

Infectious heart diseases

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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

- **Relapse** - repeat IE within 6 months and proven identical pathogen
- **Reinfection**, - new microorganism, or the same species but > 6 months

- **Early PVE** - within 1 year (usually aggressive nosocomial infection of sewing material)
- **Late PVE** - > 1 year after surgery/implantation



Pathophysiology

- IE is rare in healthy individuals despite common bacteremia (dental procedures, toothbrushing...)

x

- Any **injury to endocardial surface** (degenerative changes, impact of catheters, electrodes, prosthetic materials...) → endocardial damage, exposing EC matrix → factor 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 (TOF), *any native or surgical high-flow lesion*
- **Mitral valve prolapse**
- **Degenerative heart disease**
 - aortic stenosis in elderly ,bicuspid valve, Marfan syndrome, rarely syphilitic disease
 - mitral regurgitation



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 + from diff. sites
- 85-90% - **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, Trephonema



Diagnostic testing

Echocardiography

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

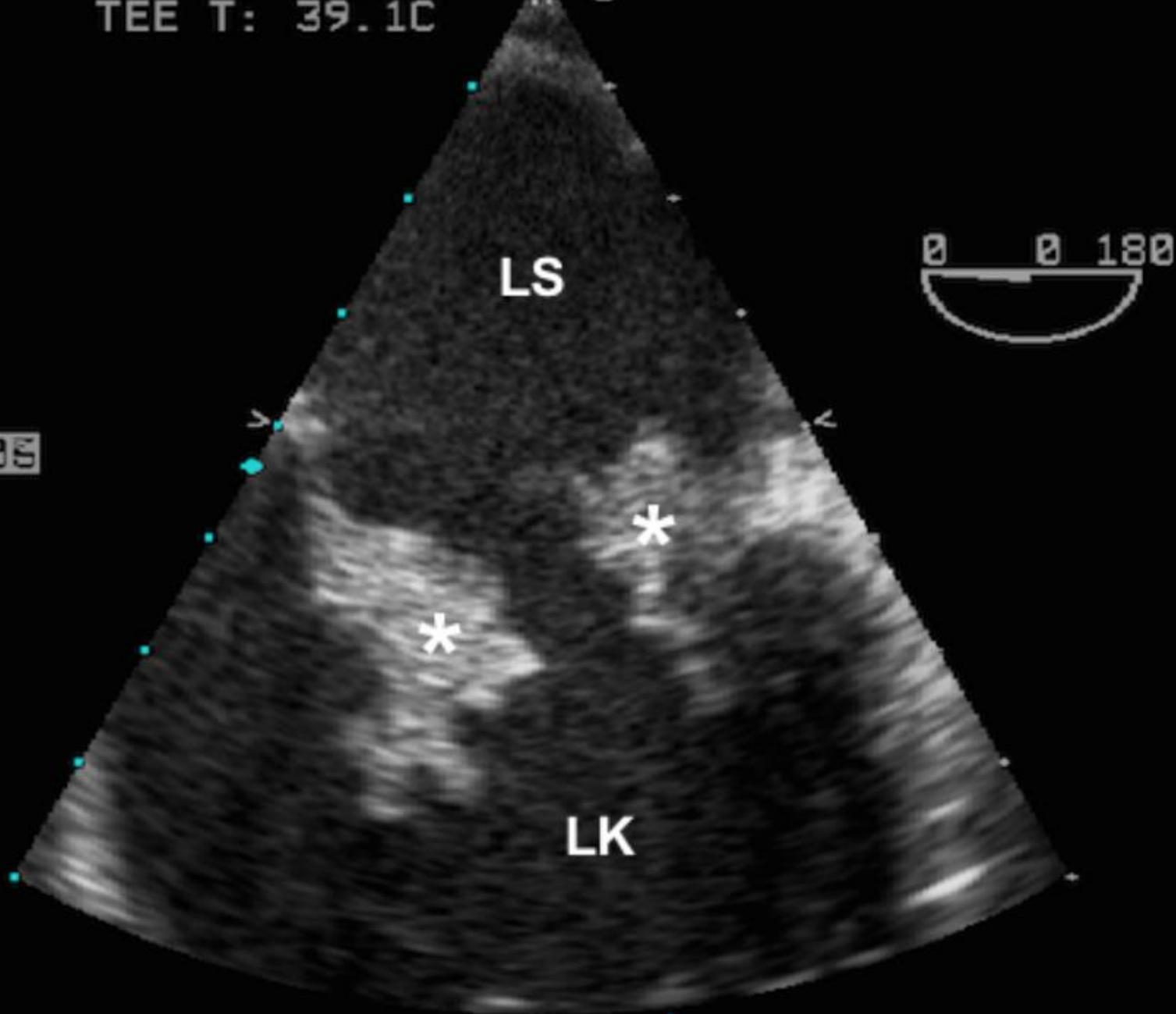


TEE T: 39.1C

044

08
4
3
FNM

X
: 00.05



Definite IE
<p>Pathological criteria</p> <ul style="list-style-type: none"> • Microorganisms demonstrated by culture or on histological examination of a vegetation, a vegetation that has embolized, or an intracardiac abscess specimen; or • Pathological lesions; vegetation or intracardiac abscess confirmed by histological examination showing active endocarditis <p>Clinical criteria</p> <ul style="list-style-type: none"> • 2 major criteria; or • 1 major criterion and 3 minor criteria; or • 5 minor criteria
Possible IE
<ul style="list-style-type: none"> • 1 major criterion and 1 minor criterion; or • 3 minor criteria
Rejected IE
<ul style="list-style-type: none"> • Firm alternate diagnosis; or • Resolution of symptoms suggesting IE with antibiotic therapy for ≤ 4 days; or • No pathological evidence of IE at surgery or autopsy, with antibiotic therapy for ≤ 4 days; or • Does not meet criteria for possible IE, as above

Duke criteria

(IE probability)

ESC guidelines 2015



Major criteria

1. Blood cultures positive for IE

- a. Typical microorganisms consistent with IE from 2 separate blood cultures:
 - *Viridans streptococci*, *Streptococcus gallolyticus* (*Streptococcus bovis*), *HACEK* group, *Staphylococcus aureus*; or
 - Community-acquired enterococci, in the absence of a primary focus; or
- b. Microorganisms consistent with IE from persistently positive blood cultures:
 - ≥ 2 positive blood cultures of blood samples drawn >12 h apart; or
 - All of 3 or a majority of ≥ 4 separate cultures of blood (with first and last samples drawn ≥ 1 h apart); or
- c. Single positive blood culture for *Coxiella burnetii* or phase I IgG antibody titre $>1:800$

2. Imaging positive for IE

- a. Echocardiogram positive for IE:
 - Vegetation;
 - Abscess, pseudoaneurysm, intracardiac fistula;
 - Valvular perforation or aneurysm;
 - New partial dehiscence of prosthetic valve.
- b. Abnormal activity around the site of prosthetic valve implantation detected by ^{18}F -FDG PET/CT (only if the prosthesis was implanted for >3 months) or radiolabelled leukocytes SPECT/CT.
- c. Definite paravalvular lesions by cardiac CT.

Minor criteria

1. Predisposition such as predisposing heart condition, or injection drug use.
2. Fever defined as temperature $>38^{\circ}\text{C}$.
3. Vascular phenomena (including those detected by imaging only): major arterial emboli, septic pulmonary infarcts, infectious (mycotic) aneurysm, intracranial haemorrhage, conjunctival haemorrhages, and Janeway's lesions.
4. Immunological phenomena: glomerulonephritis, Osler's nodes, Roth's spots, and rheumatoid factor.
5. Microbiological evidence: positive blood culture but does not meet a major criterion as noted above or serological evidence of active infection with organism consistent with IE.





Splinter hemorrhage



Osler node



Roth's spot



Janeway lesion



Treatment

- **ATB therapy**
- **Surgery** - 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)
- **glykopeptide** (vancomycine)
- **aminoglykosides** (gentamicin)
- **rifampicin** in PVE

Fungi – ATB centre expert consult



Antibiotics

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



Antibiotics

- Usually **4-6 weeks** (regardless of surgery)
- No fever and decrease in CRP are good markers of th. success, TTE follow-up (< 2weeks) 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** (> 10 mm)



FR 50Hz
15cm

M3

2D
63%
C 50
P Low
HGen



JPEG



KARDIOLOGICKÁ KLINIKA
2. LF UK a FN MOTOL

PVE

- 1% / 1 yr / patient
- risk: mechanical similar to biological (later!)
- most common manifestation – valve regurgitation
- long-term sterilisation rare - **surgery likely !!!**

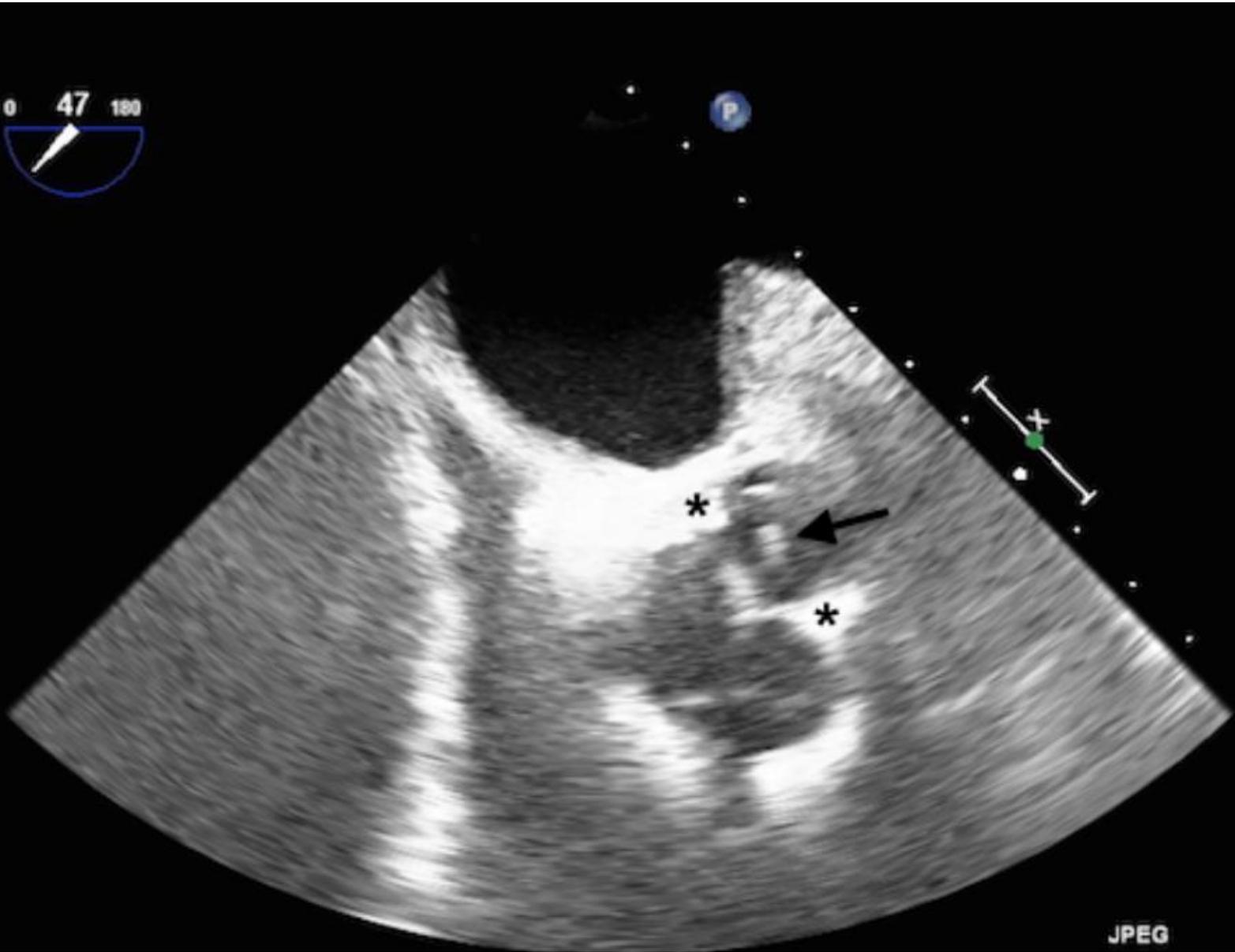
- **early PVE** = aggressive pathogens in sewing material, rapidly spreading into surroundings
- **late PVE** = NVE like



FR 52Hz
11cm

M4

2D
81%
C 50
P Off
Gen



JPEG



KARDIOLOGICKÁ KLINIKA
2. LF UK a FN MOTOL

PM/ICD IE

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



FR 52Hz
10cm

M4

2D
83%
C 50
P Off
Gen



JPEG



KARDIOLOGICKÁ KLINIKA
2. LF UK a FN MOTOL

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)



Recommendations	Class ^a	Level ^b
<p>Antibiotic prophylaxis should be considered for patients at highest risk for IE:</p> <ul style="list-style-type: none"> (1) Patients with any prosthetic valve, including a transcatheter valve, or those in whom any prosthetic material was used for cardiac valve repair. (2) Patients with a previous episode of IE. (3) Patients with CHD: <ul style="list-style-type: none"> (a) Any type of cyanotic CHD. (b) Any type of CHD repaired with a prosthetic material, whether placed surgically or by percutaneous techniques, up to 6 months after the procedure or lifelong if residual shunt or valvular regurgitation remains. 	IIa	C
<p>Antibiotic prophylaxis is not recommended in other forms of valvular or CHD.</p>	III	C



Table 6 Recommended prophylaxis for high-risk dental procedures in high-risk patients

Situation	Antibiotic	Single-dose 30–60 minutes before procedure	
		Adults	Children
No allergy to penicillin or ampicillin	Amoxicillin or ampicillin ^a	2 g orally or i.v.	50 mg/kg orally or i.v.
Allergy to penicillin or ampicillin	Clindamycin	600 mg orally or i.v.	20 mg/kg orally or i.v.

^aAlternatively, cephalexin 2 g i.v. for adults or 50 mg/kg i.v. for children, cefazolin or ceftriaxone 1 g i.v. for adults or 50 mg/kg i.v. for children.

Cephalosporins should not be used in patients with anaphylaxis, angio-oedema, or urticaria after intake of penicillin or ampicillin due to cross-sensitivity.



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, diphtheria, 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

- flu-like symptoms, arthralgias, malaise, fever
- pharyngitis, tonsillitis, upper respiratory tract infection
- Chest pain, sweats, dyspnea, palpitations
- Signs of heart failure, cardiogenic shock
- Syncope or sudden cardiac death due to underlying ventricular arrhythmias or AV block)



Diagnostic workup

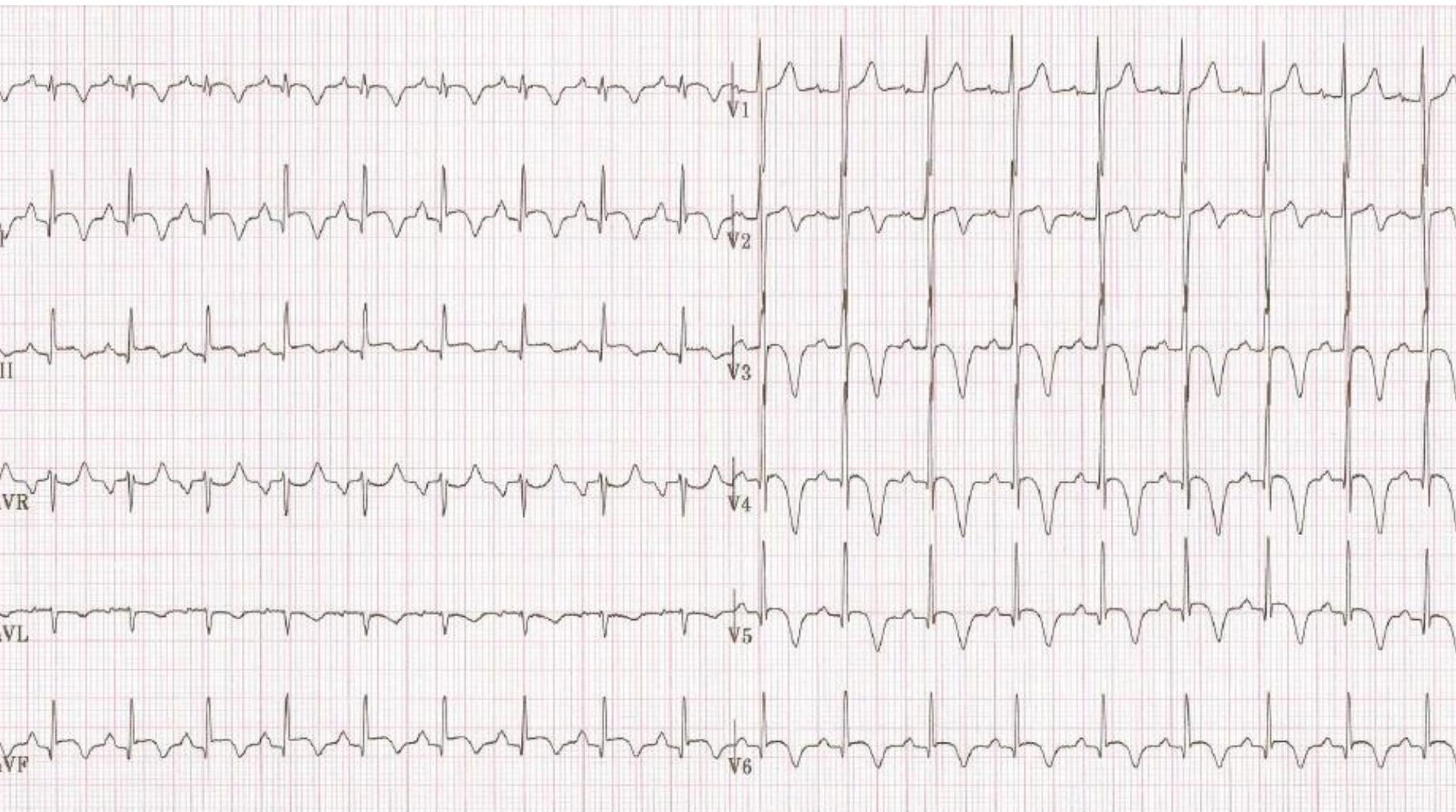
- Complete blood count, erythrocyte sedimentation rate level, CRP, rheumatologic screening, **cardiac biomarkers (Tnl, TnT)**
- Viral antibody titers - *rarely indicated*, low specificity and delayed rising, no impact on therapeutic decisions
- **ECG** - nonspecific - sinus tachycardia, ST-T changes,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



Diagnostic workup

- **CAG** (coronary angiography)- 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 myocarditis or in systemic autoimmune disease (SLE, RA...)



Follow-up

- Ongoing, chronic inflammation may cause DCM (dilated cardiomyopathy) 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 myocarditis generally have good prognosis
- Most patients with mild symptoms recover completely without any residual cardiac dysfunction, although up to 30% develop DCM
- 90% of patients with giant-cell myocarditis die or undergo Tx in 6 months



Pericarditis



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

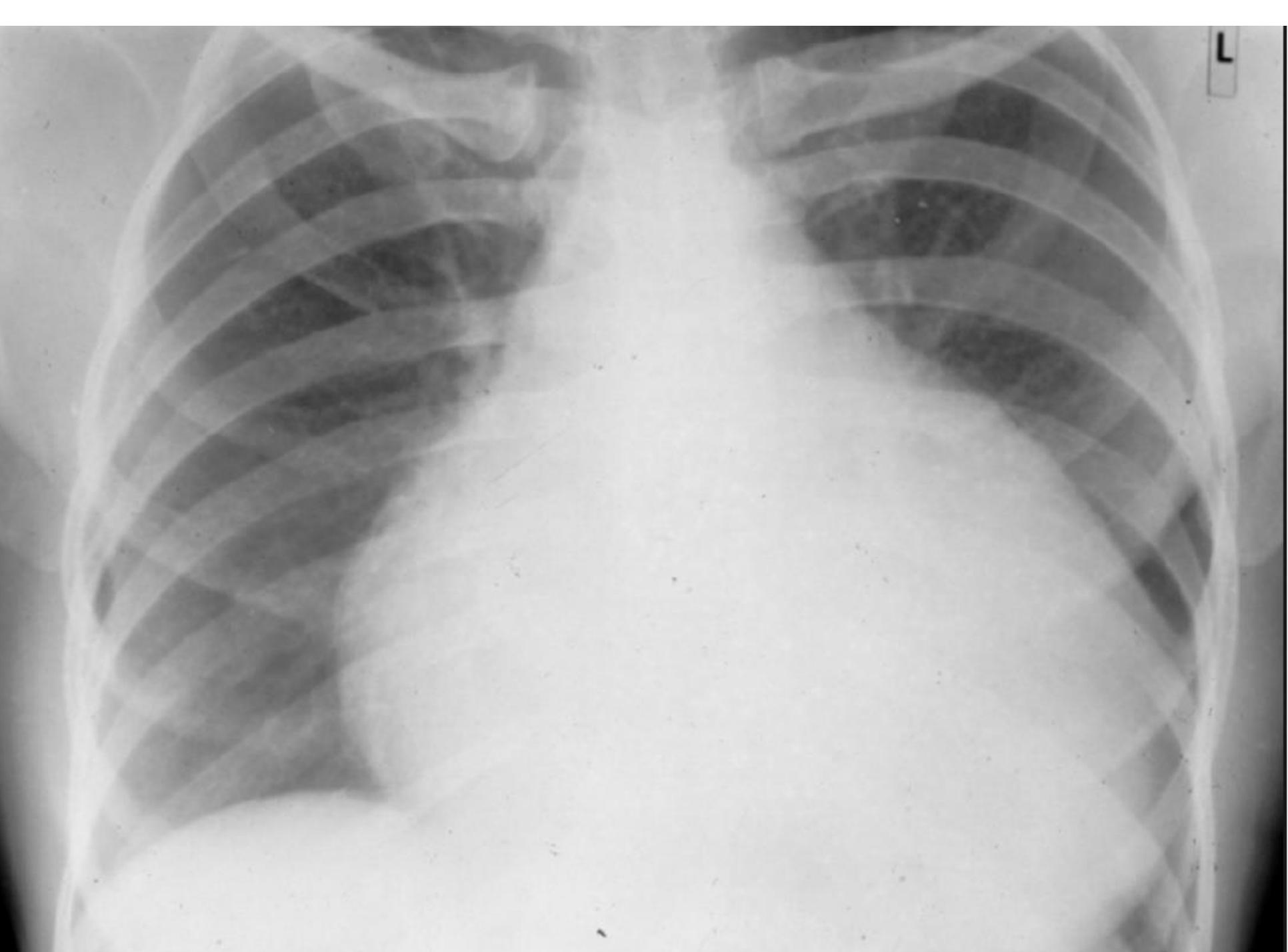
- Idiopathic causes - about 50%, likely viral
- Infectious
 - Viral – enterovirus, echovirus, parvovirus, EBV, HIV...
 - bacterial, TBC, mycotic (Candida)
- Inflammatory disorders - RA, SLE, scleroderma, rheumatic fever, Reiter sy, dermatomyositis
- Metabolic – renal failure, hypothyroidism,
- Cardiovascular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

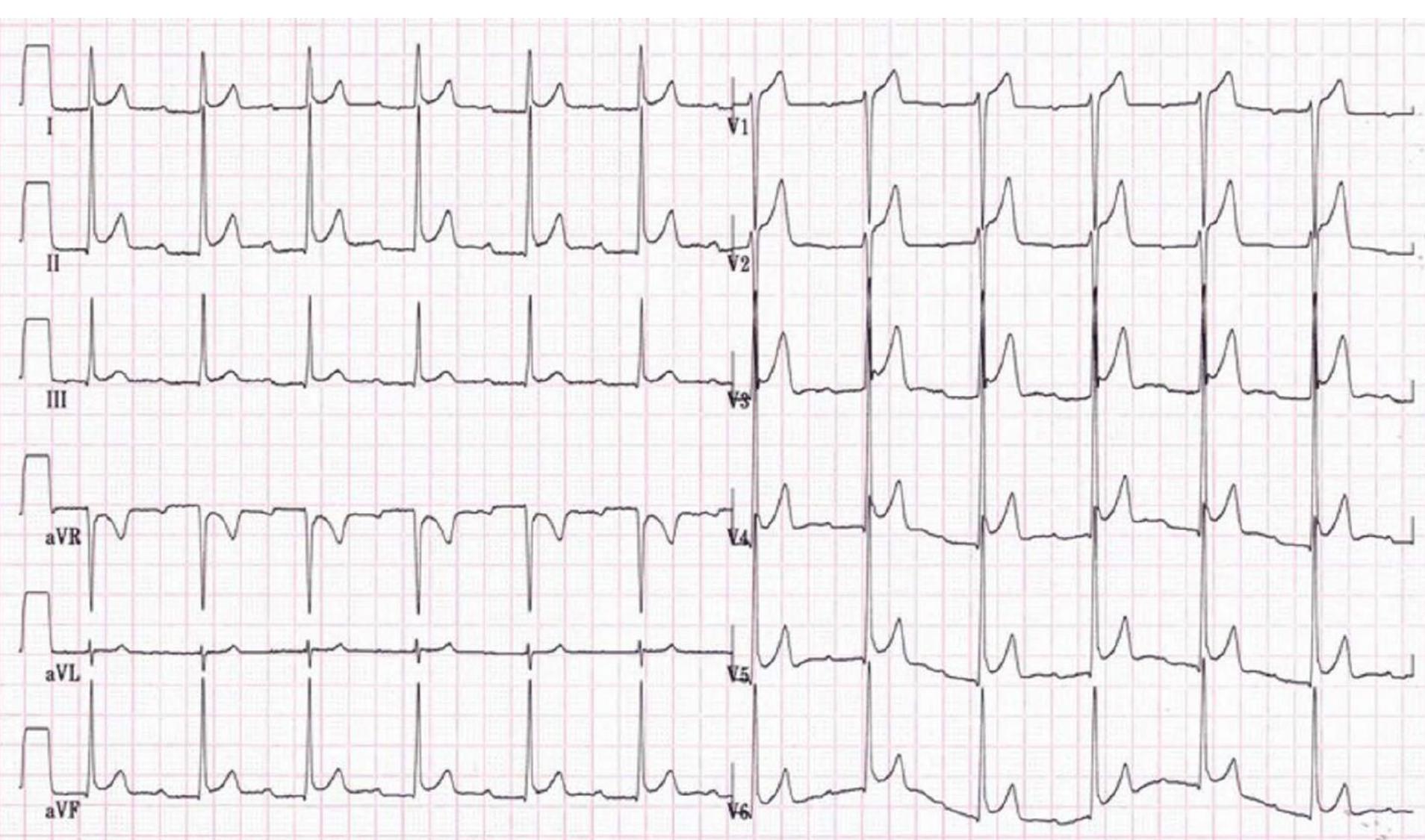


Diagnostic workup

- **ECG** changes
- **chest radiograph** (enlarged if effusion >250ml)
- **TTE** (effusion), **CT** (calcification), **MRI**
- **Laboratory studies** (Complete blood count, ESR, CRP, cardiac TnI/TnT, 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** (recurrence or beyond 14 days, 1 mg/day)
 - Corticosteroids - not for initial therapy, unless specific therapy (autoimmune) or no response to NSAIDs + colchicine
- **Pericardiocentesis** – large effusions, cardiac tamponade



Treatment

Surgical therapy

- usually when recurrence or large effusions
- pericardial window
- pericardectomy - 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 large chronic malignant effusions
- accumulation of fluid in the pericardial space
- resulting in reduced ventricular filling and subsequent hemodynamic compromise
- medical emergency



Complications

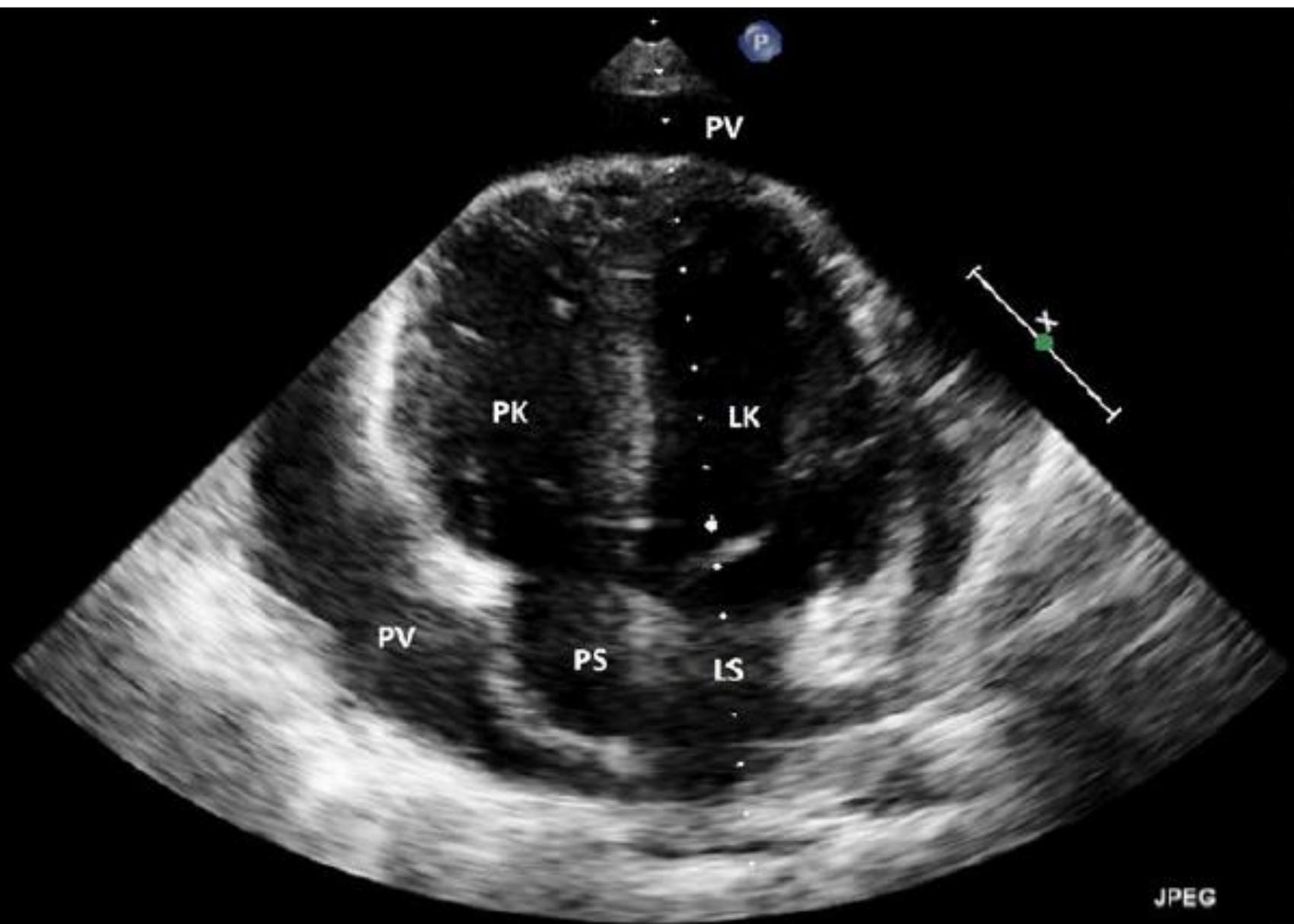
Cardiac tamponade

- Symptoms vary with the acuteness and underlying cause
- **Dyspnea, tachycardia, tachypnea, hypotension**
- **Elevated jugular venous pressure**
- **Pulsus paradoxus**
- chest pain, fever, dysphoria



3%
50
LOW
Gen

Ⓒ
R
3.4



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Thank You.



