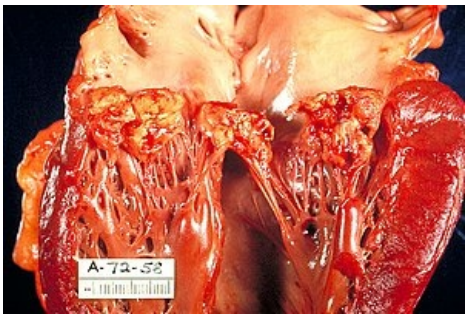
# Infectious endocarditis

***Infectious endocarditis (IE)*** is an inflammation of the endocardium characterized by the presence of **infectious vegetations** (thrombus with microorganisms) on the valve and / or wall endocardium.

## Etiology



Subacute bacterial endocarditis involving mitral valve caused by Haemophilus parainfluenzae

IE most often occurs on a primarily damaged valve or in immunocompromised patients. The causative agents may be:

- **bacteria** (viritizing streptococci, coagulase-negative staphylococci, Staphylococcus aureus, enterococci, gram-negative bacteria..., HACEK group bacteria - Haemophilus, Actiobacillus, Cardiobacterium, Eikenella, Kingella);
- **fungi** (Candida, Aspergillus);
- rarely chlamydia.

**Predispositions** are bicuspid aortic valve, pulmonary stenosis, open arterial ductus, ventricular septal defect (separate / part of Fallot's tetralogy).

**The condition for the development of infectious endocarditis** is:

- receptive terrain (= susceptible surface),
- the presence of bacteria in the blood (bacteremia), the source of which may be:
  - local purulent infection,
  - instrumental examination,
  - operation,
  - implantation of an artificial heart valve,
  - drug addiction with i.v. drug application, etc.

First, an endothelial defect develops, which is then colonized by bacteria and covered with platelets and fibrin fibres. **Infectious vegetation** is created by the multiplication of bacteria.

## Types of infective endocarditis

**Clinically**, infectious endocarditis is divided, according to pathogenesis, into infectious endocarditis:

- **on native valves** - most often in **mitral valve prolapse** and **degenerative** aortic or mitral valve defects, less often the cause is rheumatic fever and congenital heart defects,
- in intravenous drug users - the right valves (mainly tricuspid) are affected, similarly, IE can occur in the case of poorly treated central venous catheters,
- in valve replacements - similarly in pacemaker electrodes. [1]

## Classification based on the course of infectious endocarditis

Based on the course, infectious endocarditis is divided into two types:

1. **subacute** - primarily affects pre-degeneratively changed (after rheumatism) or **malformed** (bicuspid aortalis) valve or **wall of the endocardium** (atrial septal defect, tetralogy of Fallot, etc.), caused by less virulent strains (in particular streptococci, Streptococcus Viridans, Streptococcus Bovis, Enterococcus Faecalis),
2. **acute** - affects pathologically unchanged valves, caused by highly virulent strains (mainly staphylococci).

*Note: The division into acute and subacute is no longer used. The newer classifications of infectious endocarditis reflect the nature of the disease better. The newer division according to pathogenesis is as follows:* ***Infectious endocarditis on native valves, infectious endocarditis in i.v. addicts, infectious endocarditis in valve replacements.*** [2]

According to **morphology and course,** we distinguish:

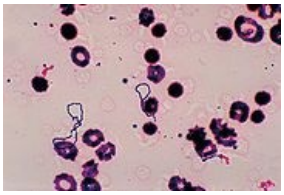
1. *endocarditis maligna acuta* (**ulcerosa**);

2. *endocarditis maligna lenta* (**polyposis**).

### Endocarditis maligna acuta (ulcerosa)

- Infectious vegetation consists of a thrombus of platelets and fibrin with bacteria, under the thrombus there is necrotic tissue with leukocyte infiltration, purulent coccal necrotic tissue, ulceration occurs.
- Infectious vegetation, necrosis and ulceration are macroscopically visible.

**Vegetation** - irregularly lobed, reddish, yellowish to a greenish, soft consistency, found mainly on the aortic (free edge or ventricular surface) and mitral (atrial surface) valve, rarely on the pulmonary, exceptionally tricuspid - in the right heart, especially in drug addicts and with a long-term catheter inserted into the right heart. It forms mainly in the spot of large pressure gradients, either just before the narrowing or directly in the place where the pathological blood flow falls.



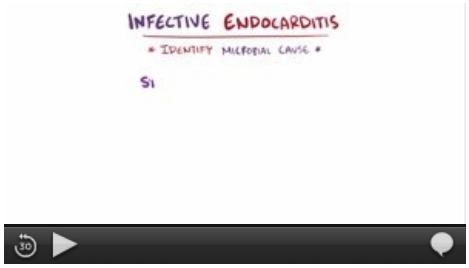
Photomicrograph of Streptococcus viridans

**Necrosis and ulceration** - they can be on the periphery of the valve (they form a "bite" of the free edge) or in its centre (valve aneurysm to its perforation), on the wall endocardium we talk about *ulcus cordis acutum*, located on the tendons leading to their rupture.

**Healing** (e.g. in antibiotic therapy) occurs by an organisation of the vegetation of granulation tissue, and the fibrous growth remains on the valve, often calcified.

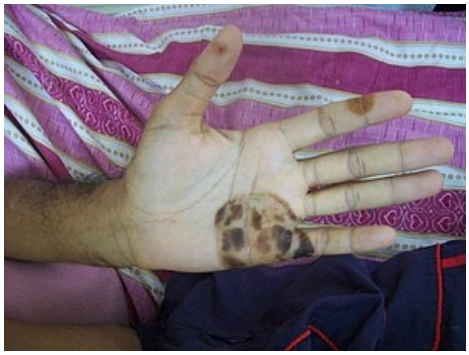
### Endocarditis lenta maligna (polyposis)

- Infectious vegetation, as in acute infectious endocarditis, consists of thrombi from platelets and fibrin with bacteria, below which there is necrosis with mild leukocyte infiltration - there is no purulent colliquation.
- Macroscopically, ulcerations are less marked, vegetation may have a polyposis shape, but there may be valve aneurysms and tendon ruptures, the mitral valve is more often affected than the aortic valve.




Video in English, definition, pathogenesis, symptoms, complications, treatment.

## Complications



Osler's lesions - immunocomplex deposits as a complication of infectious endocarditis

1. **Cardiac** (resulting in heart failure):
    1. valve damage - *insufficiency* (ulceration or perforation) or ''stenosis'' (bulky vegetation),
    2. myocardial involvement -by self- agent (transition of infection from vegetation or embolization of septic thrombi to coronary arteries - septic abscessing myocarditis) or by its toxins.
2. **Extracardiac** (resulting in mainly brain (neurological symptoms) and kidney damage (renal insufficiency)):
    1. *embolization of parts of vegetation* - leads to septic pyemia (establishment of metastatic abscesses in large (brain, spleen, kidney...) or small (lungs) circulation), in the case of bland emboli, infarcts occur (brain, kidneys, spleen, limbs, Osler's nodes in the skin),
    2. *formation of immunocomplexes* - they deposit into the walls of blood vessels, where they cause *fibrinoid necrosis* (vasculitis, glomerulonephritis, etc.).

## Screening and diagnosis

- Anamnesis,
- Objective finding,
- Laboratory examination,
- Repeated blood culture,
- Echocardiography, (Transesophageal echocardiography, TEE).

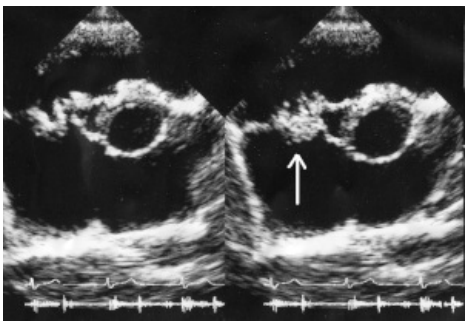
We use Durack's IE criteria to clarify the diagnosis. [3]

**Main criteria:**

- positive blood cultures - at least two;
- echocardiographic visualization of vegetation or abscess.

#### Secondary criteria:

- predisposition (valve or short-circuit defects) or i.v. drug addiction;
- fevers above 38.0 ° C;
- vascular symptoms (embolization, haemorrhage, splinter haemorrhage under the nails);
- immunological symptoms (glomerulonephritis, Osler's nodes, positive rheumatoid factor);
- one positive blood culture, or serological evidence of microorganisms.



Echocardiography - vegetation on the tricuspid valve

**Proven IE:** must meet two main, or one main and three secondaries, or five secondary criteria.

**Possible IE**: one main and one secondary, or three secondary criteria.

# Treatment

#### Pharmacological

It is difficult for ATBs to penetrate into vegetation (ATB therapy should be consulted with a microbiologist and adapted to the patient's clinical condition). Treatment usually lasts 4-6 weeks. Antibiotic therapy is terminated in patients who are afebrile for at least one week, have normal CRP levels, no new signs of embolization or new vegetation appear, and no foci are known in the body that could cause bacteremia.

CAVE !!! **Accurate identification of the microbial agent is more important than in other common infections. When IE is suspected, it is a big mistake to start ATB treatment blindly, unless it is a very critical condition. If IE is suspected after the start of treatment, it is recommended to stop ATB treatment and recruit additional blood cultures. ATB treatment must be targeted!**

#### Cardiac

As **medical prophylaxis** are used *amoxicillin + gentamicin (erythromycin)*.

Despite adequate treatment, mortality reaches tens of percent. It is always desirable to be hospitalized in a specialized workplace.

# References

### Related Articles

- Infectious endocarditis (pediatrics)
- Endocarditis
- Non-infectious endocarditis
- Congenital heart defects

### Source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2. [2]ČEŠKA, Richard. Interna. - vydání. 2015. ISBN 9788073878955.
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