



CARDIAC INFECTIONS

Rheumatic Heart Disease and Infective Endocarditis

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RHEUMATIC FEVER RHEUMATIC HEART DISEASE

RF to RHD



“Rheumatic Fever is a disease the LICKS the joints but BITES heart.”

RHD IS THE MOST SERIOUS COMPLICATION OF RF

Rheumatic Fever



- Most common cause of acquired heart disease in children & young adults worldwide
- Diffuse inflammation of connective tissues of heart, joints, brain, blood vessels & subcutaneous tissues
- Rheumatic process causes fibrosis of heart valves leading to RHD

Epidemiology

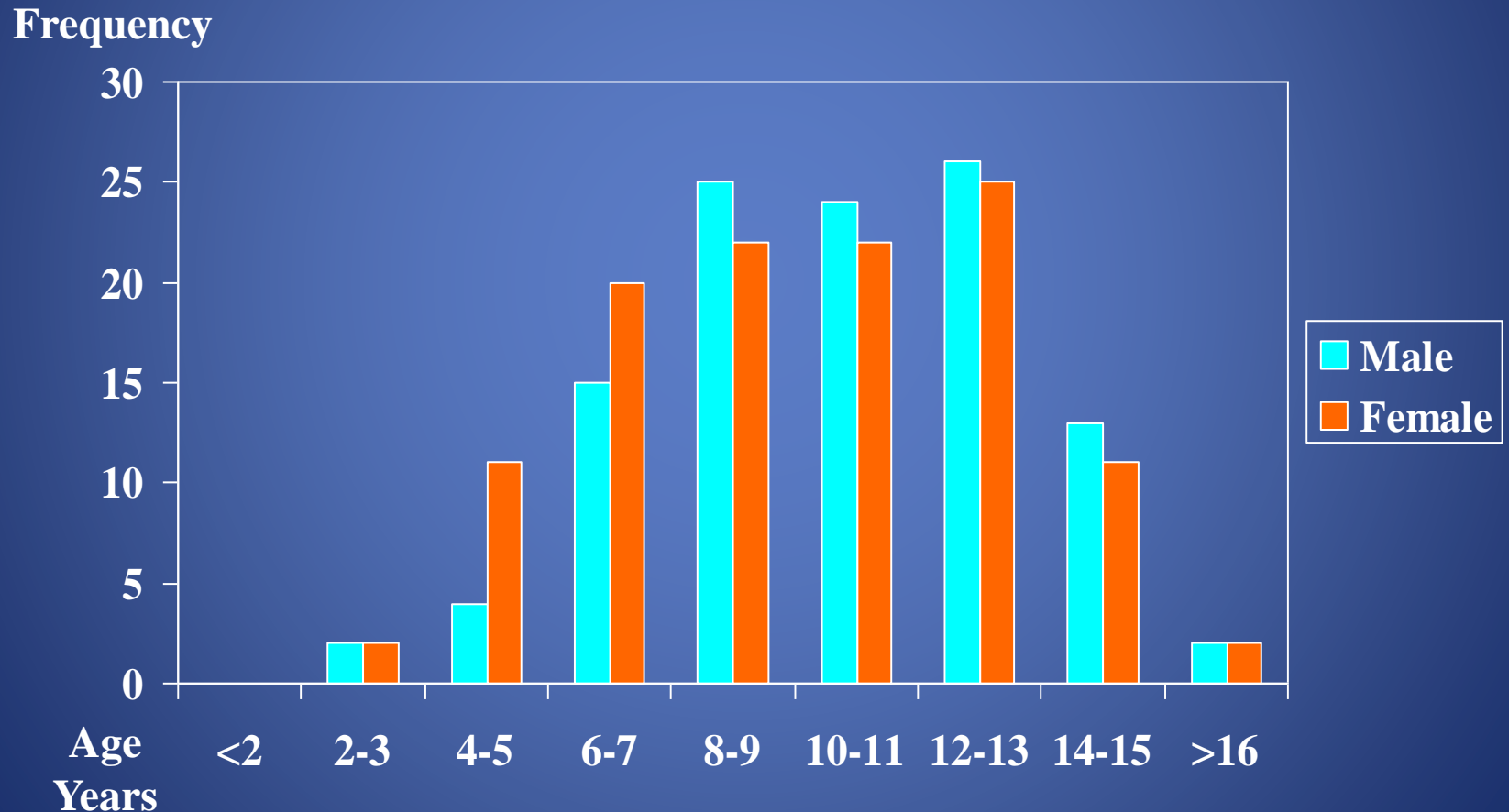


- Incidence of RF and RHD has not decreased in developing countries
- Rheumatic Fever is principally a disease of childhood, with a median age of 10 years, although is also occurs in adults
- Remains a major cause of morbidity and mortality in country



Acute Rheumatic Fever

Age and Sex Distribution (N = 117)



Rheumatic Fever & Rheumatic Heart Disease



Prevalence in school children:

Developing countries 18.6 / 1000

Philippines 1971-1980 0.9 / 1000

1981-1990 0.6 / 1000

Developed countries

USA 1971-1980 0.7 / 1000

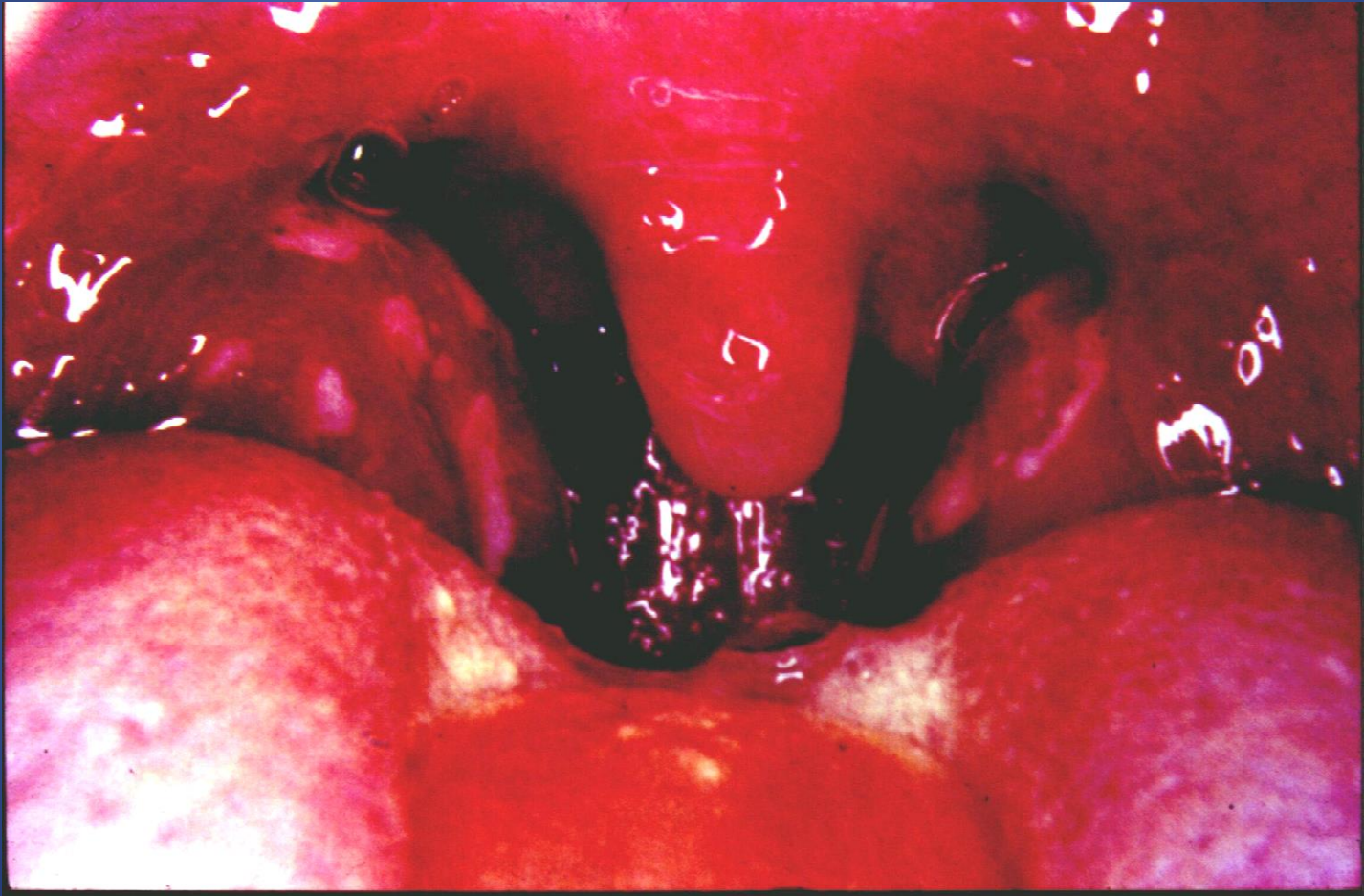
Australia 1981-1990 12.3 / 1000

Group A β -Hemolytic Streptococcus (GAS)



- Capsule
 - Non-antigenic
- Cell wall
 - M-protein
 - Induces antibodies
 - Serotypes 5, 6, & 19 cross react with myosin

Streptococcal Tonsillopharyngitis



Pathophysiology



- Rheumatic fever develops in some children and adolescents following PHARYNGITIS with a group A beta-hemolytic Streptococcus (*Streptococcus pyogenes*)
- The organisms attach to the epithelial cells of the upper respiratory tract and produce a battery of enzymes allowing them to damage and invade human tissues

Group A β -Hemolytic Streptococcus (GAS)



- Enzymes
 - Streptolysin
 - Deoxyribonuclease
 - Fibrinolysin
 - Diphosphopyridine nucleotidase
 - Hyaluronidase

Rheumatic Fever: Pathogenesis



- Actual mechanism unknown
- Postulate:
 - Autoimmune or hypersensitivity reaction to GAS produces pathogenic autoantibodies to cardiac tissues

Pathophysiology



- In 0.3-3% of cases, streptococcal pharyngitis leads to rheumatic fever several weeks after the sore throat has resolved.
- Only infections of the pharynx initiate or reactivate rheumatic fever.
- Organism spreads by direct contact with oral or respiratory secretions
- Patient remains infected for weeks after symptomatic resolution of pharyngitis



Fever in ARF

- Full manifestations of the disease may be suppressed if patient has previously taken aspirin or other NSAIDs
- Fever above 39° C with no characteristic pattern are initially present in almost every case of acute rheumatic fever
- Fever may be low grade in children with mild carditis or absent in patients with chorea
- Low grade fevers persists for 2-3 weeks



DIAGNOSIS

- A diagnosis of RHD is made after confirming antecedent rheumatic fever
- The JONES criteria require the presence of 2 major or 1 major and 2 minor criteria for the diagnosis of RF
- Additional evidence of previous group A streptococcal pharyngitis is required



Jones Criteria 1992

- Establish initial attack of acute rheumatic fever
- Not intended
 - To establish diagnosis of inactive or chronic RHD
 - To measure rheumatic activity
 - To predict course or severity of disease
- Previous RF or RHD not included as manifestation

Jones Criteria 1992 for Acute Rheumatic Fever



Major manifestations

- Carditis
- Polyarthritits
- Chorea
- Erythema marginatum
- Subcutaneous nodules

Minor manifestations

- Fever
- Arthralgia
- ↑ acute phase reactants
- Prolonged PR interval

Cardiac Manifestations of ARF



- Carditis is most commonly detected by a new murmur and tachycardia out of proportion to fever
- New or changing murmurs are considered necessary for a diagnosis of rheumatic valvulitis

Acute RF w Carditis



- Acute rheumatic heart disease often produces a pancarditis (endocarditis, myocarditis and pericarditis)
- Mitral valve is most commonly and severely affected, followed by the aortic valve

Rheumatic Carditis



- Valvulitis
 - Almost always present
 - Apical systolic murmur (Mitral regurgitation)
 - Apical mid-diastolic murmur (Carey-Coomb's)
 - Basal diastolic murmur (Aortic regurgitation)
 - Basal systolic murmur (Tricuspid regurgitation)
- Resting tachycardia
- Muffled heart sounds
- Gallop rhythm
- Pericardial friction rub
- Congestive heart failure

“Echocarditis”



- Some cardiologists have proposed that echo-Doppler evidence of mitral insufficiency, particularly in association with aortic insufficiency, may be sufficient for a diagnosis of carditis (**even in the absence of accompanying auscultatory findings**)

“Echocarditis”



- Echocardiographic findings in acute rheumatic fever (mitral regurgitation and/or aortic regurgitation) in the absence of clinical carditis
- Subclinical carditis
- Included as basis for carditis in modified Jones criteria?
- Controversial

Jones Criteria 1992



High probability of rheumatic fever :

- 2 major or
- 1 major & 2 minor manifestations

PLUS Evidence of GAS
infection



Evidence of Antecedent Group A Streptococcal Infections

- ↑ or rising Streptococcal antibody (ASO) titer
- (+) throat culture or rapid antigen test

Antistreptococcal antibodies



- Clinical features of RF begin at the time antistreptococcal antibodies are at their peak
- Useful for confirming previous group A streptococcal infection
- ASO is the most commonly used
- Sensitivity can be improved by testing for several antibodies (ASO, antiDNase, antihyaluronidase etc.)



Antistreptococcal Antibody Titers

- Reflect past & not present immunologic events
- No value in the diagnosis of acute pharyngitis
- Valuable for confirmation of previous streptococcal infections in patients suspected of having acute RF or PSGN
- Helpful, in prospective epidemiological studies, for distinguishing patients with acute infection from patients who are carriers



ASO titer

- Peaks 2-3 weeks after the onset of rheumatic fever
- Plateaus for 3-6 months
- Returning to normal levels after 6-12 months
- **Antihyaluronidase** results are frequently abnormal in RF patients with normal level ASO titer and may rise earlier and persist longer than ASO during RF

Rheumatic Fever

Laboratory Examinations



- CBC
 - Anemia ; Leukocytosis
- ECG
 - Sinus tachycardia
 - 1° AV block
 - No chamber enlargement
 - Rarely 2° AV block, low voltages, ST-T wave changes
- CXR
 - Normal
 - Cardiomegaly
 - Pulmonary congestion to edema

Acute Phase Reactants



- C reactive protein and ESR
 - Elevated in RF due to inflammatory in nature
 - Both have a high sensitivity but low specificity
 - May be used to monitor the resolution of inflammation, detect relapse when weaning aspirin or identify recurrence of disease

Chest X-ray



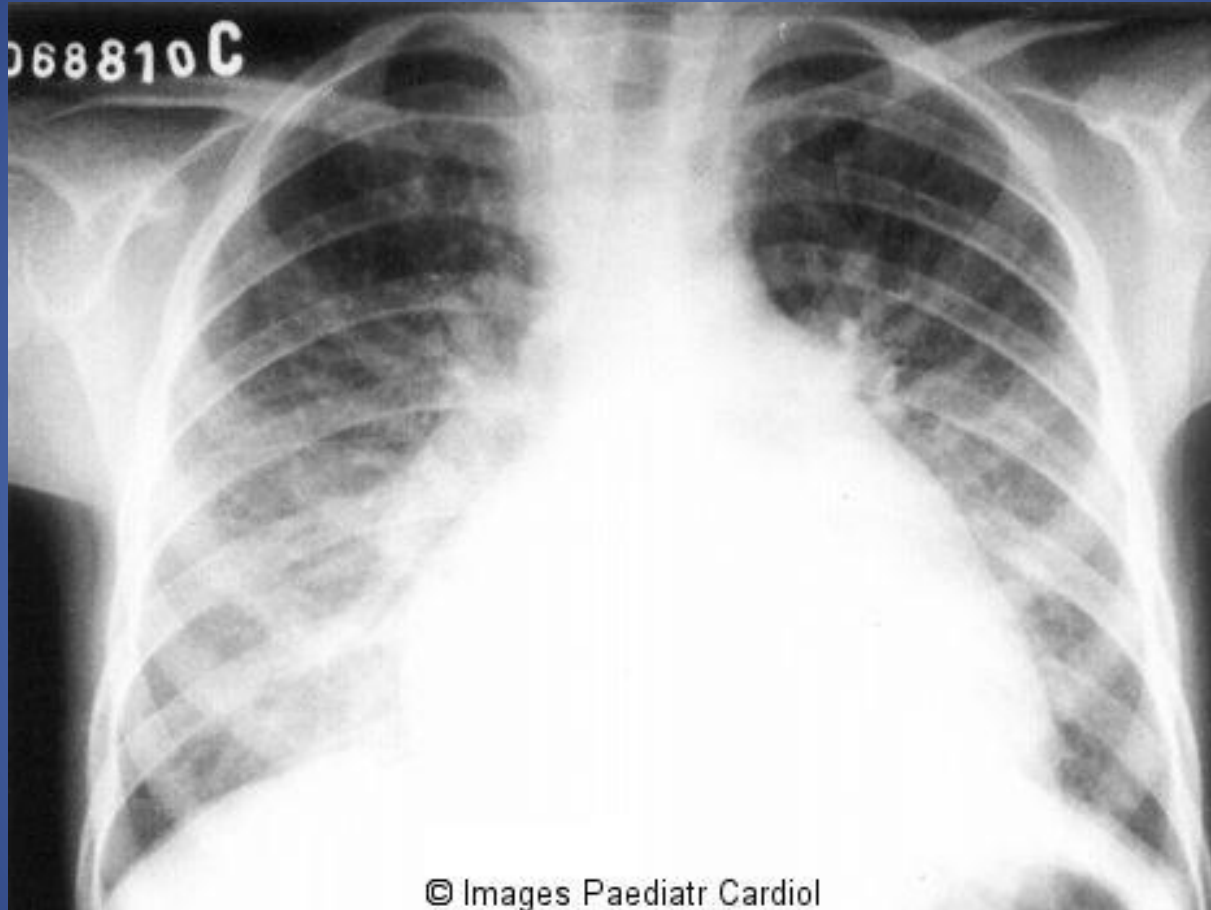
- Chest X-ray
 - Cardiomegaly may or may not be present
 - Pulmonary congestion in congestive heart failure

Rheumatic Carditis



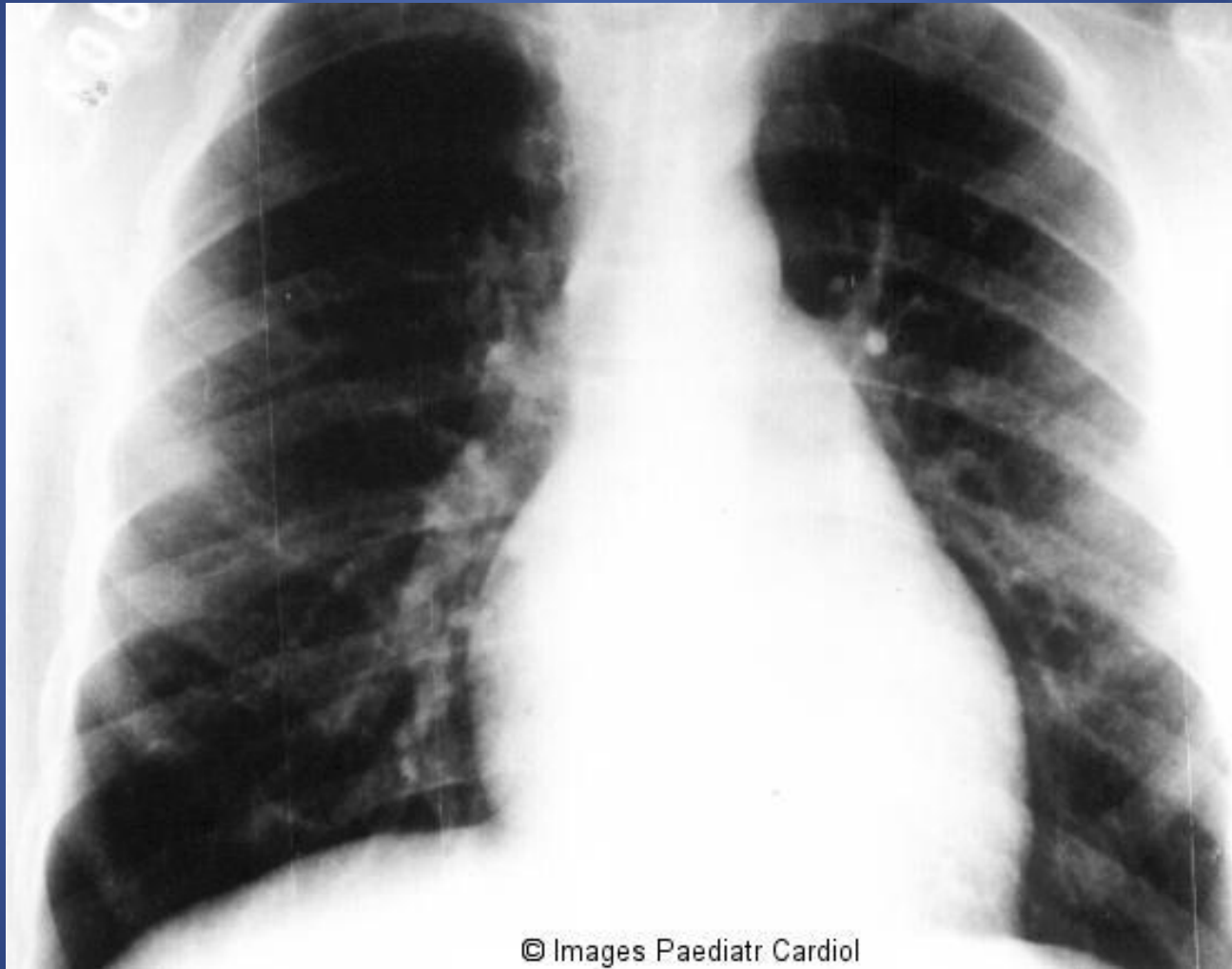
- Mild carditis – No cardiomegaly
- Moderate carditis – mild cardiomegaly
- Severe carditis – cardiomegaly with severe pulmonary congestion or edema

Rheumatic Carditis



Chest radiograph of an 8 year old patient with acute carditis before treatment

Rheumatic Carditis



© Images Paediatr Cardiol

Same patient after 4 weeks

ECHOCARDIOGRAPHY



- Is the imaging of choice to detect the presence of rheumatic carditis or rheumatic heart disease
- If the ECHO is normal, diagnosis of RHD is ruled out

Rheumatic Fever : Echocardiographic Findings



- Focal nodular thickening of valves
- Prolapsed Anterior mitral valve leaflet
- Left chambers enlargement ; normal function
- Pericardial effusion
- Valvular regurgitation – valves involved & severity

RHD - Mitral Regurgitation



© Images Paediatr Cardiol

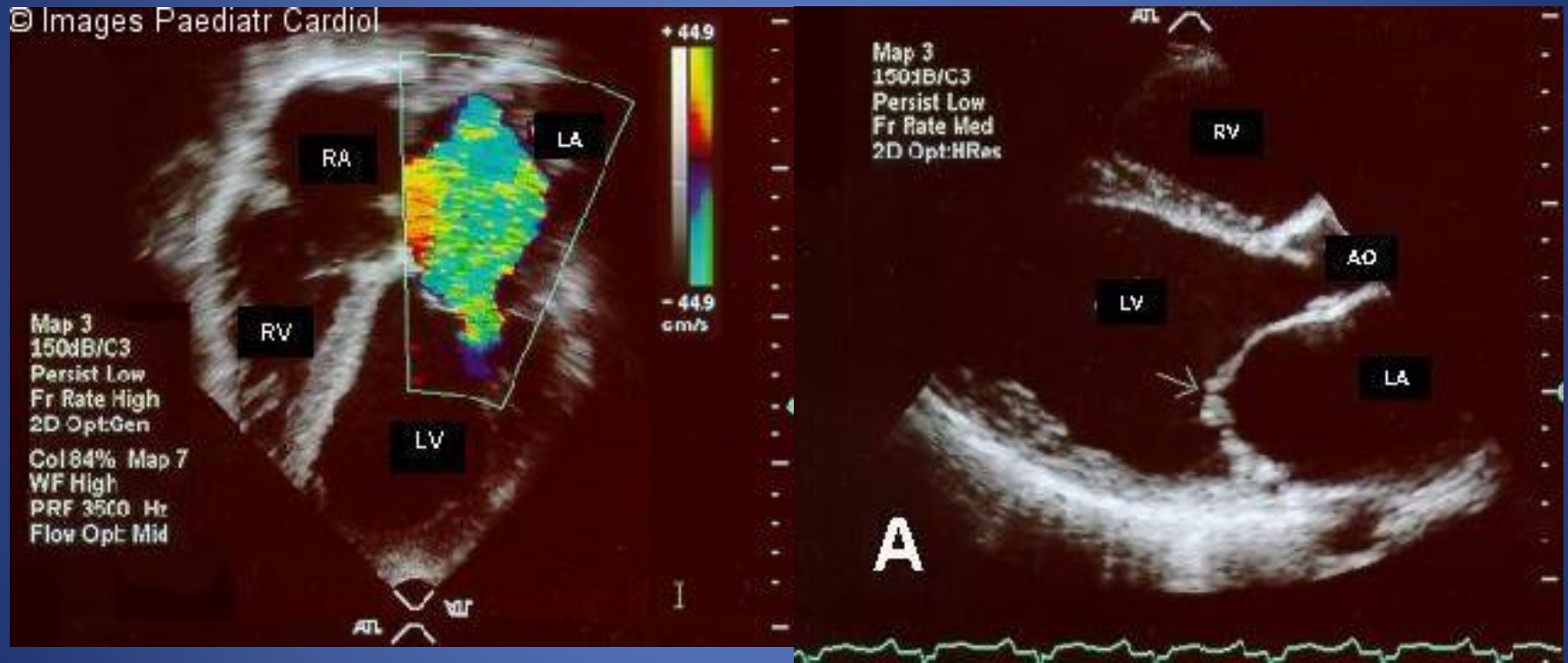
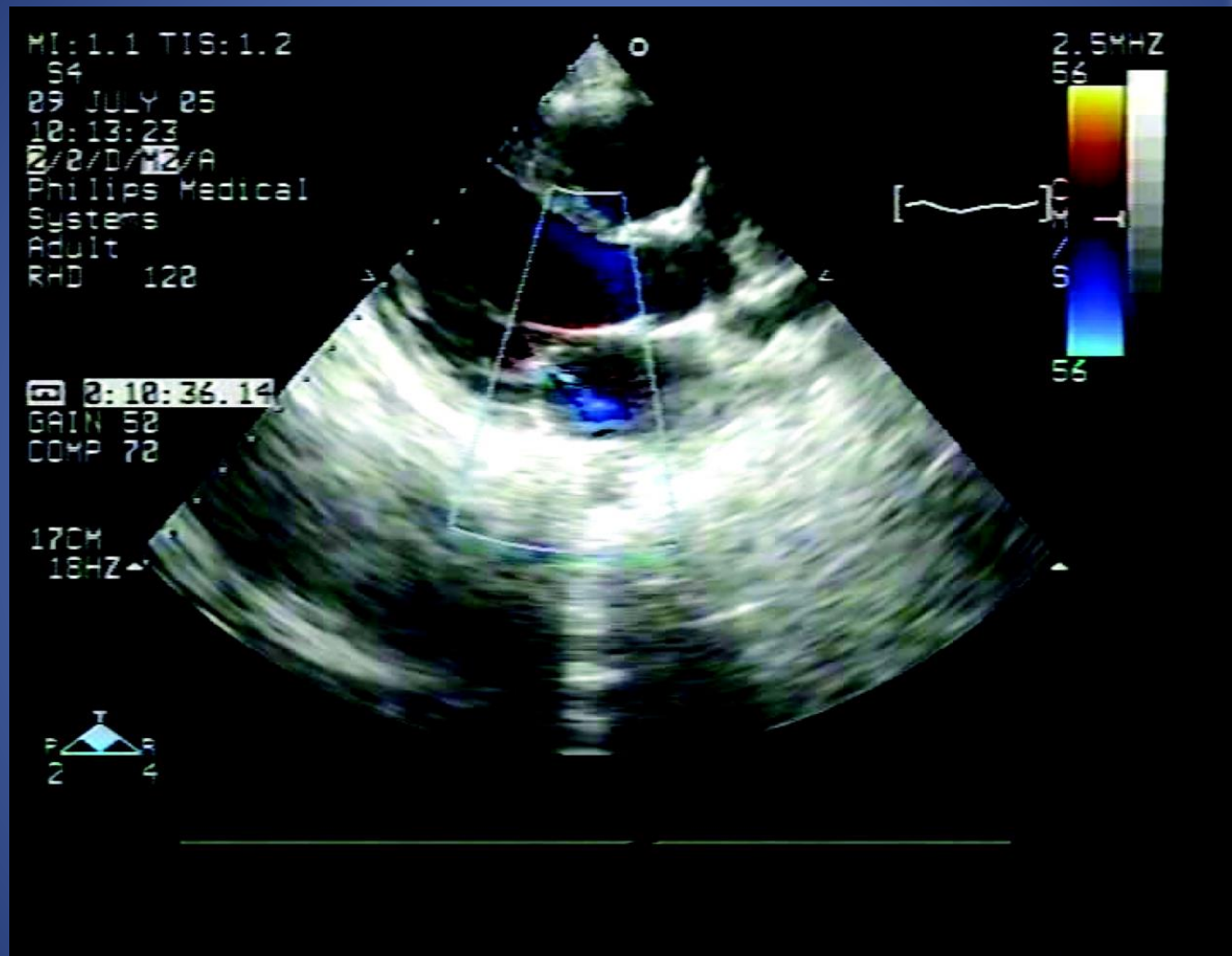




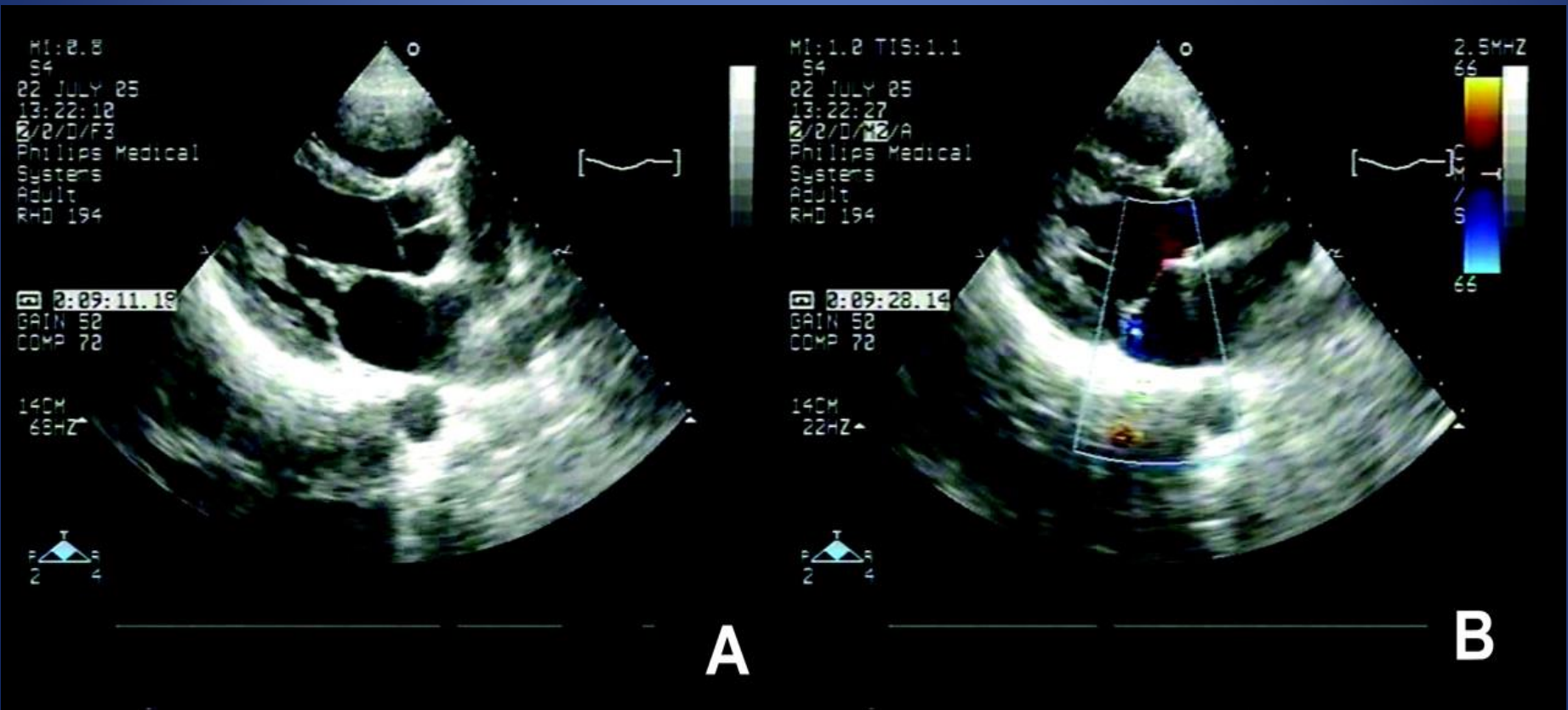
Figure 2. Significant regurgitation with the use of WHO criteria for subclinical RHD (left parasternal long-axis view).



Marijon E et al. *Circulation* 2009;120:663-668



Figure 3. Echocardiographic findings of subclinical RHD with the use of combined criteria (left parasternal long-axis view).



Marijon E et al. *Circulation* 2009;120:663-668

Echocardiogram



- Color doppler-echocardiography identifies and quantitates valve insufficiency and ventricular function
- Mild carditis -- mild regurgitation during acute phase but resolves in weeks to months
- Moderate-severe carditis – persistent mitral/aortic regurgitation

Rheumatic Mitral Regurgitation



- The most important echocardiographic features of mitral regurgitation from acute rheumatic valvulitis are annular dilatation, elongation of the chordae to the anterior leaflet, and a posterolaterally directed mitral regurgitation

Echocardiography in RHD



- Acute Rheumatic Fever – Left ventricle is frequently dilated in association with normal or increases fractional shortening
- Chronic rheumatic heart disease – Affected leaflets become diffusely thickened with fusion of commissures and chordae tendinae; calcifications may be seen

Rheumatic Heart Disease



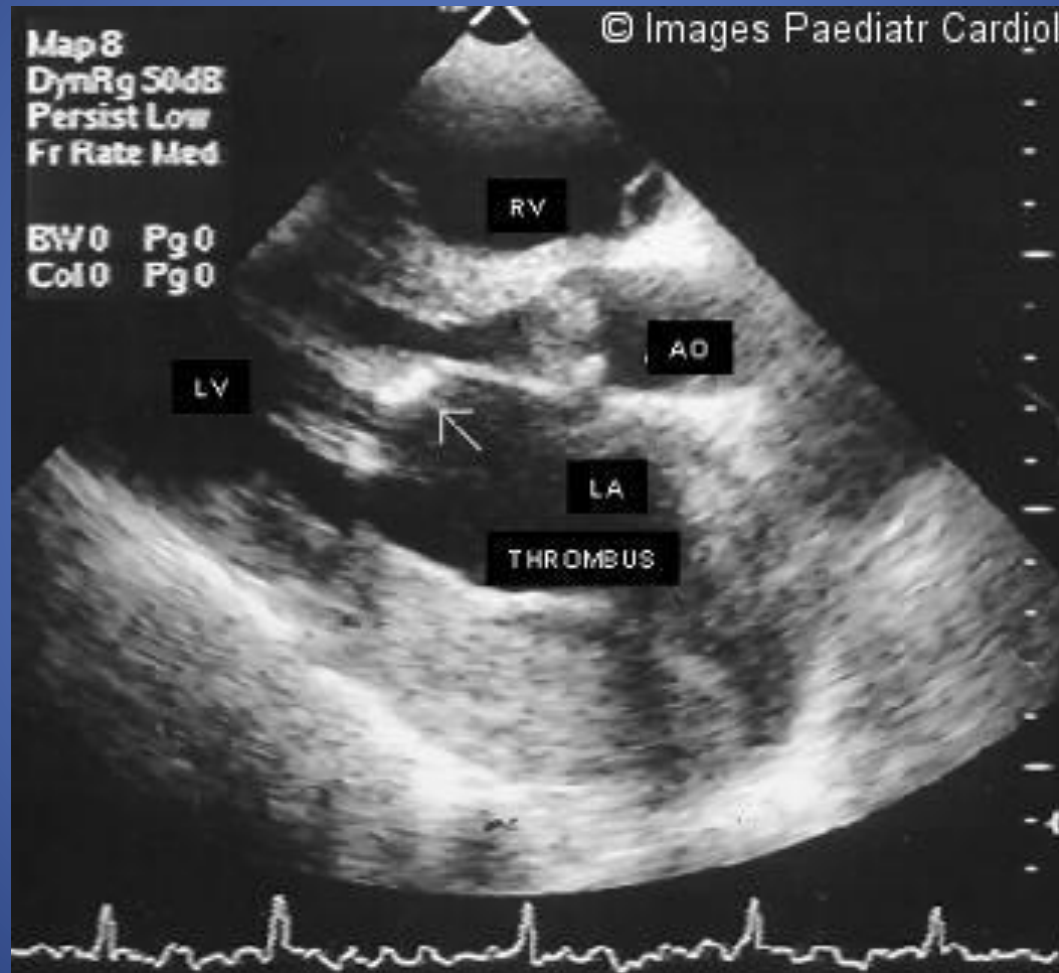
Chronic valvar heart disease as a
sequelae of rheumatic fever
and its recurrences

RHD Mitral Stenosis



Stenotic mitral valve seen from left atrium. Both commissures are fused; the cusps are severely thickened. The left atrium is huge. The valve is both incompetent & stenotic

RHD Mitral Stenosis



Rheumatic Carditis



© Images Paediatr Cardiol

Aortic valve showing active valvulitis. The valve is slightly thickened and displays small vegetations – "verrucae"



Rheumatic Fever : Management

- Antibiotic: to eradicate Streptococcus
 - PCN VK - 250 – 500 mg TID x 10d
 - Benzathine PCN - 0.6 – 1.2 MU IM
 - Erythromycin - 250mg TID x 10d
- Anti-inflammatory agents: 6 – 8 weeks
 - ASA - 100 mg/kg/day
 - Prednisone - 2 mg/kg/day
- Complete bed rest & modified activity



GAS Tonsillitis: Rheumatic Attack Rate

- 0.3% - GAS tonsillitis treated with PCN
- 3% - GAS tonsillitis untreated
- 60% - known rheumatic patients with GAS tonsillitis

Rheumatic Fever : Management



- Prophylaxis
 - 1^o - prevents 1st episode of RF
 - 2^o - prevents recurrences of RF
 - PCN VK 250 mg BID
 - Benzathine PCN 1.2 MU q 21 – 28 days
 - Duration of 2^o prophylaxis
 - Arthritis - at least 5 years
 - Carditis - at least 10 years (recurrence free & no residual heart disease)

Treatment of GAS Pharyngitis & 1° Prevention of RF



Table 8. Primary Prevention of Rheumatic Fever (40)

Agent	Dose	Mode	Duration
Benzathine	600,000 U for patients ≤ 27 kg (60 lb)	Intramuscular	Once
Penicillin G	1,200,000 U for patients > 27 kg (60 lb)		
	or		
Penicillin V (phenoxymethyl penicillin)	Children: 250 mg 2-3 times daily Adolescents and adults: 500 mg 2-3 times daily	Oral	10 d
For individuals allergic to penicillin:			
Erythromycin	20-40 mg/kg/d	Oral	10 d
Estolate	2-4 times daily (maximum 1 g/d)		
Ethylsuccinate	40 mg/kg/d	Oral	10 d
	2-4 times daily (maximum 1 g/d)		
Azithromycin	500 mg on first day 250 mg/d for the next 4 d	Oral	5 d

From Dajani et al (40) with permission.

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Azithromycin	500 mg on first day 250 mg/d for the next 4 d	Oral	5 d

From Dajani et al (40) with permission.

Table 9. Secondary Prevention of Rheumatic Fever (40)

Agent	Dose	Mode
Benzathine Penicillin G	1,200,000 U every 4 wk (every 3 wk for high-risk* pts such as those with residual carditis)	Intramuscular
Penicillin V	250 mg twice daily	Oral
	or	
Sulfadiazine	0.5 g once daily for pts ≤ 27 kg (60 lb) 1.0 g once daily for pts > 27 kg (60 lb)	Oral
For individuals allergic to penicillin and sulfadiazine:		
Erythromycin	250 mg twice daily	Oral

Abbreviations: Pts = patients. *High-risk patients include patients with residual rheumatic carditis as well as patients from economically disadvantaged populations. From Dajani et al (40) with permission.

Table 10. Duration of Secondary Rheumatic Fever Prophylaxis (40)

Category	Duration
Rheumatic fever with carditis and residual heart disease (persistent valvular disease)	≥10 y since last episode and at least until age 40 y, sometimes lifelong prophylaxis*
Rheumatic fever with carditis but no residual heart disease (no valvular disease)	10 y or well into adulthood, whichever is longer
Rheumatic fever without carditis	5 y or until age 21 y, whichever is longer

*The committee's interpretation of "lifelong" prophylaxis refers to patients who are at high risk and likely to come in contact with populations with a high prevalence of streptococcal infection, ie, teachers, day-care workers. From Dajani et al (40) with permission.

Penicillin for 2nd Prophylaxis of Rheumatic Fever



- Meta-analysis by Manyemba & Mayosi [Cochrane Library (1) 2003]
- 9 RCT & quasi-RCT with 3008 subjects ; methodological quality poor
- IM Penicillin more effective than oral penicillin
- **2 weekly or 3 weekly injections more effective than 4 weekly injections**



INFECTIVE ENDOCARDITIS

Infective Endocarditis



- Infection of the endocardium and/or heart valves that involves thrombus formation (vegetation) , which may damage the endocardial tissue and/or valves
- Many aspects of IE are similar in children and adults, but there are some manifestations that are unique to children

Epidemiology



- IE occurs less often in children than in adults and accounts for ≈ 1 in 1280 pediatric admissions per year
- Frequency of endocarditis among children seems to have increased in recent years.
- This is due in part to improved survival among children who are at risk for endocarditis, such as those with CHD and hospitalized newborn infants.

Epidemiology



- Most children with IE have an identifiable risk factor for disease
- Children with CHD have the highest risk of developing IE.
- The risk is increased in patients with complex cyanotic heart disease, especially those who undergo surgical procedures that introduce prosthetic material (conduits and shunts)



Risk Factors

- Congenital Heart Defects
- Rheumatic Heart Disease
- Intravascular shunts/conduits/prosthesis
- Intravascular catheters
- IV drug abuse
- Prematurity
- Immunocompromise



Pathogenesis

- IE is due to a series of complex interactions among blood borne pathogens, damaged cardiac endothelium, fibrin and platelets that result in the formation of an infected thrombus (vegetation) and damage to endocardium and/or heart valves.

Pathophysiology



1. **Turbulent blood flow** disrupts the endocardium making it “sticky”
2. **Bacteremia** delivers the organisms to the endocardial surface
3. **Adherence** of the organisms to the endocardial surface
4. **Eventual invasion** of the valvular leaflets

Infecting Organisms



- Common bacteria
 - S. aureus
 - Viridans group streptococci
 - Enterococci
 - Not so common bacteria
 - Fungi*
 - Coagulase negative staph*
 - Pseudomonas
 - HACEK
- *Common in neonates

Clinical Presentation



- Variable
- Dependent upon the extent of the local cardiac disease
- Degree of involvement of other organs (eg embolization)
- Causative agent

Clinical Presentation



- **Acute**

- Affects normal heart valves
- Rapidly destructive
- Metastatic foci
- Commonly Staph.
- If not treated, usually fatal within 6 weeks

- **Subacute**

- Often affects damaged heart valves
- Indolent nature
- If not treated, usually fatal by one year
- Usually Viridans strep

Clinical Presentation



- **Acute**

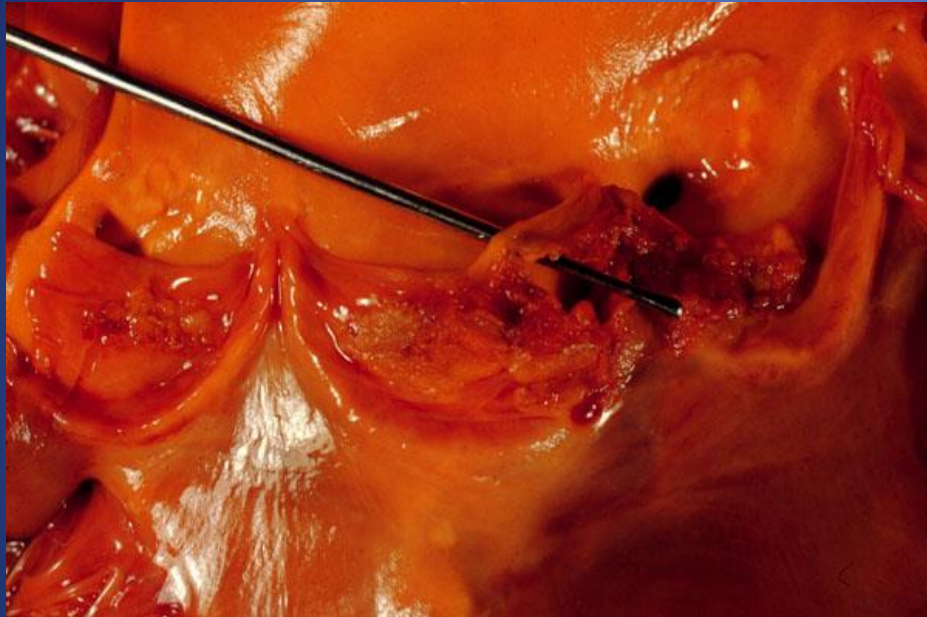
- High grade fever and chills
- Severely ill
- Hemodynamically unstable

- **Subacute**

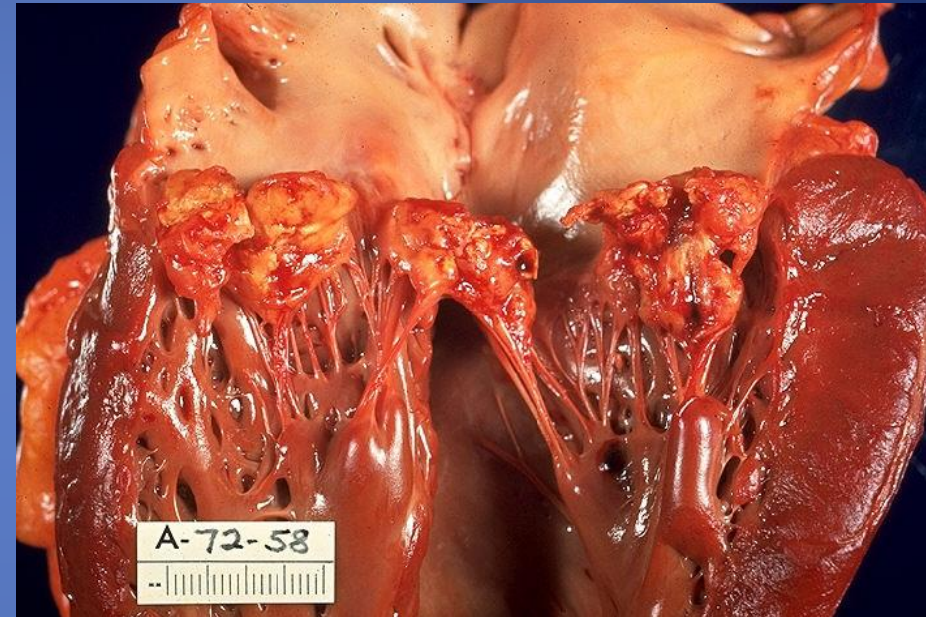
- Low grade fever
- Anorexia
- Weight loss
- Exercise intolerance
- Diaphoresis

The onset of symptoms is usually ~2 weeks or less from the initiating bacteremia

Local Spread of Infection



Acute *S. aureus* IE with perforation of the aortic valve and aortic valve vegetations.



Acute *S. aureus* IE with mitral valve ring abscess extending into myocardium.

Signs



- Fever
- Heart murmur (“new” or “changing”)
- Nonspecific signs – petechiae, subungal or “splinter” hemorrhages, clubbing, splenomegaly, neurologic changes
- More specific signs - Osler’s Nodes, Janeway lesions, and Roth Spots (not common in children)

Petechiae

1. Nonspecific
2. Often located on extremities or mucous membranes



dermatology.about.com/.../blpetechiaephoto.htm



Harden Library for the Health Sciences
www.lib.uiowa.edu/hardin/md/cdc/3184.html

Photo credit, Josh Fierer, M.D.
medicine.ucsd.edu/clinicalimg/Eye-Petechiae.html

Splinter Hemorrhages



1. Nonspecific
2. Nonblanching
3. Linear reddish-brown lesions found under the nail bed
4. Usually do NOT extend the entire length of the nail

Janeway Lesions



1. More specific
2. Erythematous, blanching macules
3. Nonpainful
4. Located on palms and soles

Osler's Nodes



American College of Rheumatology

web.rheum.bham.ac.uk/.../default/pages/3b5.htm



www.meddean.luc.edu/.../

Hand10/Hand10dx.html



1. More specific
2. Painful and erythematous nodules
3. Located on pulp of fingers and toes
4. More common in subacute IE

Making the Diagnosis



- **Pelletier and Petersdorf criteria (1977)**
 - Classification scheme of definite, probable, and possible IE
 - Reasonably specific but lacked sensitivity
- **Von Reyn criteria (1981)**
 - Added “rejected” as a category
 - Added more clinical criteria
 - Improved specificity and clinical utility
- **Duke criteria (1994)**
 - Included the role of echocardiography in diagnosis
 - Added IVDA as a “predisposing heart condition”

Modified Duke's Criteria



The clinical diagnosis of IE is made by fulfilling the modified Duke's criteria which is based upon physical and echocardiogram

Duke's Modified Criteria



MAJOR

- Positive blood culture
- Positive Echocardiogram
 - Oscillating intracardiac mass
 - Absces
 - Dehiscence of prosthetic valve
- New Valvular Regurgitation

MINOR

- Predisposing Heart Condition
- Fever
- Vascular phenomena
 - Major arterial emboli
 - Septic pulmonary infarcts
 - Janeway lesions
 - Conjunctival hemorrhage
- Immunologic phenomena
 - Glomerulonephritis
 - Osler's nodes
 - Roth spots

Modified Duke's Criteria



Definite IE

- 2 Major or
- 1 Major 3 Minor or
- 5 Minor criteria

Possible IE

- 1 Major and 1 Minor or
- 3 Minor

Rejected IE

Resolution of illness with four days or less of antibiotics

LABORATORY TESTS



- **Blood Cultures**

- Minimum of three blood cultures
- Three separate venipuncture sites
- Obtain 1-3ml in infants and 5-7mL in children
- Bacteremia is continuous so sampling can be done anytime without relation to the fever

- **Positive Result**

- **Typical organisms present in at least 2 separate samples**
- Persistently positive blood culture (atypical organisms)
 - Two positive blood cultures obtained at least 12 hours apart
 - Three or a more positive blood cultures in which the first and last samples were collected at least one hour apart

Culture negative endocarditis



- When a patient has clinical and/or echocardiographic evidence of IE but persistently negative blood cultures.
- Due to current or recent antibiotic therapy or infection caused by a fastidious organism that grows poorly in vitro.
- At times, the diagnosis can be made only by removal of vegetations during surgery or at necropsy or by growth of organisms from an excised thrombus or embolus.
- In most centers in the US, the prevalence of culture-negative endocarditis may be $\approx 5\%$ to 7% , imposing a need to be thorough and precise in accepting a diagnosis of culture-negative endocarditis.^{5,7,28}

Additional Labs



- CBC: Low hemoglobin/hematocrit (ANEMIA)
- Elevated ESR and CRP
- Urinalysis: Hematuria, proteinuria, and red cell casts suggestive of glomerulonephritis

Imaging



- Chest x-ray
 - Look for multiple focal infiltrates and calcification of heart valves
- EKG
 - Rarely diagnostic
 - Look for evidence of ischemia, conduction delay, and arrhythmias
- Echocardiography

ECHOCARDIOGRAPHY



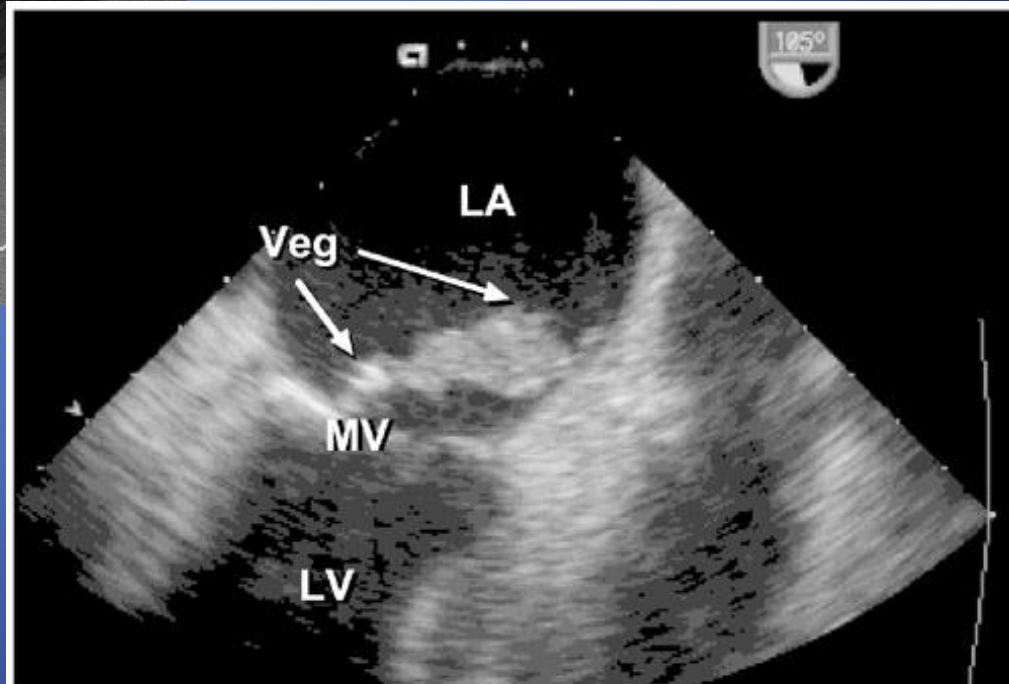
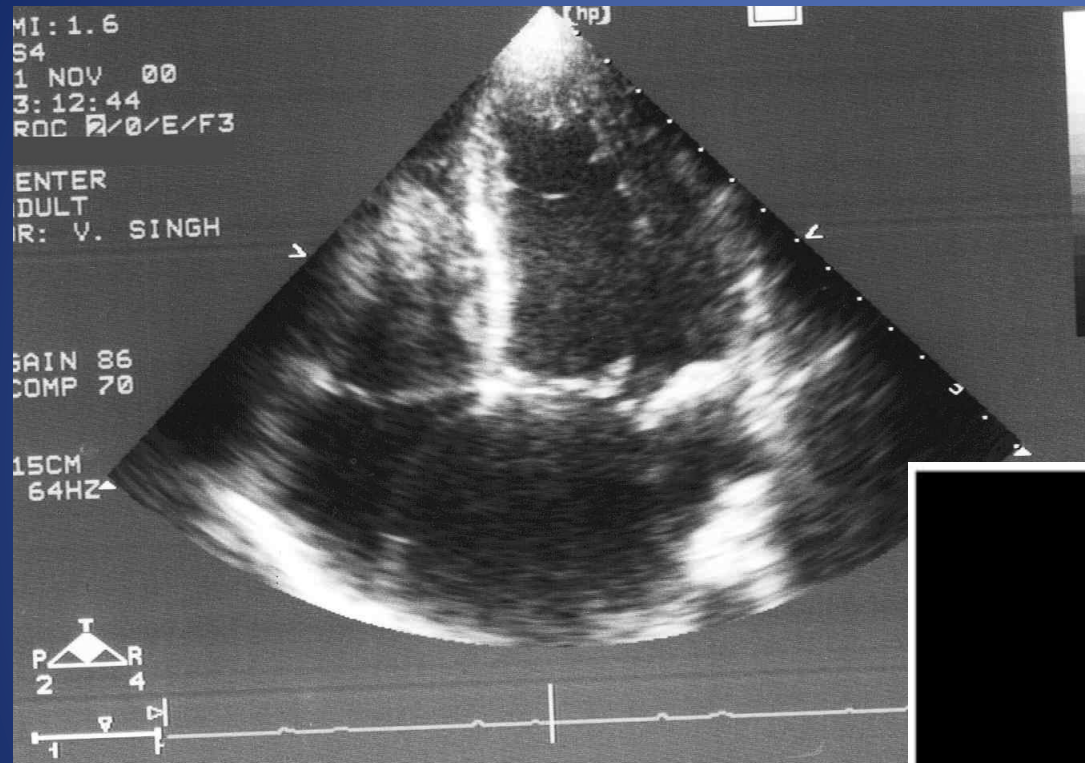
- Should be performed on all patients in whom there is a reasonable suspicion of IE to detect presence of a vegetation
- Main modality to detect endocardial infection
- Can identify the size and location of vegetation , extent of valve damage ,ventricular function and pericardial effusion etc.

Indications for Echocardiography

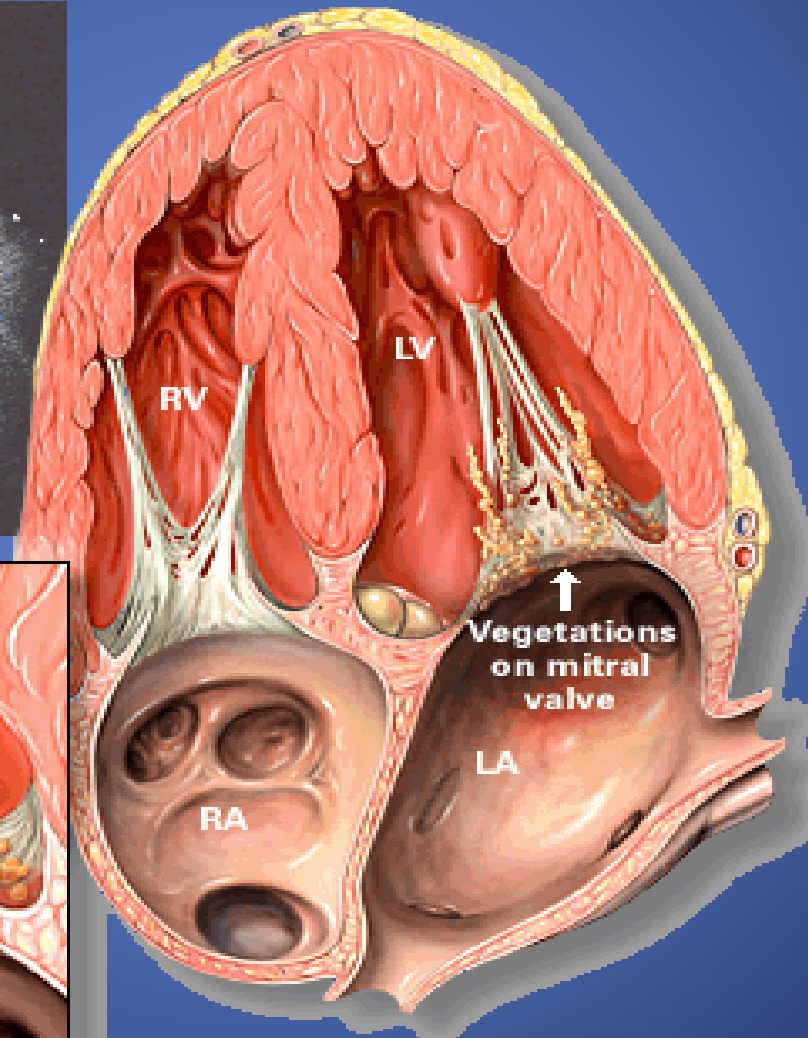
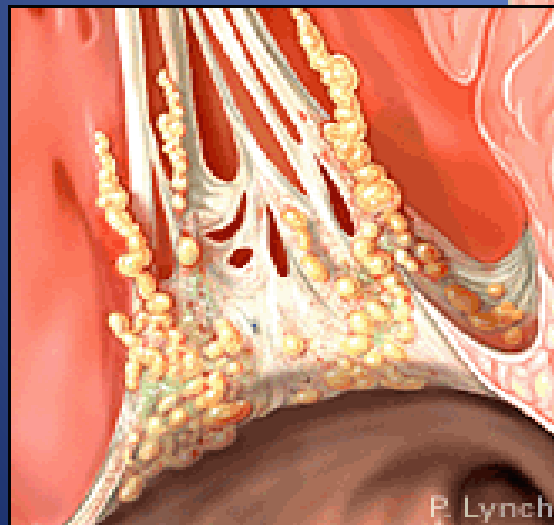
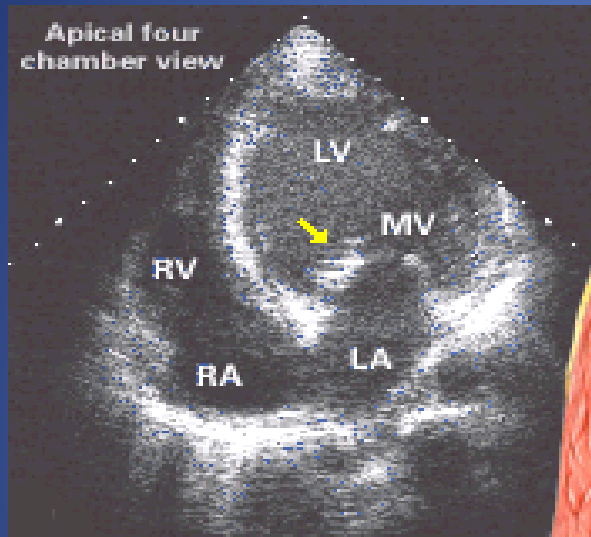


- Transthoracic echocardiography (TTE)
 - First line if suspected IE
 - Native valves
- Transesophageal echocardiography (TEE)
 - Prosthetic valves
 - Intracardiac complications
 - Inadequate TTE
 - Fungal or *S. aureus* or bacteremia

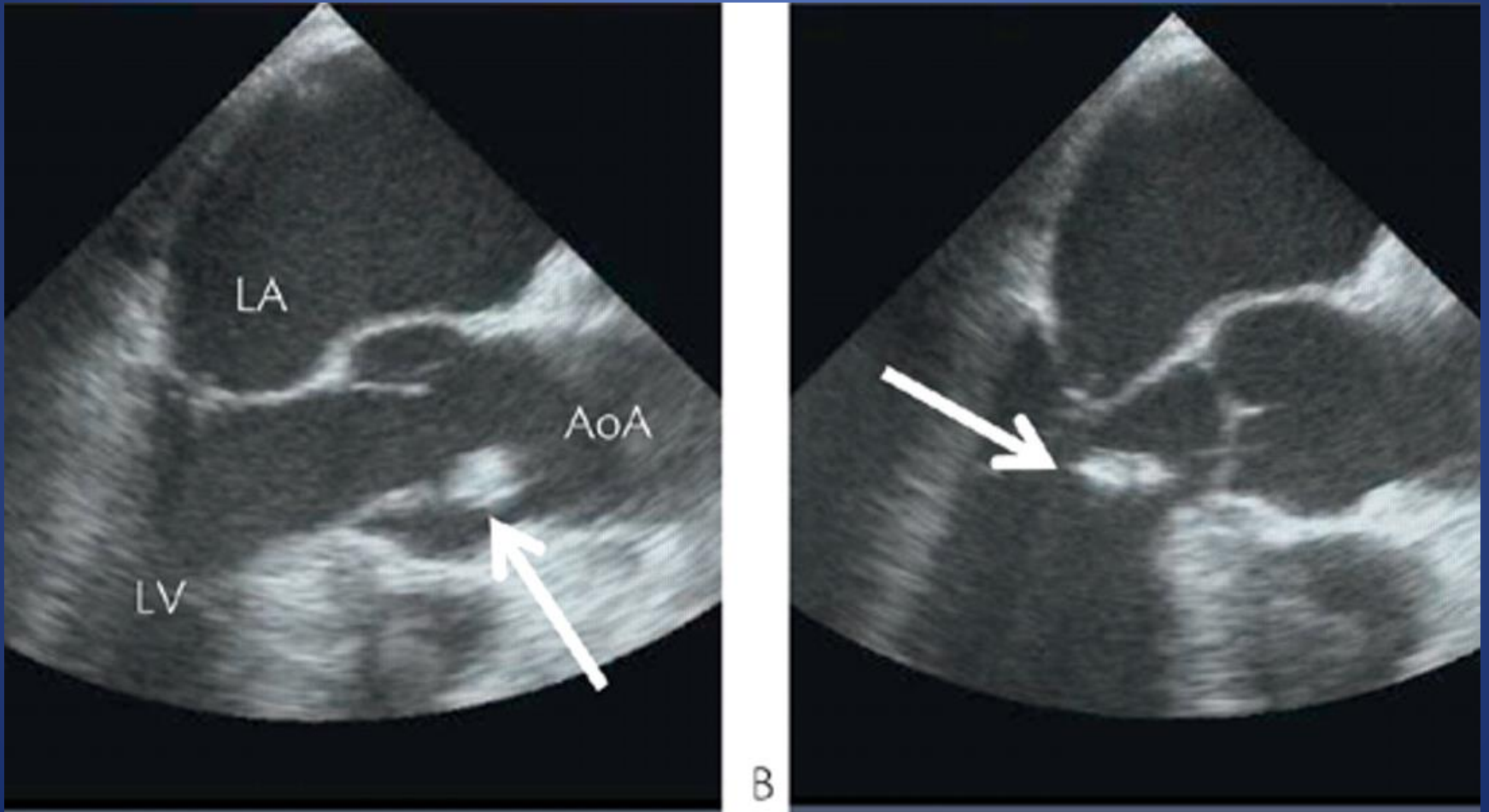
Vegetation



Vegetations



Vegetation (TEE)



Complications



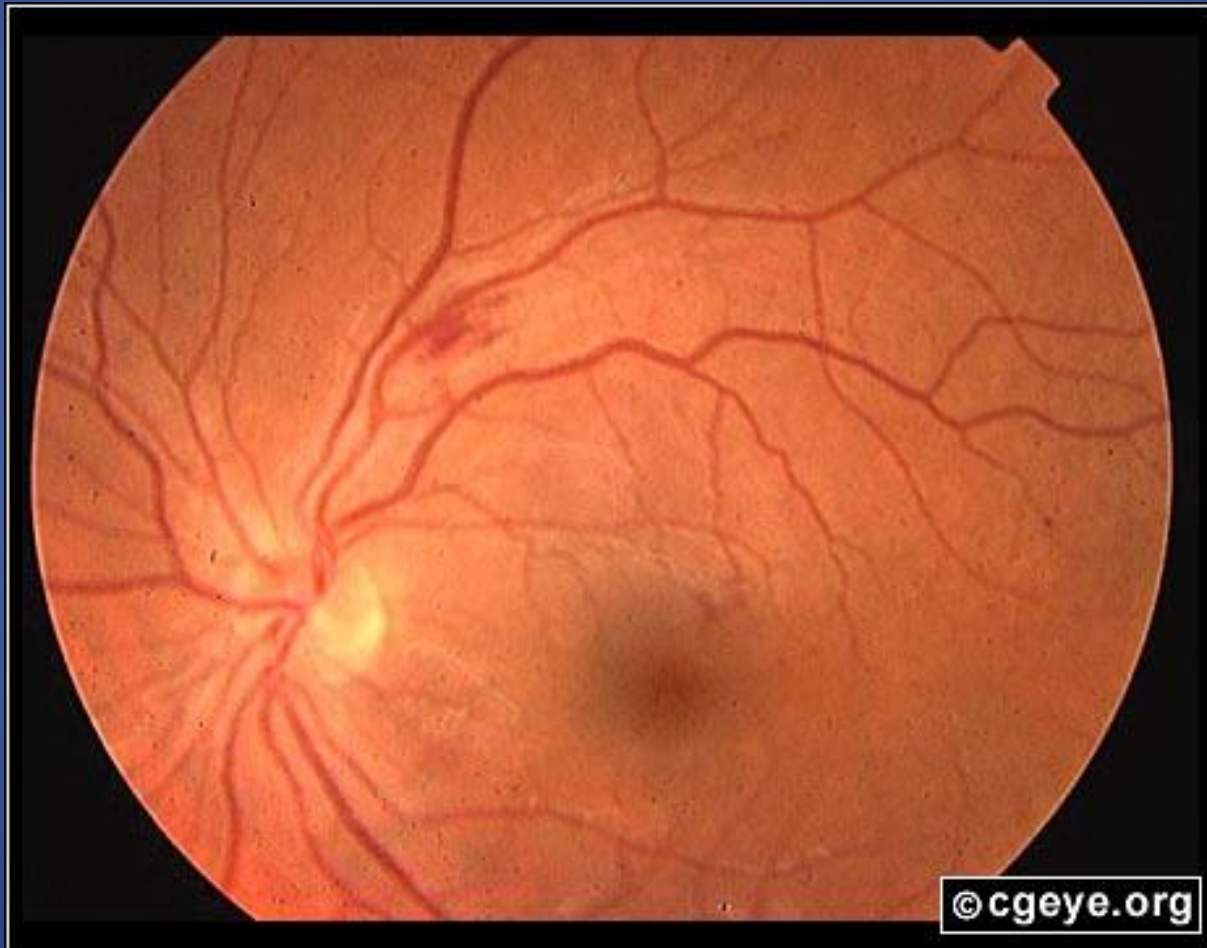
- Heart Failure
- Metastatic Infections due to Septic emboli (Osteomyelitis, Pneumonia, Abscesses)
- Embolic phenomenon (Stroke, MI)
- Glomerulonephritis

Septic Pulmonary Emboli



<http://www.emedicine.com/emerg/topic164.htm>

Septic Retinal Embolus



Risk Factors for IE Complications



- Prosthetic cardiac valves
- Left sided involvement
- Staphylococcus aureus or fungal IE
- Previous IE
- Prolonged symptoms > 3months
- Cyanotic congenital heart disease
- Systemic to pulmonary shunts
- Poor clinical response to antimicrobial therapy



Treatment

- Antibiotic choice, dosage, and duration of treatment are dependent upon the underlying causative microbial agent
- In acute IE, blood culture should be done as quickly as possible so empiric antibiotic therapy can be started
- Patients who fail medical therapy with persistent vegetations despite antibiotic therapy may be candidates for surgical intervention



Treatment

- Parenteral antibiotics
 - High serum concentrations to penetrate vegetations
 - Prolonged treatment to kill dormant bacteria clustered in vegetations
 - Usually 4-8 weeks
 - Broad coverage until organism is isolated
- Surgery
 - Intracardiac complications
- Surveillance blood cultures
 - During and after treatment



Prophylaxis

- Revised guidelines from AHA state that antibiotic prophylaxis should be reserved for patients with highest risk of IE
- High Risk Patients:
 - Prosthetic cardiac valves
 - Previous IE
 - Cardiac valvar disease after transplantation
 - Unrepaired cyanotic CHD
 - Palliative shunts and conduits
 - Within 6 months of complete repair with a prosthetic material or device
 - Residual defect at the site or adjacent to the site of prosthetic patch



Prophylaxis

- All dental procedures that involve manipulation of gingival tissues
- Procedures on respiratory tract or infected skin, dermal structures or musculoskeletal tissue
- Any other procedure that is potentially bacteremic
- Focus should shift towards good oral hygiene

Prophylaxis



TABLE 5.1 Antibiotic prophylaxis for high-, moderate-, and low-risk cases

High risk

Previous infective endocarditis [34]
Complex cyanotic congenital heart disease, transposition of great arteries, Fallot's tetralogy, Gerbode's defect [35–37]
Surgically constructed systemic pulmonary shunts or conduits
Mitral valve prolapse with mitral regurgitation or thickened valve leaflets [38–42]
Prosthetic heart valves (5 times greater than those with native valves) [43,44]

Moderate risk

Acquired valvular heart disease, e.g. rheumatic heart disease—aortic stenosis, aortic regurgitation, mitral regurgitation
Non-cyanotic congenital cardiac defects, e.g. bicuspid aortic valve, primum atrial septal defect, patent ductus arteriosus [45], coarctation of aorta [47], atrial septal aneurysm/patent foramen ovale [48], ventricular septal defect [49]
Other structural cardiac defects, e.g. aortic root replacement [46], hypertrophic obstructive cardiomyopathy [50–53], subaortic membrane [54]

Low-risk cases not requiring antibiotic prophylaxis

Isolated secundum atrial septal defect^a [55], pulmonary stenosis
Surgically-repaired atrial septal defect, ventricular septal defect or patent ductus arteriosus, post Fontan or Mustard procedure without residual defect/murmur
Previous coronary artery bypass surgery
Mitral valve prolapse without regurgitation
Innocent heart murmurs^b
Cardiac pacemakers/defibrillators^{c,d}
Coronary artery stent implantation^c
Heart/Heart and lung transplant^e

^a Antibiotic prophylaxis is recommended for up to 12 months after ASD/PFO catheter-based closure procedures.

^b If unsure as to the exact nature of the murmur and the need for prophylaxis, an opinion should be sought from a cardiologist. In an emergency or when it is difficult to obtain specific advice then antibiotic prophylaxis should be given prior to dental or surgical treatment.

^c Unless being performed in patients at moderate or high risk of endocarditis, when antibiotic prophylaxis is advisable.

^d Pre- and post-procedure antibiotics are generally used routinely for surgical prophylaxis.

^e Within the first 6 months after heart/heart-lung transplantation, patients should receive antibiotic prophylaxis.



Summary

- RHD and IE are 2 very important acquired cardiac infections in pediatrics
- Diagnostic criteria have been established to guide us in the treatment of our patients
- Echocardiography play a major role in the initial diagnosis of these conditions
- Prolonged treatment and appropriate prophylaxis are needed to ensure cure and prevent further recurrences