Cardiac inflammations (pediatrics)

[1]Infective Endocarditis

Infective Endocarditis (IE) is a disease caused by an infectious agent that affects the endocardium, heart valves and adjacent structures. Inflammation can be caused by bacteria, fungi, chlamydia, rickettsia or viruses.

Risk Factors

Risk factors in children:

- congenital heart defects;
- rheumatic heart defects (rare);
- iatrogenic – long-term central venous catheters;
- intravenous drug use;
- bicuspid aortic valve;
- mitral valve prolapse with regurgitation;
- degrees cardiac operations using conduits and vascular prostheses, with artificial valves.**Cite error: Invalid <ref> tag; invalid names, e.g. too many**

Classification

- IE native flaps,
- IE of drug addicts (predisposes to tricuspid valve involvement with risk of pulmonary embolism),
- IE of valve prostheses (early/late onset - limit 2 months after surgery).

The division of IE into acute and subacute form is already obsolete and is not used. Division according to the inducing agent is recommended. Microorganisms with low virulence (e.g. α-hemolytic streptococci) usually cause a "subacute" form, on the contrary, *Staphylococcus aureus* and other pyogenic bacteria cause "acute" forms.

Risks of infective endocarditis

high risk;

- valve prostheses (for life),
- degrees heart surgery (up to 6 months after surgery),
- aortic defects,
- Tetralogy of Fallot,
- mitral insufficiency,
- PDA,
- VSD,
- CoA,
- Marfan syndrome,
- History of IE.

medium risk;

- mitral stenosis,
- tricuspid defects,
- mitral prolapse,
- hypertrophic cardiomyopathy.

Clinical picture

We always suspect IE in high-risk patients (see above) with febrile condition. It is always necessary to keep in mind the mitigated forms of IE at p.o. treatment with antibiotics, which was administered out of embarrassment, fevers may then subside. Endocarditis is most often manifested by ``temperatures *and* ``*nonspecific problems* such as myalgia, arthralgia, headaches, fatigue. If the disease lasts longer, we find splenomegaly, the skin has a color with a touch of white coffee (caffé au lait). Other late symptoms are embolization manifestations on the periphery: splinter-like subungual hemorrhages, petechiae on the skin or subconjunctivally, red spots on the palms (*Janeway's spots*), painful induration on the tips of the fingers (*Osler's nodes*). Embolization may reveal fundus examination (hemorrhagic retinal lesions = Roth spots) or hematuria. In up to 30% of patients, the first clinical symptom of IE may be an acute embolic cause. The basin of the internal carotid artery is most often affected. Clinical symptoms are hemiplegia, aphasia, mental disorders, rarely blindness with retinal artery involvement. In general, IE of the left heart causes embolization to the periphery with subsequent ischemia, infarction in sterile emboli, abscesses in infectious emboli, or mycotic aneurysm. Embolization from the right heart to the lungs is often asymptomatic due to the good filtering properties of the lungs, or symptoms of pulmonary embolism with subsequent cough, auscultatory and X-ray findings on the lungs are manifested.

Sometimes there may even be a picture of *Löhlein's nephritis* with hematuria, proteinuria and a decrease in glomerular filtration. It is a manifestation of microembolization to the kidneys or a consequence of focal or diffuse glomerulonephritis, which causes deposits of immune complexes in the glomeruli. Up to 20% of children have neurological symptoms: meningitis, brain abscesses, toxic encephalopathy.

Significant findings are a newly formed or changed *heart murmur* due to valve involvement, rarely the inflammatory process can affect the conduction system of the heart and cause AV block. Heart failure is the most common cause of deathi.

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Diagnostics

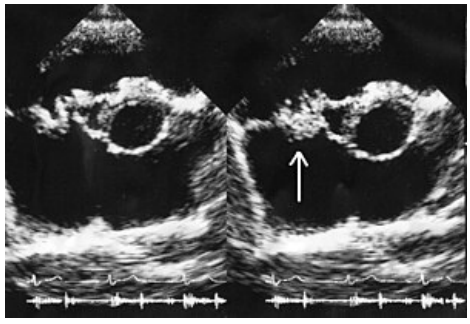
Laboratory examination

From common laboratory findings, high sedimentation, leukocytosis, microscopic hematuria, proteinuria, positive rheumatoid factor, elevated CIK values are indicative of the diagnosis of IE. We often find anemization and hypergammaglobulinemia. Correct collection of **blood culture'** is crucial for the diagnosis and treatment of IE. We take 3 blood cultures within 24 hours, and in case of negative findings on the second day of incubation, another 2 blood cultures are taken. In case of clinical suspicion of IE, blood cultures are also taken from subfebrile or afebrile patients. Blood culture negativity may be due to previous antibiotic therapy, IE caused by rickettsiae, chlamydiae, viruses, or slow-growing organisms. Molecular biological methods such as PCR bring further possible precision in the diagnosis of IE. Negative blood cultures can also support the diagnosis of sterile thrombotic endocarditis occurring most often in antiphospholipid syndrome.

False positivity of blood cultures is caused by contamination during non-sterile sampling. It is most often coagulase negative staphylococci, corynebacteria, transiently colonizing enterobacteria, pseudomonads, etc. The etiological agent is evidenced by repeated findings, isolation of the same strain from other biological samples and a corresponding clinical picture.

Echocardiography

Echocardiography is a very valuable method for confirming IE. This examination reliably reveals vegetation on the endocardium and valves and is also important in monitoring the development of possible valvular regurgitation or other heart findings. For unclear findings, we also use transesophageal echocardiography. Echocardiographic diagnosis of IE on an artificial valve is very difficult, in which vegetations are often hidden in the shadow of a strong signal caused by echodense material.



Tricuspid valve vegetation (ECHO)

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Special examination

When embolization into the pulmonary or systemic flow is suspected, special imaging examinations CT scan, MRI are indicated to prove or rule out septic emboli or abscesses.

We always consult a dentist or ENT doctor to rule out focal infection. Remediation of the infectious site is carried out during the treatment of IE.

Diagnostic criteria

Currently, the criteria proposed by Durack from Duke University in the USA (the so-called Duke criteria) are recommended for the diagnosis of IE, which are based on a combination of clinical, laboratory and echocardiographic findings. According to these criteria, patients with suspected IE are divided into 3 categories: *proven IE, possible IE* and *ruled out IE*.

Definition of IE Criteria (Duke Criteria):

main criteria

- *positive blood culture:* from 2 different blood samples a typical microorganism for IE was detected (*Streptococcus viridans* including nutritional variants or *Streptococcus bovis*, or microorganisms of the *HACEK* group; *Staphylococcus aureus* ' or *Enterococcus spp.*, if no other primary source of infection was detected) / repeatedly positive blood cultures, if there was: the same finding in 2 blood cultures taken within 12 hours or more or the same finding in 3 or 3 out of 4 blood cultures, if the interval between the first and last sampling was greater than 1 hour
- *signs of endocardial involvement:* echocardiographic findings corresponding to IE (fluttering intracardiac structures on the valve or on surrounding structures at the site of accelerated blood flow, or on foreign material for which there is no other anatomical explanation; abscess; newly formed paravalvular dehiscence at the site of artificial valves) / newly formed valve regurgitation

secondary criteria

- *predisposition:* structural heart disease, abusus i.v. drugs,
- "temperatures" ≥ 38.0° C,
- *vascular symptoms:* embolization, septic pulmonary infarction, intracranial hemorrhage, conjunctival hemorrhage, and skin petechiae,
- *immunological symptoms:* glomerulonephritis, Osler nodules, Roth spots, rheumatoid factor,
- *microbiological finding:* a positive blood culture that does not meet the main criteria above, or serological evidence of active infection consistent with IE,
- "Echocardiographic finding" corresponding to IE, but not meeting the above main criteria.

Diagnosis of IE (Duke Criteria):

proven IE

- pathological criteria (at least 1 criterion): proven microorganism by culture or histologically in vegetation or embolization of vegetation or intracardiac abscess, or histological evidence of active IE in vegetation or in intracardiac abscess,
- clinical criteria: 2 main criteria, or 1 main and 3 minor criteria, or 5 minor criteria.

maybe IE
1 main + 1 minor criteria / 3 minor criteria

IE excluded
proven other diagnosis explaining the symptoms of the disease / disappearance of IE symptoms during 4 days of antibiotic treatment / absence of IE findings during surgery or at autopsy after antibiotic treatment during 4 days

Therapy

For ``empirical treatment *or when the blood culture is negative, we choose a combination of oxacillin 200 mg/kg/day every 4 hours + gentamicin 3 mg/kg/day every 12 hours IV, for patients allergic to penicillins then vancomycin 40 mg/kg /day for 6 hours + gentamicin 3 mg/kg/day for 12 hours i.v.*

With a positive blood culture, we take into account the type of microbe detected, its sensitivity and MIC. As a rule, when penicillin-sensitive streptococci are detected, we administer Penicillin G 200,000–400,000 I.U./kg/d i.v. at 4 p.m., possibly + gentamicin. When enterococci and other resistant streptococci are detected, we administer ampicillin 200–300 mg/kg/day every 6 hours i.v. + gentamicin. Gram-negative IE (HACEK) is treated with a combination of III cephalosporins. generation, e.g. ceftriaxone 100 mg/kg/day every 12 hours i.v. or ampicillin 200–300 mg/kg/day IV every 6 hours + gentamicin 3 mg/kg/day IV every 12 hours, Fungal IE is treated with amphotericin B: the initial test dose is 0.1 mg/kg, if it is well tolerated, we increase the dose to 0.5 mg/kg for 1 day and continue for at least 6–8 weeks with a maintenance dose of 1 mg/kg/d i.v. It is usually necessary to supplement the surgical procedure (replacement of infected cstupidly, excision of infected tissue). Aminoglycosides are administered for 14 days (longer administration is associated with a high risk of nephrotoxicity), other antibiotics for 4–6 weeks.

'*Generally* we always choose bactericidal ATB, when combining them we make sure to achieve a synergistic effect. Periodic determination of serum bactericidal activity and monitoring of serum concentration are important, especially with potentially toxic antibiotics (gentamicin, vancomycin). We demonstrate the effectiveness of ATB therapy by eradicating bacteremia in blood culture. In the first 8 weeks after the end of treatment, periodic blood culture control is important, because this is the period when most relapses occur.

In the treatment proposal, we can follow the available guidelines in even more detail, from the current recommendations, for example, according to the American Heart Association ([www.americanheart.org]).

Prevention

Prevention of IE consists of targeted administration of antibiotics to all at-risk individuals prior to surgical or diagnostic procedures known or suspected to cause transient bacteremia. As a rule, these are procedures in the oral cavity, nasopharynx, digestive or urogenital tract. [2] IE prevention significantly reduced the incidence of this devastating infection after its introduction. The most important thing is to prevent bacteremia in children with structural heart disease. Pay particular attention to the timely treatment of all dental affections, including minor tooth decay, even the first dentition, increased oral hygiene, vigorous therapy of purulent skin affections and respiratory bacterial infections. Prevention of IE, however, does not mean flat-rate treatment of all even non-bacterial infections with antibiotics or permanent administration of antibiotics. Cardiology patients are provided with identification with established principles. In summary, it can be said that the most effective prevention of IE is timely and complete correction of the heart defect.

diseases requiring routine IE prevention

- with a congenital heart defect, with the exception of an atrial septal defect,
- with rheumatic or other valvular disease,
- with obstructive form hypertrophic cardiomyopathy,
- with mitral valve prolapse and concomitant regurgitation.[2]

diseases requiring IE risk prevention

- the first 6 months after cardiac operations and after interventional catheterization procedures,
- lifelong in patients with an artificial valve including bioprostheses and allografts, after aortopulmonary coupling operations
- in complex cyanotic heart defects (functionally single ventricle, Tetralogy of Fallot, transposition of great arteries)
- after IE.

diseases not requiring IE prevention

- isolated atrial septal defect,
- atrial septal defect and open trachea 6 months after surgery without residual findings,
- mitral valve prolapse without regurgitation,
- past febris rheumatica or Kawasaki disease without valvular involvement,
- functional murmurs,
- implanted pacemaker or defibrillator,
- coronary bypasses.

performances requiring prevention of IE in patients at risk

- dental procedures, accompanied by bleeding from the gums or mucous membrane, including professional cleaning of tartar,
- tonsillectomy and adenotomy,
- surgery affecting the mucous membrane of the intestines or the respiratory system,
- bronchoscopy with a rigid bronchoscope,
- dilatation of the esophagus and sclerotization esophageal varices,
- gall bladder surgery,
- cystoscopy and urethral dilation,
- vascularization of the bladder, if an infection is present*,
- urological operations, if there is an infection*,
- prostate surgery,
- incision and drainage of infected tissues*,
- vaginal delivery, if there is an infection*,
- vaginal hysterectomy.

> asterisk – for these procedures, in addition to the recommended prophylaxis, antibiotics are administered according to sensitivity

performances that do not require IE prevention

- dental procedures in which there is no bleeding from the gums or mucous membranes, e.g. treatment of tooth decay above the level of the gums,
- loss of the first dentition,
- cardiac catheterization diagnostic,
- endotracheal intubation,
- bronchoscopy with a flexible bronchoscope including biopsy*,
- endoscopic GIT examination including biopsy*,
- transesophageal echocardiography,
- section cesarean,
- if no infection is present: uncomplicated delivery, dilatation of the cervix and curettage, insertion and removal of the intrauterine body*.

> asterisk - except for the risk group of patients

This article has been translated from WikiSkripta; ready for the **editor's review**.

Myocarditis

Myocarditis is a disease characterized by an inflammatory infiltrate of the heart muscle with necrosis or degeneration of adjacent myocytes. They form a very clinically variable group, from the most severe forms of severe **heart failure** or sudden death, to a mild or asymptomatic course of the disease. Inflammatory heart diseases rarely affect the pericardium pericardium , myocardium or endocardium itself, , more often the inflammation affects the whole heart, ie we speak of **pancarditis**.

Etiology

Infection

- enteroviruses: (the most common cause is ***Coxsackie B***, viruses , the most severe course is described in serotypes B3 and B4), echoviruses are less common,
- influenza,
- adenoviruses,
- parvovirus B19,
- parotitis,
- EBV, cytomegalovirus,
- hepatitis C virus,
- mykoplasma,

- chlamydia,
- borrelie,
- neisseria,
- leptospira,
- streptococci,

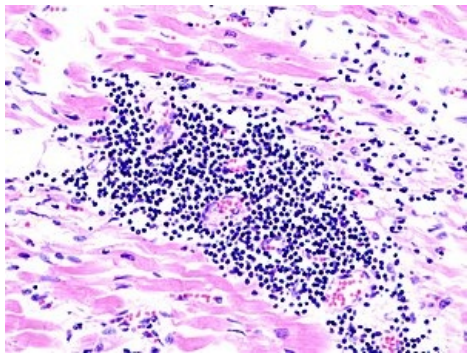
Autoimmunity

- Systemic lupus erythematosus,
- Juvenile idiopathic arthritis,
- Kawasaki disease,

Allergy
Idiopathic form.

Pathogenesis

Viral myocarditis is now considered a three-phase disease: the viral infection phase, the autoimmune phase and, in genetically predisposed individuals, the phase with transition to dilated cardiomyopathy. These 3 phases can flow smoothly into each other with a difficult to distinguish transition. However, each phase differs in pathogenesis, diagnosis and, to some extent, therapeutic approach.


Microscopic picture of viral myocarditis

Phase 1 = viral phase.

Viruses enter the body through the gastrointestinal (enteroviruses) or respiratory tract (adenoviruses). The critical phase of viral infection is the entry of the viral genome into myocytes. This is made possible mainly by binding to the immunoglobulin receptor on the surface of myocytes (CAR = coxsackie-adenovirus receptor). The virus proliferates and subsequently the immune system.is activated . Virus titers gradually fall and antibodies levels rise . However, the activation of the immune system can continue despite the elimination of the virus.

Phase 2 = autoimmune.

Fragments of viral proteins on the surface of myocytes are bound to newly synthesized HLA class I molecules. Monocytes release cytokines (IL-1, IL-6, IL-12 a TNF-alfa), which, among other things, activate T cells and stimulate B - Lymphocytes proliferation and differentiation and antibody production. The formation of an antigen-antibody complex activates the complement system. IL-2 activates cytotoxic T cells, which preferentially destroy myocardial cells with viral protein fragments. Naturally cytotoxic NK cells are activated by interferon and specifically destroy only virus-infected myocytes. Both T-lymphocytes and NK cells thus reduce the number of contractile cells.

Phase 3 = dilated cardiomyopathy.

Cytokines activate enzymes such as gelatinase, collagenase or elastase, at the same time support fibroblast activity and lead to fibrosis associated with dilated cardiomyopathy.

The clinical picture

Diagnosis is often difficult.

- Suspicion of myocarditis always arouses unexplained heart failure, especially with a history of ongoing or previous viral fever with fever.
- The disease can have a very different course, from inconspicuous manifestations of tachycardia in febrile illnes to a severe fulminant course with circulatory collapse and shock.
- In children and adolescents with a fulminant course, we often find information about previous physical exertion during the onset of infection.

Anamnestically we find fatigue, paleness, annoyance or apathy.

- Some children may complain of abdominal pain, decreased appetite, vomiting.
- Dyspnoea and tachypnoea tend to occur even with low physical activity.
- An important symptom is muscle pain or decreased tone.
- Children often have fevers and tachycardia that do not match their body temperature and are also present during sleep.
- In the heart of severe myocarditis we hear muffled sounds and gallop.
- A systolic murmur at the tip is heard during significant mitral regurgitation.
- At the bases of the lungs, crunches can be heard, we find congestion with hepatomegaly, leaky eyelids and sometimes perimalleolar edema.
- The skin on the periphery is cold, the peripheral pulsation is less tactile.

The fulminant course is manifested by a shock condition accompanied by signs of heart failure, heart rhythm disorders (supraventricular tachycardia, ventricular extrasystoles, atrial fibrillation, AV blocks), impaired consciousness and vomiting.

- The most difficult course is in newborns and infants.
  - There is often a fulminant onset with pulmonary edema and cardiogenic shock.
- A sudden onset of severe heart failure to cardiogenic shock, pulmonary edema, major cardiomegaly, and left ventricular dysfunction are typical.
- Symptomatology develops after "normal" virosis.
- Large-cell myocarditis has a poor prognosis with progression and death within 18 months if no heart transplant is performed.

Diagnostika

Laboratory Tests

Myolysis of the heart muscle in the acute phases of the disease is confirmed by evidence of increased levels of the cardiac isoenzyme creatine kinase MB = = CK-MB mass and troponin. LDH and transaminase levels are also elevated .

Finding a viral agent is usually difficult. Possibility is a serological examination or analysis of material from endomyocardial biopsy by PCR (variant rt-PCR to detect enteroviruses).

Immunological examination evaluates the signs of activation of the organism's cellular response or subpopulation of T-lymphocytes. We assess the humoral immune response by determining the total levels of IgG, IgA and IgM and examining specific autoantibodies, such as anti-alpha-myosin. These autoantibodies usually occur secondarily.

EKG

Myocarditis is characterized by **low** ECG waveform **voltage** in all leads and **flattening or inversion of T waves.** In the left precordium, there is a noticeably small to faded Q oscillation. Sometimes depressions of the ST sections and expansion of the QRS complex are evident. Characteristic of myocarditis are **heart rhythm disorders** = satrial or ventricular extrasystoles, ventricular tachycardia, AV block (grade III AV block has been described in borreliosis, rubella, coxsackie and RS viruses). The ECG changes during the disease and therefore its dynamics must be monitored. Template:The changing ECG curve over time is typical of acute myocarditis!

Echocardiography

Echocardiographic examination shows cardiac dysfunction, which may only be regional. The thickness of the left ventricular wall may vary during repeated examinations depending on when the disease was found. Tissue edema is typical of the acute phase, which results in an increase in **left ventricular**. wall thickness . In the later stages, the left ventricle dilates. Secondary mitral regurgitation is a relatively common finding. Wall thrombican rarely be seen in the left ventricle.

Cardiac biopsies

Cardiac biopsy **is not routinely used** to determine myocarditis . The reason is the risk of the procedure and the relatively low yield of the examination. Cardiac biopsy is indicated in patients with subacute or chronic disease to distinguish it from dilated cardiomyopathy before deciding to initiate immunosuppressive therapy.

The so-called **endomyocardial biopsy** is performed, for which the so-called Dallas criteria are developed, which represent a uniform histological grading. According to histology, active myocarditis is defined by the presence of inflammatory infiltrate and necrosis. Immunocytochemical methods using monoclonal antibodies have recently been used to detect various types of lymphocytes in the myocardium.

Other methods

X-rays show non-specific cardiomegaly, at the beginning there may be a normal finding. We can also use myocardial scintigraphy using the radioisotope gallium.

Differential diagnosis

Various causes of circulatory failure can mimic acute myocarditis. Newborn heart failure can cause hypoxia, hypoglycemia, metabolic defects and severe sepsis. Myocarditis may be associated with febris rheumatica, systemic connective tissue diseases and other autoimmune diseases. Anomalous distance of the left coronary artery from the lungs is usually associated with severe circulatory failure in myocardial ischemia. Patients with endocardial fibroelastosis, Pompe disease or medionecrosis of the coronary arteries may also have symptoms similar to acute myocarditis. Many other congenital heart defects, such as aortic coarctation nebo aortic stenosis, are manifested by severe heart failure. Echocardiography reliably determines the diagnosis.

Therapy

The therapy is mostly **symptomatic**, its extent and intensity depend on the severity of the patient's condition and the complications accompanying the disease.

The goal of **acute heart failure** treatment is to maintain adequate cardiac output, ie sufficient tissue perfusion. In acute conditions with significant left ventricular dysfunction, we choose inotropic treatment, usually dopamine nor a combination of dopamine + dobutamine. Vasodilatory therapy is important, which reduces the work of the left ventricle.ECMO is indicated for severe heart failure that does not respond to conventional resuscitation therapy.

In the treatment of **chronic heart failure** in children is still the drug of choic digoxin. ZTherefore, offensive digitization is inappropriate. Treatment for heart failure includes ACE inhibitors (captopril 0.1 to 1 mg / kg for up to 8 hours) and diuretics. Captopril reduces afterload and at the same time serves as a scavenger of free oxygen radicals and thus reduces the extent of myocardial necrosis. In the chronic phases of the disease after the transition to dilated cardiomyopathy, gradually increasing doses of β-blockers (carvediol) have been used recently.

Heart rhythm disorders are a serious complication of prognosis myocarditis and must therefore be treated intensively. Amiodarone except antiarrhythmic effects of inhibiting production of TNF-α and IL-6.

Antiviral therapy for viral myocarditis (ribavirin) in acute viremia reduces viral replication in the myocardium and reduces myocardial damage. Treatment with interferon γ in patients with myocarditis and a proven adenoviral or enterovirus etiology results in improved left ventricular function. However, its effect is limiting in this treatment only in the early phase of the disease, later administration is no longer important. Some authors even warn against interferon, as it can support the body's autoimmune response. In the acute phase of the disease, a beneficial effect on the improvement of left ventricular function was observed after administration of high dose gamma globulins. Gamma globulin is administered at 2 g / kg by infusion over 24 hours.

The effect of **immunosuppressive therapy** is ambiguous. Treatment was found to have a positive effect in patients with evidence of circulating cardiac autoantibodies and in the absence of the viral genome in the myocardium. Immunosuppressive therapy (prednisone, azathioprine, cyclosporin A) is usually initiated in the subacute or chronic phase of the disease and should be reserved for the autoimmune phase of myocarditis. Administration of immunosuppression in the early stages of a viral infection may worsen the course of the disease.

Prognosis

The prognosis of myocarditis is generally **uncertain**. Mortality is high in newborns with acute myocarditis, older children have a better prognosis. Cardiac arrhythmias worsen the prognosis. It also depends on the improvement of myocardial function and the duration of its significant dysfunction. The longer the dysfunction lasts after the acute phase, the worse the long-term prognosis. Patients with fulminant myocarditis tend to have a good long-term prognosis if they overcome a period of transient cardiac dysfunction.

Kategorie:Vložené články Kategorie:Pediatrie Kategorie:Vnitřní lékařství Kategorie:Kardiologie

Pericarditis

Pericarditis (pediatrics)

Links

Source

- HAVRÁNEK, Jiří: *Heart inflammation*. (edited)

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