



# CVS MODULE – 4

## RHEUMATIC HEART DISEASE

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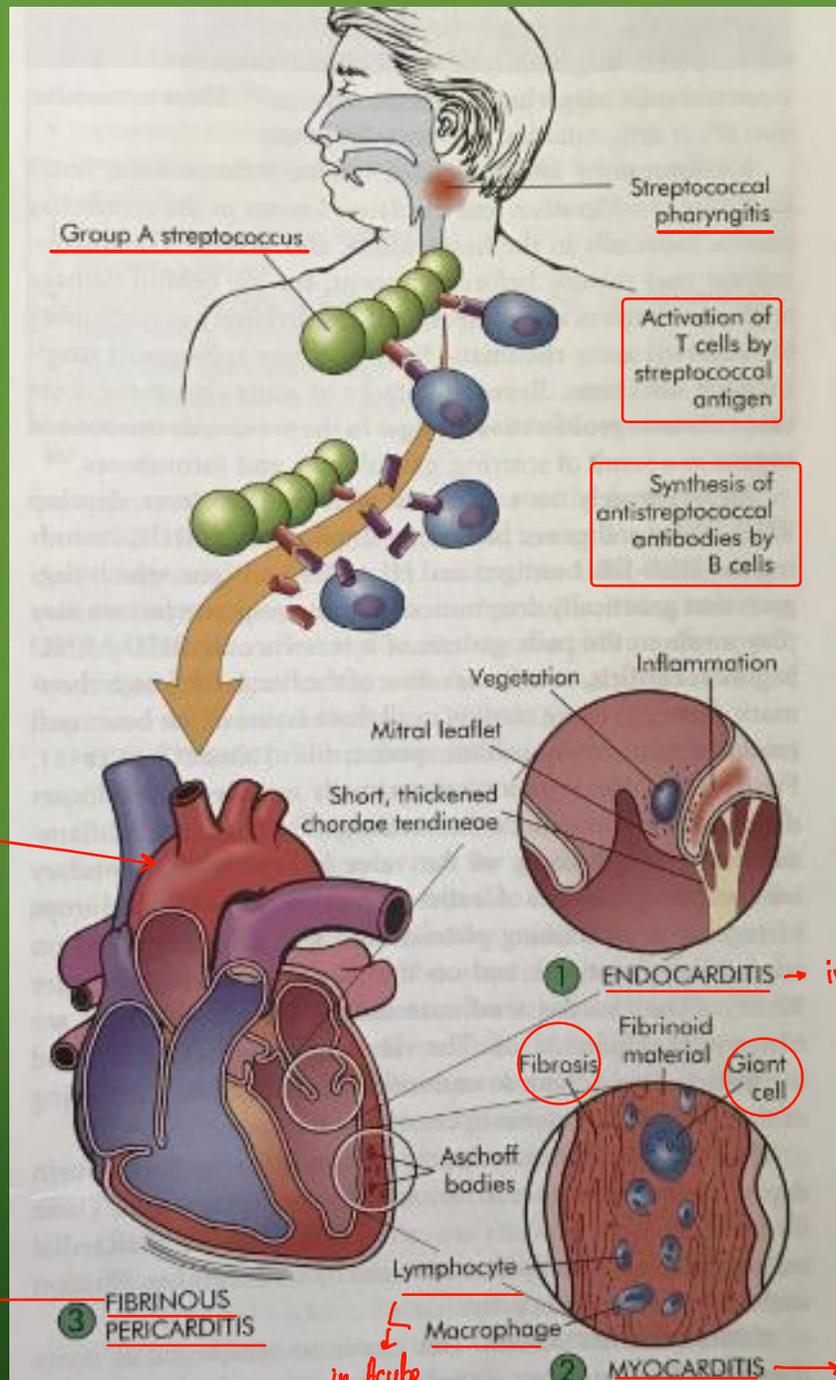
# RHEUMATIC HEART DISEASE

- Rheumatic heart disease is the cardiac manifestation of rheumatic fever.
- Rheumatic fever is an acute, immunologically mediated, multisystem inflammatory disease that occurs after group A  $\beta$ -hemolytic streptococcal infections (usually pharyngitis, but also occasionally infections at other sites, such as skin). → *cellulitis*
- valvular inflammation and scarring produce the most important clinical features, rheumatic heart disease is essentially the only cause of acquired mitral stenosis.

# PATHOGENESIS

- The pathogenic mechanism involves autoantibodies and T cells directed against group A streptococcal cell wall components that cross-react with heart or brain.
- Predisposing factors include genetic and environmental factors such as poverty and overcrowding. → rate of infection ↑

# Pathogenesis



. ASD \*

similar structure  
valv!

All layer ] Endocarditis

1 ENDOCARDITIS → inflammatory valve - vegetation

M.C in children

Restrictive

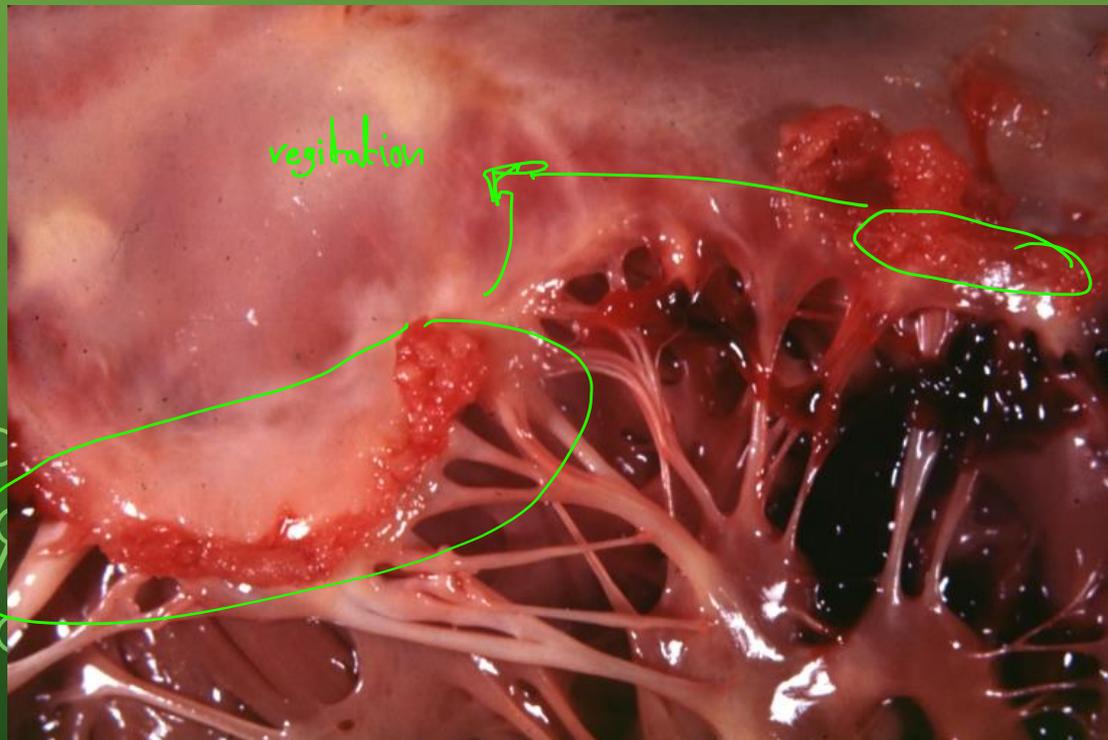
in acute

weakening → Heart failure

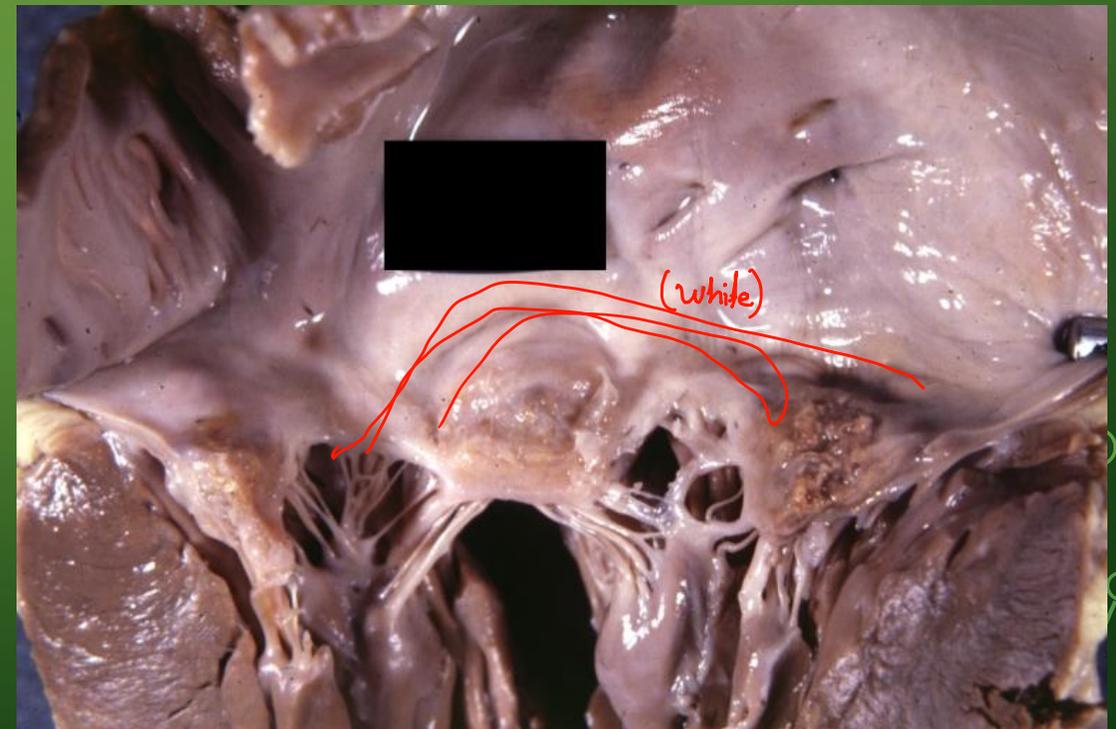
# GROSSING

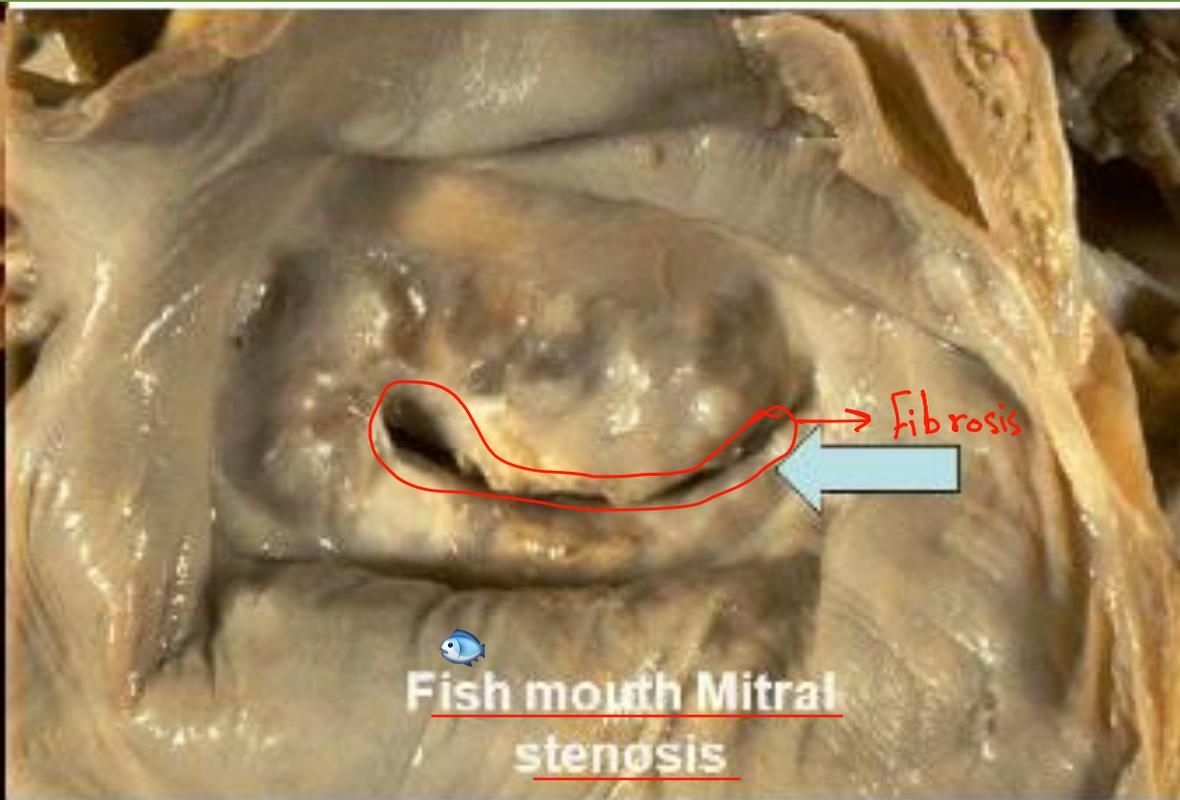
Acute

## MITRAL VALVULITIS



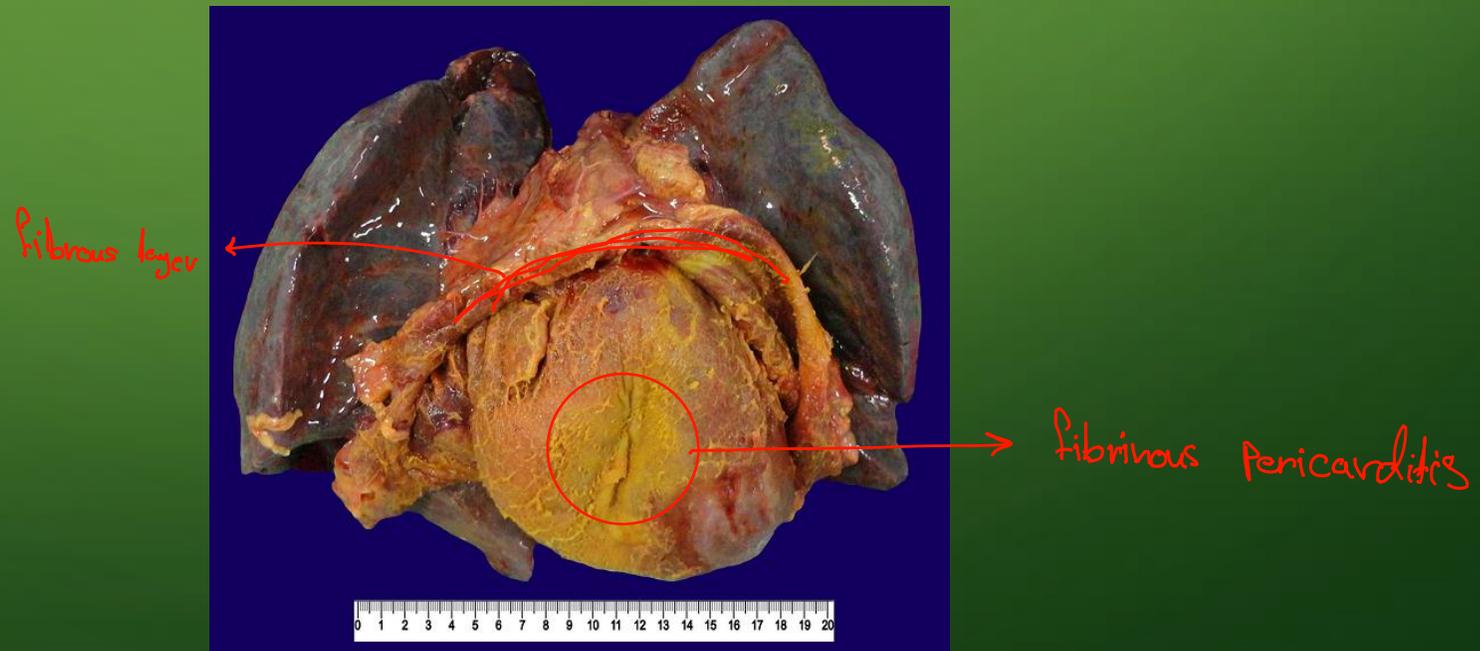
## MITRAL SCARRING





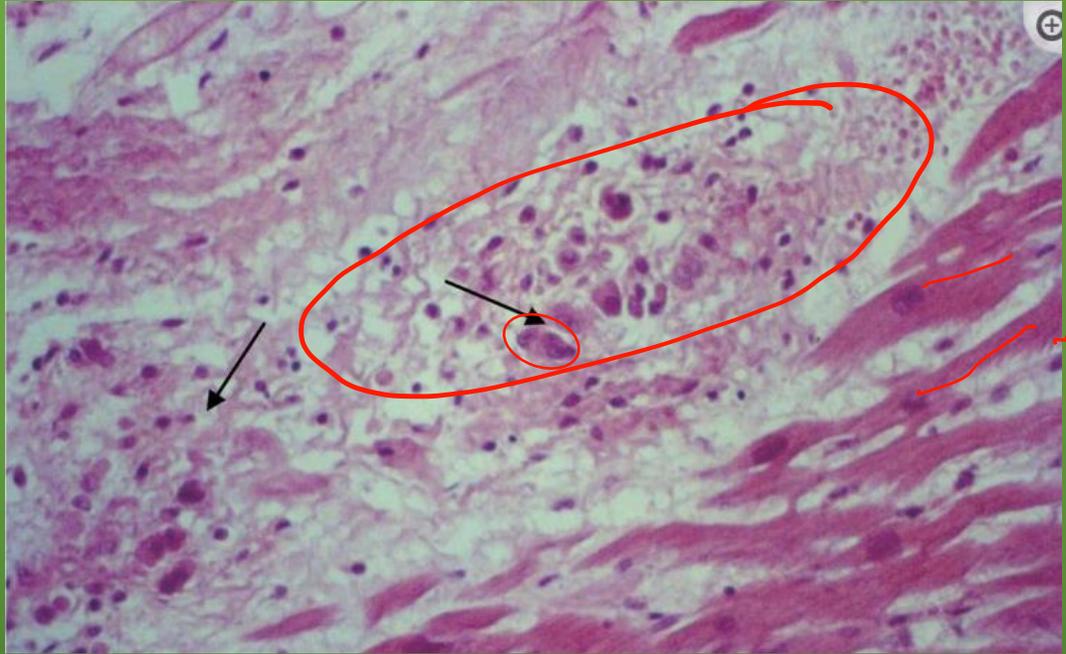
The pericardium may exhibit a fibrinous exudate, which generally resolves without sequelae.

↓  
good prognosis



# HISTOLOGY

- Acute rheumatic valvulitis manifests with :
  - ✓ active inflammation characterized by lymphocytic infiltration, Aschoff bodies, and Anitschkow cells (have abundant cytoplasm and nuclei with chromatin that is centrally condensed ). *Modified macrophage.*
- Chronic rheumatic valvulitis is associated with:
  - ✓ *New blood vessels*  
neovascularization, valvular fibrosis and/or dystrophic calcification.
- During acute rheumatic fever, Aschoff bodies can be found in any of the three layers of the heart—pericardium, myocardium, or endocardium (including valves).



[Giant cell-hypoxyle]  $\leftarrow$   $\uparrow$  myocyte  
 $\leftarrow$  granulomatous inflammation  
 $\uparrow$   
Aschoff nodules

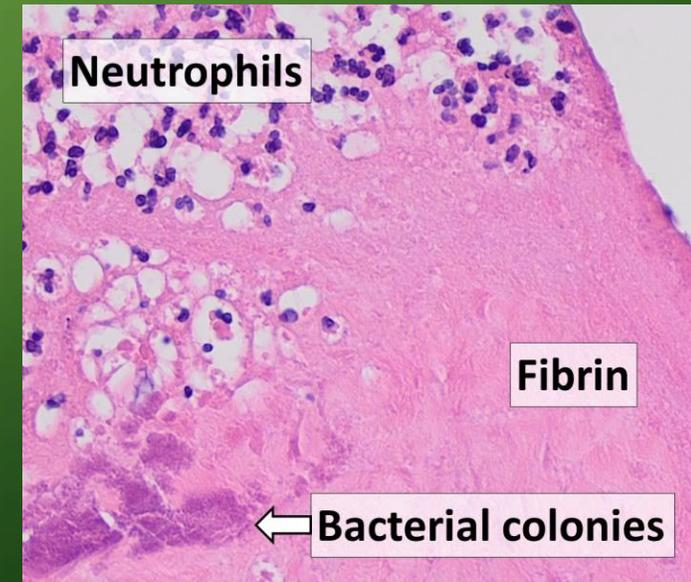
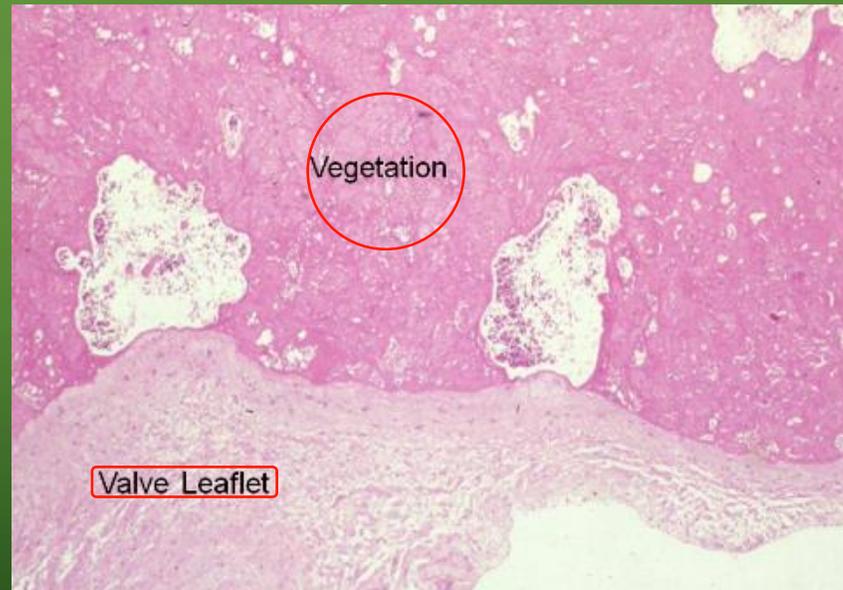
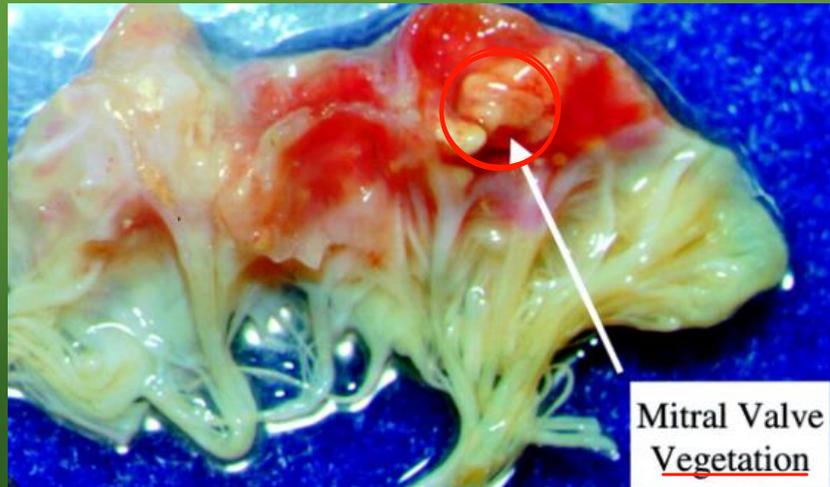


Anitschkow cells.

# HISTOLOGY CONT.:

- Valve involvement results in fibrinoid necrosis and fibrin deposition along the lines of closure forming 1- to 2-mm vegetations—verrucae—that cause little disturbance in cardiac function.

(تسم آخدر)



# CLINICAL FEATURES

- Acute rheumatic fever occurs most often in children; the principal clinical manifestation is carditis.
- However, about 20% of first attacks occur in adults, with arthritis being the predominant feature.
- Symptoms in all age groups typically begin 2 to 3 weeks after streptococcal infection.  
 *on stethoscope walking on snow*  
عند استخدام السماعة، تسمع صوت مثل المشي على الثلج  
*(fibrinous pericarditis)*
- The clinical signs of carditis include pericardial friction rubs and arrhythmias; myocarditis may be sufficiently severe to cause cardiac dilation and resultant functional mitral insufficiency and CHF.

12-10-20

# RHEUMATIC FEVER

## DUCKETT-JONES DIAGNOSTIC CRITERIA

### MAJOR CRITERIA

“CASES” is the Mnemonic

Carditis

Arthritis

Subcutaneous nodules

Erythema marginatum

Sydenham's chorea

### MINOR CRITERIA

“FRAPP” is the Mnemonic

Fever

[Acute phase reactant]

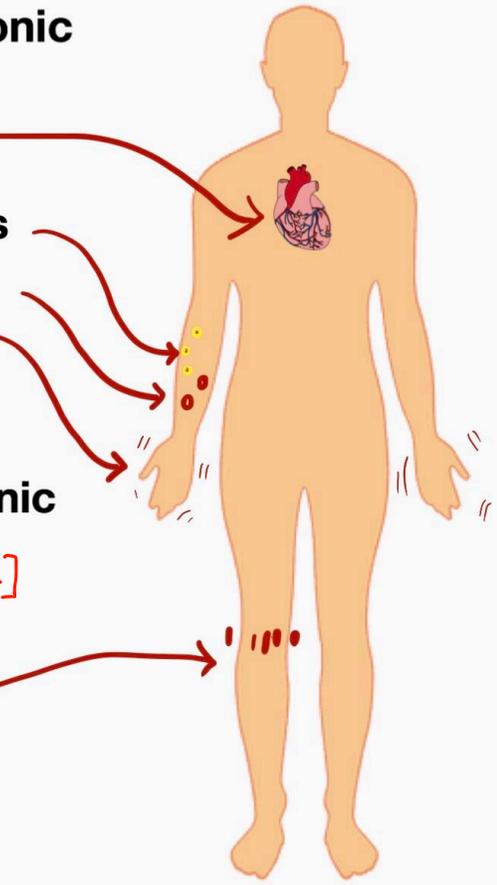
Raised ESR/CRP

Arthralgia

Prolonged PR Interval

Previous RF

neurological sign



(red) سرورجواخمة جریا

## Erythema Marginatum



There must be evidence of streptococcal infection plus:

2 major or 1 major + 2 minor

## LAB

- ESR, CRP.



specific

- Elevated serum titers of antibodies against one or more streptococcal antigens (e.g., streptolysin O or DNAase) .
- Cultures are negative for streptococci at the time of symptom onset

Latex agglutination  
Slide test  
ASO

# TREATMENT

- Treatment strategies for acute rheumatic fever (ARF) can be divided into the following:
  - ✓ Management of the acute attack
  - ✓ Management of the current infection
  - ✓ Prevention of further infection and attacks

→ Penicillin ✓