVE - prosthetic valve endocarditis
• IVDU - intravenous drug users
• IE on PM / ICD electrodes
Classification

- **Relaps** - repeat IE within 6 months / proven identical pathogen
- **Reinfection**

- **Early PVE** - within 1 year (usually aggressive nosocomial infection of sewing material)
- **Late PVE** - beyond 1 year after implantation
Pathophysiology

• IE is rare in healthy individuals despite common bacteremia (dental procedures, toothbrushing...)
• Any injury to endocardial surface (degenerative changes, impact of catheters, electrodes, prosthetic materials...) → endocardial damage, exposing EC matrix → F III, platelet activation, fibrin-platelet (sterile) vegetation → increase risk of bacterial seeding
NVE

- Rheumatic valvular disease – usually mitral valve followed by the aortic valve
- Congenital heart disease - patent ductus arteriosus, ventricular septal defect, tetralogy of Fallot, or any native or surgical high-flow lesion
- Mitral valve prolapse
- Degenerative heart disease - calcific aortic stenosis in elderly / due to a bicuspid valve, Marfan syndrome, rarely syphilitic disease
Clinical presentation

• Variable!
• Fever (95%), signs of systemic disease (nausea, weight loss....)
• Heart murmur (85%)
• Septic embolization (20-50%)
  - brain, kidneys, spleen
  - pulmonary
• Peripheral microembolization less common
Diagnostic testing

Blood cultures

• 3 sets (aero + anaerobe) at different times and from diff. sites
• 85-90% known from first 2 sets (streptococci, staphylococci, enterococci)
• 10% culture negative
  • (usually due to previous ATB th.)
  • less commonly HACEK (*Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella*)
• Fungi – Candida, Aspergillus
• Intracellular pathogens: Coxiella, Bartonella, Chlamydia, Mycoplasma, Legionella, Treponema
Diagnostic testing

Echocardiography

- TTE - low sensitivity (40-60%)
- TEE - sensitivity 90 - 100%
- vegetations / abscess / new prosthetic valve dehiscence = specific
- new regurgitation / obstruction = not specific
Treatment

• Primary **ATB therapy**
• **Surgery** should be performed in high-risk patients
  • Age/comorbidities/PVE/DM
  • Complicated IE (heart failure, shock...)
  • High-risk agents (S.aureus, fungi...), ATB failure
  • TTE/TEE high-risk morphology parameters – risk of embolisation
Antibiotics

- beta-lactam (penicillin, cefalosporin) or
- glykopeptide (vancomycine)
- +/- aminoglykosides (gentamicin) - intensify/shorten th.
- +/- rifampin in PVE
Antibiotics

- **Streptococci**: PEN/CEF + GENTA, VANCO in PEN allergy
- **Enterococci**: like streptococci, PEN resistance common
- **Staphylococci**: MET/OXA + GENTA
- **Empiric therapy** - should focus on S. aureus
- **HACEK/early PVE/fungi** - require expert ATB consult
- **PVE** - th. like NVE but prolonged (min. 6w) + RIFAMPIN
Antibiotics

• Duration of therapy usually 4-6 weeks (regardless of surgery)
• No fever and decrease in CRP are good markers of therapy success, TTE follow-up (<2 weeks) necessary
• Cessation of therapy in case of:
  • normal CRP (1w), favourable TTE, no embolisation (2w), absence of focus for potential reinfection
Surgery

- progressive heart failure (emergency in shock)
- signs of ATB th. failure - continuous fever, abscess, vegetation, valve dehiscence..
- embolization potential (>15mm)
PVE

• 1% / patient / 1 yr
• risk: mechanical = biological
• most common manifestation – valve regurgitation
• long-term sterilisation rare - surgery likely !!!

• early PVE = aggressive pathogens in sewing material rapidly spreading into surroundings
• late PVE = NVE like
PM/ICD IE

- IE anywhere on/close to electrodes
- S. aureus most likely
- Electrode withdrawal necessary (embolisation during withdrawal common, rarely clinically significant)
IVDU

- Most commonly Tricuspid valve
- S. aureus, pseudomonas, G-, fungi, polymicrobial
- Fever, septic pulmonary embolisation (cough, hemoptysis, pulmonary abscesses, ...)
- Mortality <10%, but high likelihood of recurrence, surgery common
Prevention

- Restrictive approach
- High risk patients only
  - Prosthetic valve implant
  - Previous IE
  - Congenital Heart Disease patients
- High-risk procedures (dental)
Myocarditis
Overview

• Inflammatory disease of the myocardium with a wide range of clinical presentations, from subtle to devastating
• Usually manifests in otherwise healthy person
• Wide variety of infectious organisms, autoimmune disorders and exogenous agents

• **Acute phase** (0-2wks): direct cytotoxic effect + cell mediated cytotoxicity
• **Chronic phase** (>2wks): mainly autoimmune
Etiology

• “Idiopathic” in 50% of cases
• Viruses - Enterovirus, Coxsackie A,B, Adenovirus, Parvovirus B19, Influenza, CMV, EBV, HCV, HIV...
• Bacterial - streptococci, TBC, diptheria, Borrelia, Chlamydia, Mycoplasma
• Fungi – Aspergillus, Candida
• Protozoa – Trypanosoma cruzii
• Drugs - anthracyclines, cocaine
• Rheumatic fever
• Autoimmune disorders – SLE, Sarcoidosis, Sjögren sy, Churg-Strauss sy, Wegeners granulomatosis, „giant-cell“ myocarditis"
Clinical presentation

• Patients may refer recent flu-like symptoms, arthralgias, and malaise or pharyngitis, tonsillitis, upper respiratory tract infection, fever
• Chest pain, sweats, dyspnea, palpitations
• Signs of heart failure, shock
• Palpitations, syncope, or sudden cardiac death due to underlying ventricular arrhythmias or AV block
Diagnostic workup

• Complete blood count, Erythrocyte sedimentation rate level, rheumatologic screening, **cardiac enzymes**
• Viral antibody titers - rarely indicated, low specificity and delayed rising, no impact on therapeutic decisions
• ECG - nonspecific - sinus tachycardia, ST-T changes, AV blocks
• Echocardiography (TTE) - to estimate dysfunction and rule-out other causes
Diagnostic workup

• SKG - to rule-out CAD
• MRI (late gadolinium enhancement) - extent of inflammation and cellular edema, nonspecific
• EMB (endomyocardial biopsy) - routine use rarely helpful, mandatory in rapidly progressive HF to rule-out giant-cell myocarditis
Treatment

- Standard HF treatment (ACEi, BB, ARB, diuretics, inotropics,...IABP, LVAD in shock)
- Avoid physical stress (several months)
- Routine use of immunosuppression not recommended
- Immunosuppression used in giant-cell myokarditis or in systemic autoimmune disease (SLE, RA...
Follow-up

- Ongoing, chronic inflammation may cause DCM and subsequent HF
- Patients with a history of myocarditis should be monitored at 1-3 months interval initially, with gradual return of physical activity
- Any evidence of residual cardiac dysfunction should be treated in the same manner as for chronic HF
Prognosis

- Patients who survive fulminant myokarditis generally have good prognosis.
- Most patients with mild symptoms recover completely without any residual cardiac dysfunction, although up to 30% develop D-CMP (dilated cardiomyopathy).
- 90% of patients with giant-cell myocarditis die or undergo Tx in 6 months.
Pericarditis
Clinical presentation

• Acute pericarditis is an inflammation of the pericardium characterized by chest pain, pericardial friction rub, and serial ECG changes

• Pain
  • usually precordial or retrosternal with referral to the trapezius ridge, neck, left shoulder, or arm
  • quality is usually pleuritic, range from sharp, dull, aching, burning, or pressing,
  • intensity varies is worse during inspiration, when lying flat, or during swallowing and with body motion, it may be relieved by leaning forward while seated
Clinical presentation

• Intermittent fever
• dyspnea/tachypnea (!myocarditis, pericarditis, and cardiac tamponade)
• cough, dysphagia
• Any form of pericardial inflammation may cause effusion

!!!! Myocarditis - myopericarditis
Pericarditis - perimyocarditis
Etiology

• Idiopathic causes - most common, about 50%, likely viral
• Infectious
  • Viral – enterovirus, echovirus, parvovirus, EBV, HIV...
  • bacterial, TBC, mycotic (Candida)
• Inflammatory disorders - RA, SLE, scleroderma, rheumatic fever, Reiter sy, dermatomyositis
• Metabolic – renal failure, hypothyroidism,
• Cardiovascular disorders - acute MI, Dressler syndrome,
• Iatrogenic – postpericardiotomic sy, catheterization
• Neoplasms – adjacent / secondary / paraneoplastic
• Drugs, Irradiation
• Trauma, Pneumonia, Pulmonary infarction...
Diagnostic workup

• ECG changes
• chest radiograph (enlarged if effusion >250ml)
• Echo (effusion), CT (calcification), MRI
• Laboratory studies (Complete blood count, ESR, CRP, TnI, electrolytes, BUN, creatinine, thyroid hormones + specific (RF...))
• CAG ?
Treatment

- If specific cause revealed, treat accordingly (ATB)
- Idiopathic or viral treated for symptom relief
  - NSAIDs in full dose for 7-14 days (600-800 mg ibuprofen/day), consider PPI as gastric protection
  - Colchicine (add if recurrence or beyond 14 days, 1 mg/day)
  - Corticosteroids - not for initial therapy, unless specific therapy (AI) or no response to NSAIDs + colchicine
- Pericardiocentesis – large effusions, cardiac tamponade
Treatment

- surgical th. usually when recurrence or large effusions
- pericardial window
- pericarpectomy - in constrictive pericarditis
Complications

• The most common complication of idiopathic acute pericarditis is recurrence, 15 -30 %

Constrictive pericarditis
• - Acute and subacute forms of pericarditis may deposit fibrin → pericardial effusion → further pericardial inflammation, chronic fibrotic scarring, calcification, and restricted cardiac filling
Complications

Cardiac tamponade

• especially in acute pericardial hemorrhage or in large chronic malignant effusions) accumulation of fluid in the pericardial space
• resulting in reduced ventricular filling and subsequent hemodynamic compromise
• medical emergency