ACUTE RHEUMATIC FEVER. INFECTIVE ENDOCARDITIS

Minsk BSMU 2024

МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ
БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ
КАФЕДРА ПРОПЕДЕВТИКИ ВНУТРЕННИХ БОЛЕЗНЕЙ

ОСТРАЯ РЕВМАТИЧЕСКАЯ ЛИХОРАДКА. ИНФЕКЦИОННЫЙ ЭНДОКАРДИТ

ACUTE RHEUMATIC FEVER. INFECTIVE ENDOCARDITIS

Учебно-методическое пособие

Минск БГМУ 2024

УДК [616-002.77-036.11+616.126-022](075.8)
ББК 54.1я73
О-76

Рекомендовано Научно-методическим советом университета в качестве учебно-методического пособия 20.12.2023 г., протокол № 12

А в т о р ы: И. И. Бураков, Ю. В. Репина, М. В. Шолкова, Г. М. Хващевская

Р е ц е н з е н т ы: канд. мед. наук, зав. отделом пульмонологии и хирургических методов лечения болезней органов дыхания Республиканского научно-практического центра пульмонологии и фтизиатрии Е. И. Давидовская; каф. пропедевтики внутренних болезней Витебского государственного ордена Дружбы народов медицинского университета

Острая ревматическая лихорадка. Инфекционный эндокардит = Acute
О-76 rheumatic fever. Infective endocarditis : учебно-методическое пособие / И. И. Бураков [и др.]. – Минск : БГМУ, 2024. – 20 с.

ISBN 978-985-21-1517-9.

Содержит сведения об острой ревматической лихорадке и инфекционном эндокардите.

Предназначено для студентов 3-го курса медицинского факультета иностранных учащихся, обучающихся на английском языке по специальности «Лечебное дело».

УДК [616-002.77-036.11+616.126-022](075.8)
ББК 54.1я73

ISBN 978-985-21-1517-9

LIST OF ABBREVIATIONS

ADB — DNase antibodies in serum
ARF — acute rheumatic fever
ASO — Antistreptolysin O
CRP — C-reactive protein
GAS — Group A streptococcus
ESR — erythrocyte sedimentation rate
HIV — human immunodeficiency viruses
IE — infective endocarditis
NYHA — New York Heart Association Functional Classification of heart failure
RF — rheumatic fever
RHD — rheumatic heart disease

EXPLANATORY NOTE

Total duration of classes is 3,0 hours.

Acute rheumatic fever (ARF) and infective endocarditis (IE) have some similarity. They are caused by microorganisms, both are inflammatory diseases, in both cases inflammation can be located at heart valves and at final stage both diseases can lead to valvular deformation and fibrosis.

The key difference of pathogenesis between the two diseases is the following, unlike infective endocarditis, which is purely due to infectious causes, acute rheumatic fever is an autoimmune disease. Clinical signs of ARF and IE have a lot of specific features, presented later.

The purposes are to study definition, etiology, pathogenesis, symptoms, diagnosis, principles of treatment and prevention of acute rheumatic fever and infective endocarditis.

Objectives of the class:

1. To teach the etiology, pathogenesis, clinical signs of acute rheumatic fever (basic and additional complaints, history of the disease, results of inspection, palpation, percussion, auscultation, laboratory and instrumental investigations), principles of treatment and prevention.

2. To teach the etiology, pathogenesis, clinical signs of infective endocarditis (basic and additional complaints, history of the disease, results of inspection, palpation, percussion, auscultation, laboratory and instrumental investigations), principles of treatment and prevention.

ACUTE RHEUMATIC FEVER

Rheumatic fever is a systemic inflammatory process initiated by group A beta-hemolytic streptococcal infections.

Often, younger children in particular do not recall antecedent pharyngitis, which usually occurs 2 to 4 weeks before symptom onset.

Rheumatic fever most often affects children, and onset is usually characterized by an acute febrile illness that can cause large-joint migratory arthritis, central nervous system involvement (Sydenham's chorea), characteristic rash, and carditis with inflammation of heart valves and subsequent damage.

The chronic stage of RF involves all the layers of the heart (pancarditis) causing major cardiac sequelae referred to as rheumatic heart disease (RHD). In spite of its name suggesting an acute arthritis migrating from joint to joint, it is well known that it is the heart rather than the joints which is first and major organ affected. Decades ago, William Boyd gave the dictum "rheumatism licks the joint, but bites the whole heart".

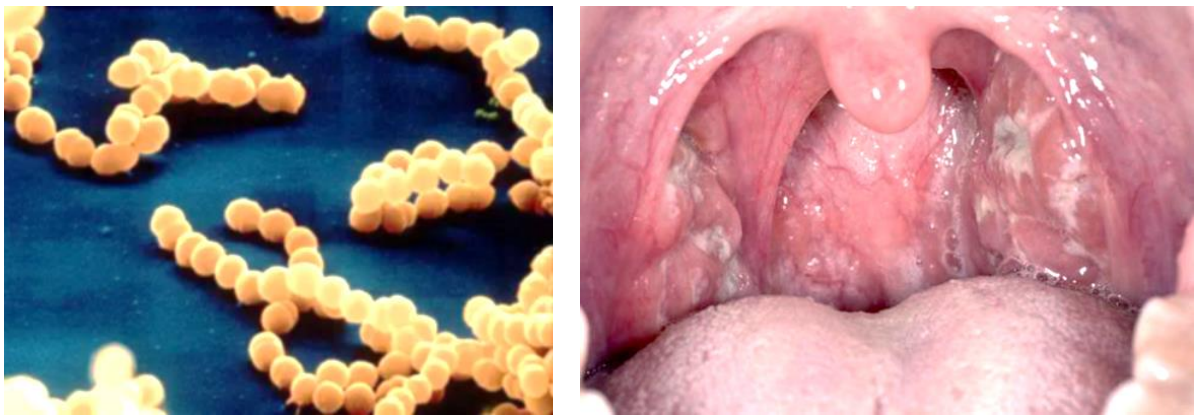
EPIDEMIOLOGY

The disease appears most commonly in children between the age of 5 to 15 years when the streptococcal infection is most frequent and intense. Both the sexes are affected equally, though some investigators have noted a slight female preponderance. The geographic distribution, incidence and severity of acute rheumatic fever is generally related to the frequency and severity of streptococcal pharyngeal infection. The disease is seen more commonly in poor socioeconomic strata of the society living in damp and overcrowded places which promote interpersonal spread of the streptococcal infection. Its incidence has declined in the developed countries as a result of improved living conditions and early use of antibiotics in streptococcal infection.

ARF is common in the Middle and Far East, eastern Europe and South America. It is rare in the UK, western Europe and North America. This decline in the incidence of rheumatic fever (from 10 % of children in the 1920s to 0.01 % today) parallels the reduction in all streptococcal infections and is largely due to improved hygiene and the use of antibiotics.

ETIOLOGY AND PATHOGENESIS

Group A streptococcus (GAS) bacteria (Fig. 1) is a Gram positive, beta-hemolytic coccus in chains. It is responsible for a range of diseases in humans.



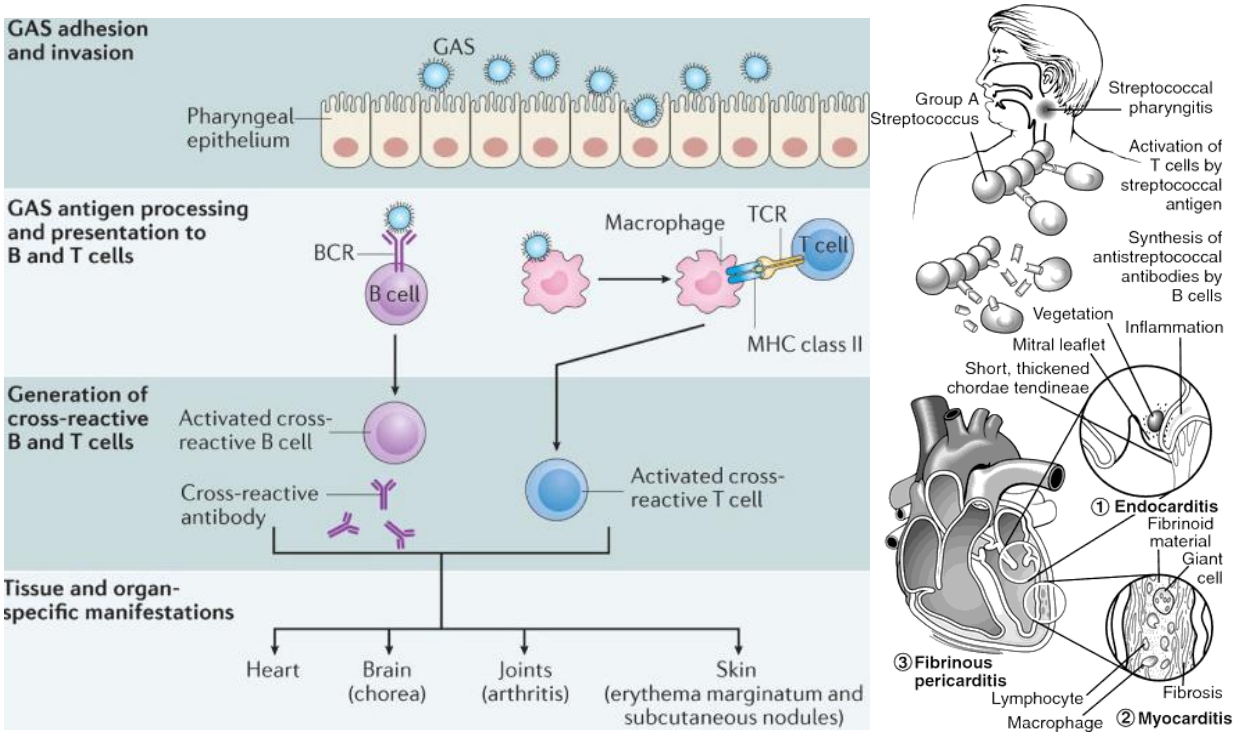
a ***b***

Fig. 1. GAS bacteria:
a — Streptococcus as seen in scanning electron microscope; *b* — Streptococcal Pharyngitis

These diseases include strep throat (acute pharyngitis) and skin and soft tissue infections such impetigo and cellulitis. These can also include rare cases of invasive (serious) illnesses such as necrotizing fasciitis (flesh eating disease) and toxic shock syndrome. Several virulence factors contribute to the pathogenesis of GAS, such as M protein, hemolysins, and extracellular enzymes.

Group A streptococcal (GAS) infectious is the etiologic precursor of acute rheumatic fever, but host and environmental factors are important. Mechanism of ARF includes the body's autoimmune response to an infection caused by GAS. So, AFR is an autoimmune inflammatory process that develops as a sequela of streptococcal infection. Patients with RF have high of antibodies to the antigens of GAS such as anti-streptolysin O (ASO), and DNase antibodies in serum (ADB).

GAS M proteins share epitopes (antigenic-determinant sites that are recognized by antibodies) with proteins found in synovium, heart muscle, heart valve, and other tissue. Patient's immune system produces antibodies against GAS proteins, but molecular mimicry by GAS antigens leads to damage cells and organs of host (Fig. 2).



a ***b***

Fig. 2. Pathogenesis of acute rheumatic fever:
a — immunological issue; *b* — damaged organs

Genetic host risk factors include the D8/17 B-cell antigen and certain class II histocompatibility antigens. Undernutrition, overcrowding, and lower socioeconomic status predispose to streptococcal infections and subsequent episodes of rheumatic fever.The natural history of GAS infection, acute rheumatic fever and chronic rheumatic disease (RHD) are summarized in Fig. 3.

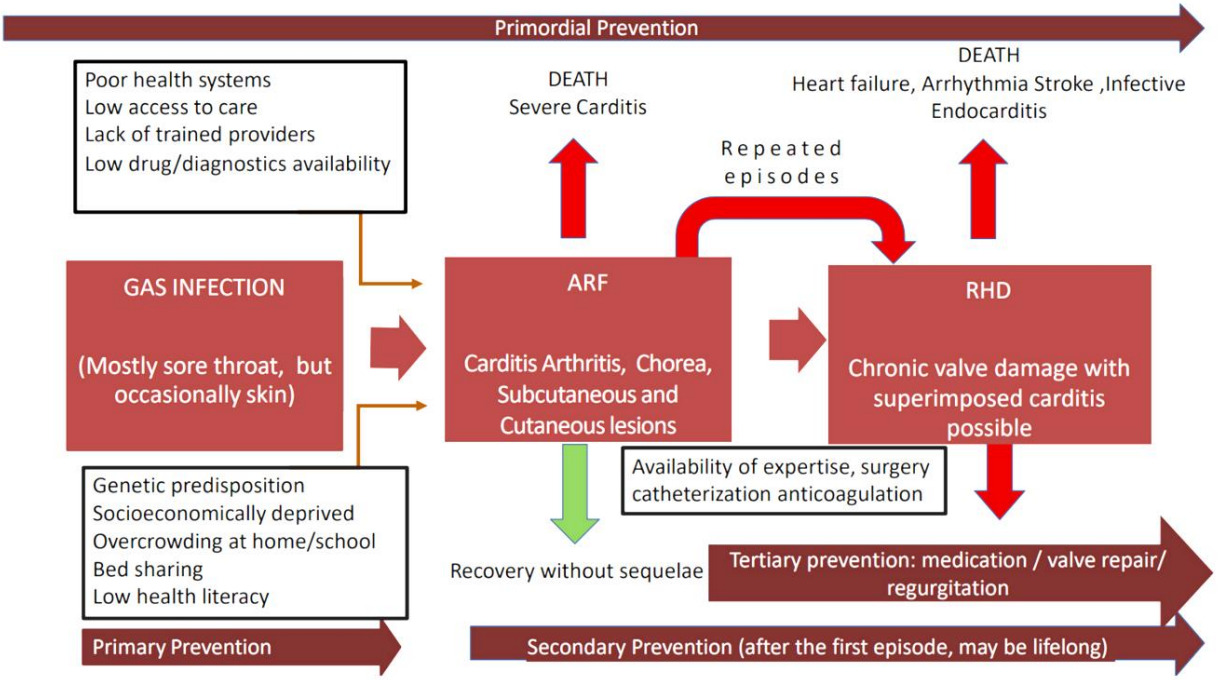


Fig. 3. Natural history of GAS infection

CLASSIFICATION OF RHEUMATIC FEVER

1. Clinical variants:
– acute rheumatic fever;
– chronic rheumatic disease.
2. Clinical manifestations:
a) basic clinical manifestations — carditis, arthritis, chorea, rheumatic nodules, and erythema marginatum;
b) additional clinical manifestations — arthralgia, (poly)serositis, abdominal syndrome.
3. Degree of activity: minimal, moderate, maximal (high) activity.
4. Functional class of chronic heart failure according to NYHA.

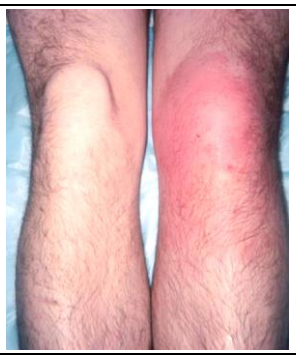
CLINICAL FEATURES

Acute rheumatic fever starts suddenly (usually 2–3 weeks after tonsillitis or pharyngitis), with fever, joint pain, malaise and loss of appetite. There are five most common signs for ARF (Table 1):

1. Arthritis.
2. Carditis.
3. Chorea.
4. Erythema marginatum.
5. Subcutaneous nodules.

Table 1

Clinical Signs of Acute Rheumatic Fever

Carditis	
Carditis is the most important aspect of this disease as it has major long-term implications. Endocarditis, myocarditis and pericarditis can present.	
The diagnosis of carditis requires the finding of – new cardiac murmurs; – cardiomegaly; – pericarditis (pericardial pain, pericardial friction rub, pericardial effusion); – congestive cardiac failure. *Endocarditis involves the valves, particularly the mitral valve. Rheumatic heart disease is the most common serious complication and usually occurs 10 to 20 years after the original attack.*	
Arthritis	
– usually involves the large joints (knees, ankles, elbows, and wrists); – joints are swollen, red, hot and painful; – asymmetrical; – migratory arthritis (inflammation on some joint resolves without treatment and starts on other joint)	
Erythema marginatum	
– Erythema marginatum is a rash with ring shape, macule or papule extends outward while the center returns to normal. – This rash is painless, without pruritus, spreads centrifugally at trunk and proximal parts of extremities. – 10–15 % of patients with ARF	
Subcutaneous nodules	
– Subcutaneous nodules are firm, painless; – about 0.5–1 cm in diameter; – mainly over bony prominences and tendons; – resolve after a few weeks	
Sydenham's chorea	
– 5–20 % of cases, more common in females; – late neurological manifestation (3 months and more after the episode of ARF); – involuntary movements; – face and limbs muscles;	

End of the Table 1

– rapid, irregular, and aimless movements of the arms and legs, trunk, and facial muscles (handwriting disorders, grimaces); – speech may be explosive and halting; – anxiety, sadness, and emotional lability


Other clinical manifestations of acute rheumatic fever are very rare and usually completely reversible (lungs — specific rheumatic pneumonia, pleurisy; digestive system — rheumatic peritonitis; liver — hepatitis, kidneys — proteinuria, hematuria).

LABORATORY AND INSTRUMENTAL TESTS

Laboratory and instrumental investigations are in depends of ARF activity (Table 2).

Table 2

Acute Rheumatic Fever Laboratory and Instrumental Investigations

Laboratory tests	
CBC	↑ ESR, Anemia, Leukocytosis (rare)
Biochemical tests	↑ CRP, ↑ a2-globulin, ↑ γ-globulin, ↑ fibrinogen
Microbiology	– positive GAS throat culture; – positive rapid antigen test for GAS
Immunological tests	↑ GAS antibodies (ASO, anti-DNase B)
Instrumental tests	
ECG	– AV-block 1st or 2nd degree (prolonged PR-interval); – extrasystoles; – decreased voltage; – T wave inversion; – S-T depression
Echocardiography	– valve edema; – mitral regurgitation; – left ventricle dilatation; – pericardial effusion; – ↓ ejection fraction

DIAGNOSIS CRITERIA OF RHEUMATIC FEVER

There is no specific laboratory test for a diagnosis of acute rheumatic fever. Modified Jones criteria for initial diagnosis are in Table 3.

Table 3

Modified Jones Criteria

Major	Minor
Carditis Chorea Erythema marginatum Polyarthritis Subcutaneous nodules	Polyarthralgia, ↑ ESR (> 60 mm/h) ↑ CRP (> 30 mg/L) Fever (≥ 38,5 °C) Prolonged PR interval (on ECG)

Diagnosis of acute rheumatic fever requires 2 major or 1 major and 2 minor manifestations and evidence of group A streptococcal infection (elevated or rising antistreptococcal antibody titer (ASO, anti-DNase B), positive throat culture, or positive rapid antigen test).

TREATMENT

For general management, patients should limit their physical activities if they have symptoms of arthritis, chorea, or heart failure. When the clinical syndrome has subsided (e.g. no fever, normal pulse rate, normal ESR, normal WBC count) the patient can have no physical restrictions.

Treatment includes:

– Antibiotics (10-day course of amoxicillin, cefalexin or azithromycin, or a single injection of benzathine penicillin). Benzathine penicillin G 900 mg (dosage for child is calculated according the weight) given intramuscularly as a single dose.

– Aspirin (children and adolescents 15 to 25 mg/kg orally 4 times a day 2 weeks and taper the dose over another 4 weeks). Other non-steroidal anti-inflammatory drugs may be uses (naproxen).

– Sometime corticosteroids (prednisolone 1 mg/kg orally for patients with moderate to severe carditis).

Prevention of recurrence may be necessary with long-term oral penicillin. The optimal duration of ARF prophylaxis is uncertain. Children without carditis should receive prophylaxis at least for 5 years or until age 21 (whichever is longer). Children with carditis or should receive prophylaxis for more than 10 years.

PROGNOSIS

More than 50 % of those who suffer ARF with carditis will later (after 10–20 years) develop chronic rheumatic valvular disease, predominantly affecting the mitral and aortic valves.

INFECTIVE ENDOCARDITIS

Infective (bacterial, septic) endocarditis (IE) is a severe general infectious disease characterized by inflammation of the endocardium and ulceration of the heart valves in the presence of sepsis.

EPIDEMIOLOGY

IE is a relatively rare but life-threatening disease. In a systematic review of the global burden of IE, crude incidence ranged from 1.5 to 11.6 cases per 100,000 person-years. Untreated, mortality from IE is uniform. Even with the best-available therapy, contemporary mortality rates from IE are approximately 25 %.

It is common to find infective endocarditis as a complication of congenital heart disease or rheumatic heart disease. In developed countries, congenital heart disease is the most important predisposing factor. In approximately 30 % of patients with infective endocarditis, a predisposing factor is acknowledged and identified. If a history of dental procedure is recognized, the time range from the procedure may range from 1 to 6 months prior to the onset of symptoms. The existence of endocarditis after routine heart surgery is low; however, in the setting of prosthetic material use, this can be a predisposing factor.

ETIOLOGY

There are two main microorganisms causing infective endocarditis:

– Streptococcus viridans, which is the normal component of oral cavity flora (50% to 60% of cases);

– Staphylococcus aureus (20 % to 30 % of cases).

Other bacterial causes include Enterococci, Gram negative bacilli HACEK group (Haemophilus species, Aggregatibacter species, Cardiobacterium hominis, Eikenella corrodens, and Kingella kingae), and fungi. Prosthetic valve endocarditis is caused most common by Coagulase negative staphylococci (Staphylococcus epidermidis). In 10 % of IE cases no organism is identified (“Culture negative” endocarditis). Causes for the culture negative endocarditis are prior antibiotic therapy; difficulty in isolating the offending agent, when microorganism is deeply embedded in enlarged vegetation that it cannot be released into blood.

Risk factors for Infective endocarditis are as follows:

– Congenital heart disease;

– Acquired heart disease (rheumatic fever);

– Indwelling vascular catheters;

– Intracardiac devices (prosthetic valve, pacemaker);

– Immunodeficiency (immunodepressive drugs, HIV infection);

– Intravenous drug use (tricuspid valve endocarditis);

– Hemodialysis;

– Bacteriemia (septicemia).

PATHOGENESIS

Normal endothelium is resistant to infection by most bacteria and to thrombus formation. Endothelial injury causes aberrant flow and allows direct infection by virulent organisms and the development of a platelet-fibrin thrombus that subsequently serves as a site of bacterial attachment during transient bacteremia. Microorganisms that cause endocarditis generally enter the bloodstream from mucosal surfaces, the skin, or sites of focal infection.
Pathogenesis of IE is present at Fig. 4.

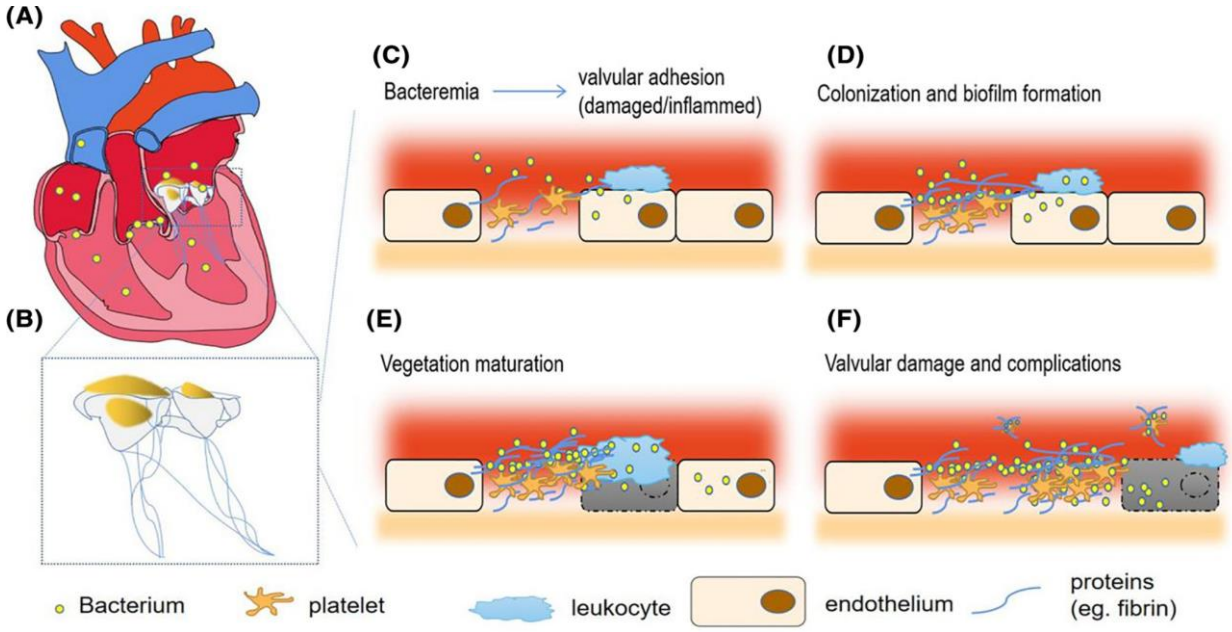


Fig. 4. Pathogenesis of Infective endocarditis
(Clinical Cardiology. 2021. Vol. 44, Is. 3, P. 307–315):
A, *B* — mitral valve vegetations in infectious endocarditis; *C* — bacteria in the bloodstream adhere to damaged or inflamed valves and cause invasive infection; *D* — bacterial persistence and proliferation lead to valve colonization and biofilm formation (especially Staphylococcus aureus); *E* — the valvular lesion progresses further, followed by vegetation maturation; *F* — severe valvular damage and disseminated vegetation particles result in clinical symptoms and various complications

The pathophysiologic consequences of infective endocarditis are the following:

– damage to intracardiac structures;

– embolization of vegetation fragments, leading to infection or infarction of remote tissues;

– hematogenous infection of sites during bacteremia;

– tissue injury due to the deposition of circulating immune complexes or immune responses to deposited bacterial antigens.

PATHOLOGICAL ANATOMY

Bacterial grows at valves is characterized by the presence of ulcerous endocarditis. Ulcerated surfaces become covered with polyp-like thrombotic mass (vegetation) which sometimes looks like cauliflower (Fig. 5). The valves become sclerosed and disfigured.

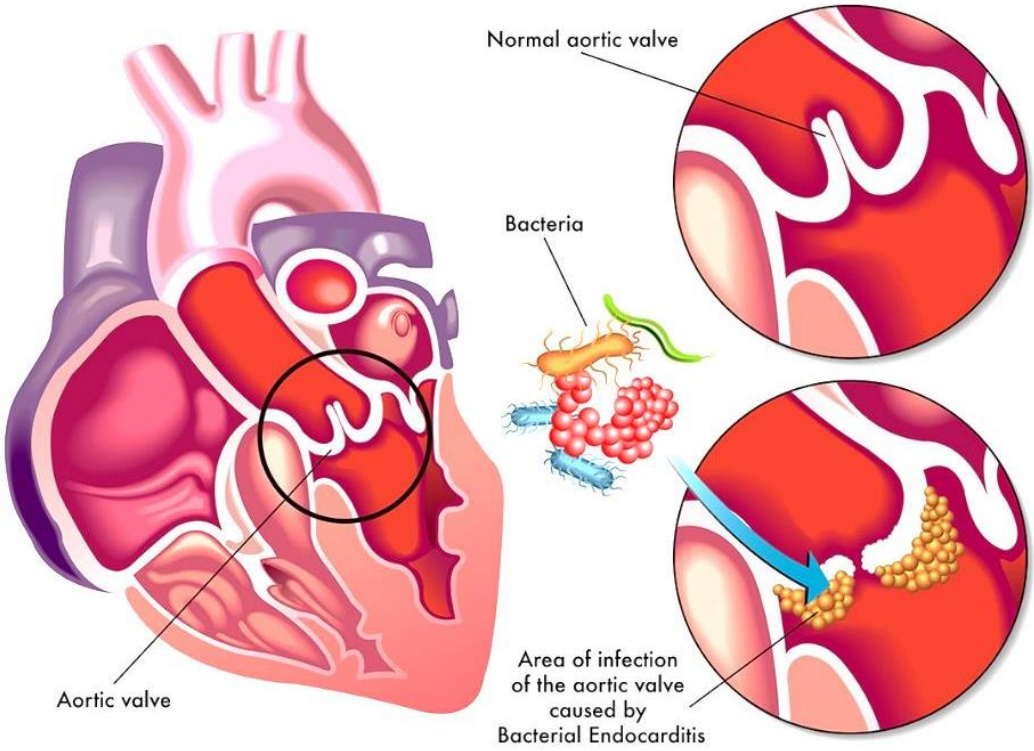


Fig. 5. Vegetation formation in case of Infective endocarditis

Types of infective endocarditis:

– Native valve endocarditis, acute and subacute.
– Prosthetic valve endocarditis, early and late.
– Intravenous drug use-related endocarditis.

CLINICAL PICTURE

The most typical complaints of IE are fever and dyspnea. As a rule, subfebrile fever first develops, which is followed by irregular elevation of temperature to 39 °C and more.

Chills and excessive sweating are characteristic of the infective endocarditis. Other complaints are weakness, weight loss, palpitation, arthralgias (joint pain), myalgias (muscular pain). Pain may be due to embolism (severe headache, heart and chest pain, abdominal, lumbar pains).

Manifestations of central nervous system involvement (in about 35 % of patients) may range from transient ischemic attacks and toxic encephalopathy to brain abscess and subarachnoid hemorrhage.

Objective examination of a patient in case of IE can include following points (Table 4).

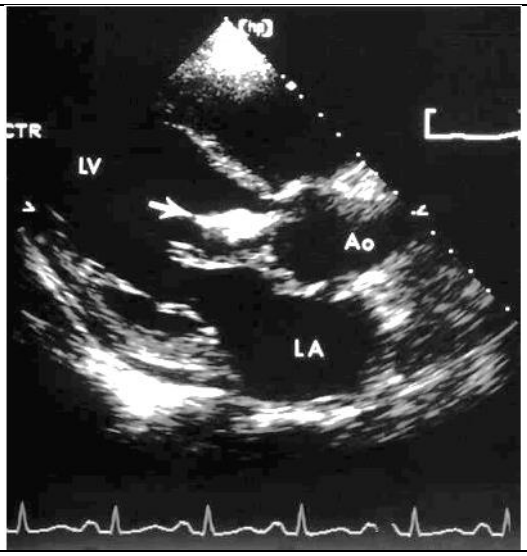
Table 4

Objective Examination in Case of Invective Endocarditis

Method	Result
Visual examination	Pale skin and visible mucosa due to anemia and aortic incompetence. Skin sometimes becomes yellowish-grey (“coffee with milk”). Small hemorrhages in the skin, mucosa of the mouth (especially the soft and hard palate), on conjunctiva, and the eyelid folds (the Libman–Lukin symptom) indicate affection of the joints. Positive Konchalovsky–Rumpel–Leede sign. Digital (nail) clubbing. Osler nodes (painful nodes in the fingers, palms, toes, soles). Roth spots (retinae). Janeway lesions (thenar, hypothenar). Splinter hemorrhages (small linear hemorrhages under the fingernails)
Digital clubbing	
Libman–Lukin symptom	
Roth’s spots	
Janeway lesions	

Method	Result
Osler nodes	
Splinter hemorrhages	
Percussion	Relative heart dullness borders are expanded. Hepatomegaly (due to heart failure). Splenomegaly
Palpation	Pulse is weak. Tachycardia. Apical impulse is shifted to the left. Hepatomegaly. Splenomegaly
Auscultation	Tachycardia. Heart sounds are weak. Murmurs (according the affected valve). Murmurs can be organic and functional. Organic: – Diastolic murmur at aorta in case of aortic insufficiency. – Systolic murmurs of mitral and tricuspid insufficiency. Functional murmurs due to anemia and ventricle dilatation
Laboratory investigations	
CBC	Hypochromic anemia (caused by increased hemolysis and inhibited erythropoiesis), ↑ ESR, neutrophilic leukocytosis, monocytosis
Urinalysis	Proteinuria, hematuria, casts
Biochemical blood tests	Dysproteinemia (hypoalbuminaemia, ↑ gamma-globulins)
Blood culture media	Positive blood culture may detect the causative agent of the endocarditis
Instrumental investigations	
ECG	Tachycardia. Conduction disorders (His bundle block, AV block). Signs of ischemia (ST segment elevation) in case of septic embolism to coronary artery
Transthoracic echocardiography	Vegetations (absence of a visible vegetation does not exclude diagnosis)

End of the Table 4

Method	Result
Parasternal long-axis view echocardiogram of a patient with a large aortic valve vegetation (arrow). Ao, Aorta; CTR, cardiothoracic; LA, left atrium; LV, left ventricle. *(From Zipes DP. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 7th ed. Philadelphia: Saunders; 2005)*	
Abdominal ultrasound	Septic emboli (spleen infarction, liver infarction, liver abscess, mycotic aneurism)

DIAGNOSIS OF INVECTIVE ENDOCARDITIS

Clinical picture of infective endocarditis is variable and nonspecific. Table 5 shows modified Duke criteria of diagnosis.

Table 5

Diagnosis of Infective Endocarditis (modified Duke criteria)

Major criteria	Minor criteria
1. Positive blood culture – Typical organism from two cultures. – Persistent positive blood cultures taken > 12 hours apart. – Three or more positive cultures taken over > 1 hour. **2. Endocardial involvement** – Positive echocardiographic findings of vegetations. – New valvular regurgitation	– Predisposing valvular or cardiac abnormality. – Temperature (pyrexia) ≥ 38 °C. – Immunologic phenomena. – Microbiologic evidence. – Blood cultures suggestive: organism grown but not achieving major criteria. Suggestive echocardiographic findings

Definite endocarditis = two major, or one major and three minor, or five minors.

Possible endocarditis = one major and one minor, or three minors.

TREATMENT OF ENDOCARDITIS

1. Antibiotics.

Parenteral antibiotics preferred to ensure consistent and therapeutic antibiotic levels. Antibiotic choice should be guided by culture and sensitivity results. Empiric antimicrobial therapy (before cultures and sensitivities are known) may

include ampicillin, gentamicin, fluocinolones, vancomycin. When blood culture and microorganism sensitivity are available, antibiotics are prescribed according the findings. Duration of therapy is 4 to 6 weeks, depending on the etiologic agent (shorter courses associated with risk of relapse).

2. Surgery valvular replacement.

Surgery is indicated in case of severe heart failure, severe valve dysfunction, prosthetic valve infection, invasion beyond the valve leaflets, recurrent systemic embolization, large mobile vegetations, or persistent sepsis despite adequate antibiotic therapy for more than 5–7 days.

ENDOCARDITIS PROPHYLAXIS

Preventive measures to reduce the risk of infective endocarditis include:

– Maintenance of oral hygiene.

– Timely treatment of infection with pathogens that cause endocarditis.

– Antibiotic prophylaxis prior to invasive procedures.

Antibiotics prophylaxis should be offered to patients with high-risk cardiac conditions who are undergoing procedures that are likely to cause bacteremia. Amoxicillin, cefazolin, ceftriaxone, azithromycin or clarithromycin can be used two times (one hour before procedure and two hours after).

Procedures likely to cause bacteremia include:

– Dental procedures that involve manipulation of the gingiva or periapical region of the teeth, or perforation of the oral mucosa (not routine dental cleaning).

– Procedures of the respiratory tract that will lead to an incision or biopsy of the respiratory mucosa.

– Gastrointestinal or Genito-urinal procedures, only in patients with active infections.

– Procedures involving infected skin or musculoskeletal tissue.

– Cardiac surgery involving placement of prosthetic material.

High-risk cardiac conditions include:

– Prosthetic cardiac valves, bioprosthetic and homograft.

– Presence of prosthetic material used for valve repair.

– Previous infective endocarditis.

– Unrepaired cyanotic congenital heart disease, including palliative shunts and conduits.

– Completely repaired congenital heart defect with prosthetic material or device during the first 6 months after the procedure.

– Repaired congenital heart disease with residual defects at the site or next to the prosthesis.

– Cardiac transplant recipients who develop cardiac valvopathy.

SELF-ASSESSMENT QUIZ

1. Which species of bacteria typically causes infective endocarditis?

a) Staphylococcus;
b) Helicobacter;
c) Bacillus;
d) Mycobacterium.

2. Which of the following Duke criteria is a major criterium for infective endocarditis?

a) Predisposing heart condition or intravenous drug use;
b) Blood cultures positive for typical infective endocarditis organisms;
c) Immunologic findings such as glomerulonephritis;
d) positive GAS throat culture.

3. A 46-year-old man with history of recent intravenous drug abuse presents with fever and is found to have infectious endocarditis. He states that the heroin he injects is not always pure. What cardiac valve do you think he is most likely to have an infectious endocarditis vegetation on?

a) Mitral;
b) Aortic;
c) Tricuspid;
d) Pulmonary.

4. Which of the following is NOT a significant risk-factor for endocarditis?

a) Congenital valve disease;
b) Prosthetic heart valve;
c) Infectious endocarditis 5 years ago;
d) Rheumatic heart disease as a child;
e) Pneumonia in the last 30 days;
f) Recent colonoscopy.

5. Which symptom presented is NOT a sign of acute rheumatic fever?

a) truncal rash;
b) joint tenderness;
c) nausea;
d) handwriting disorders.

6. While most people with rheumatic fever recover, which part of the body may be permanently damaged?

a) digestive tract;
b) ears;
c) heart;
d) lungs.

7. Which of the following are usually the first symptoms of rheumatic fever? Select all that apply.

a) Palpitation;
b) Fever;
c) Joint pain;
d) Rash.

8. According to the Jones Criteria, which of the following sets of symptoms would indicate a positive diagnosis for acute rheumatic fever? Select all that apply.

a) Carditis, fever, and an elevated WBC count;
b) Positive strep throat culture, arthritis, chorea;
c) Recent scarlet fever, carditis, fever, arthralgia;
d) Elevated C-reactive protein, carditis, fever.

Answers: 1 – a; **2** – b; **3** – c; **4** – e; **5** – c; **6** – c; **7** – b and c; **8** – b and c.

REFERENCES

Basic

1. *Пронько, Т. П.* Пропедевтика внутренних болезней = Propedeutics of internal diseases : учеб. пособие / Т. П. Пронько. Минск : Адукацыя і выхаванне, 2020. 472 с.

Additional

2. *Bickley, L. S.* Bates' guide to physical examination & history taking / L. S. Bickley ; guest ed. R. M. Hoffman. Philadelphia [etc.] : Wolters Kluwer, 2017. 1034 p.

3. *Consensus* guidelines on pediatric acute rheumatic fever and rheumatic heart disease. Working Group on Pediatric Acute Rheumatic Fever and Cardiology Chapter of Indian Academy of Pediatrics / A. Saxena [et al.] // Indian Pediatr. 2008. Vol. 45 (7). P. 565–73.

4. *2023* ESC Guidelines for the management of endocarditis: Developed by the task force on the management of endocarditis of the European Society of Cardiology (ESC) Endorsed by the European Association for Cardio-Thoracic Surgery (EACTS) and the European Association of Nuclear Medicine (EANM) / V. Delgado [et al.] // European Heart Journal. ehad193. https://doi.org/10.1093/eurheartj/ehad193.

TABLE OF CONTENTS

Учебное издание

Бураков Иван Иванович
Репина Юлия Викторовна
Шолкова Мария Владимировна
Хващевская Галина Михайловна

ОСТРАЯ РЕВМАТИЧЕСКАЯ ЛИХОРАДКА. ИНФЕКЦИОННЫЙ ЭНДОКАРДИТ

ACUTE RHEUMATIC FEVER. INFECTIVE ENDOCARDITIS

Учебно-методическое пособие

На английском языке

Ответственный за выпуск Э. А. Доценко
Переводчик Ю. В. Репина
Компьютерная вёрстка Н. М. Федорцовой

Подписано в печать 08.04.24. Формат 60×84/16. Бумага писчая «Xerox office».
Ризография. Гарнитура «Times».
Усл. печ. л. 1,16. Уч.-изд. л. 0,85. Тираж 90 экз. Заказ 183.

Издатель и полиграфическое исполнение: учреждение образования
«Белорусский государственный медицинский университет».
Свидетельство о государственной регистрации издателя, изготовителя,
распространителя печатных изданий № 1/187 от 24.11.2023.
Ул. Ленинградская, 6, 220006, Минск.