Non-infectious endocarditis

Non-infectious endocarditis is an inflammation of the endocardium that is not caused by an infectious agent (bacteria or fungi).

Rheumatic endocarditis

A fibrinous interstitial inflammation that is part of rheumatic fever.

Rheumatic fever (Febris rheumatica)

Acute systemic inflammation that mainly affects children after a persistent infection with *group A β-hemolytic streptococcus*, usually pharyngitis or tonsilitis. It works on the basis of cross-reactivity of antibodies against the **M-protein** of the the streptococcal envelope with some **autoantigens**. It mainly affects the **heart** (pancarditis), **joints** (arthritis), **brain** (chorea minor), **skin and subcutaneous tissue**. The most serious is the heart disease due to the possibility of complications such as:

- **development of post-rheumatic valve defects**,
- **development of infectious endocarditis**.

The joint damage usually resolves without sequelae.

- **pericardial consequences** – pericarditis serosa or serofibrinosa,
- **myocardial consequences** – the presence of microscopic **Aschoff nodules**, especially perivascularly. They heal with small scars, most of them in the septum - might lead to problems with the conduction system and subsequent dysrhythmias (heart rhythm involvement),
- **endocardial consequences** – in the acute phase it manifests as *endocarditis verrucosa* - a formation of warty growths on the valves (especially mitral and aortic, rarely tricuspid and exceptionally pulmonary) and on the wall endocardium (especially in the left atrium - the *MacCallum field*). On the valves, these growths form on their end areas: on the atrioventicular valves, on the atrial area and on the semilunar valves, on the ventricular area.

Macroscopically the growths are reddish, stiff, size 1–3 mm, with an irregular and jagged surface.

Microscopically, **fibrinoid necrosis** of the ligament of the valve occurs. The necrosis is permeated with exudate and swells and rises above the surface of the valve. The resulting zone of Aschoff cells delimits it from one side only.

In the healing phase, exudation gives way to **proliferative changes** that lead to a thickening of the valve (on the inlet surface) and tendons, the newly formed vessels growing from the edge of the valve to its free edge. The growths are organized by the growth of non-specific granulation tissue from the base, the surface becomes leveled, smooth and overlaps with the endocardium. This causes the **valve to thicken** and later can lead to scarring, retraction and shortening of the valve. The **tendons** are thickened, shortened and sometimes fused together.

Complications

Post-rheumatic heart defects

They appear about 10 years after acute endocarditis and lead to heart failure.

- **atrioventricular valves** – shortening of tendons and valve tips - the valve is pulled to the wall of the chamber and its mobility is limited - – **insufficiency**; fusion of free edges – **stenosis** (the shape of the opening resembles a fish's mouth or a buttonhole),
- **semilunar valves** – thickening of the free edge of the valve (*entropion* or *ektropion*) – **insufficiency**; fusion at the commissures – **stenosis**,

Infection

These are mainly streptococcal infections. **Subacute infectious endocarditis** occurs.

Thrombosis

Thrombi are formed on the valve with the possibility of an **embolism** into the circulation (brain, kidneys).

Nonbacterial thrombotic endocarditis

Formerly referred to as marantic endocarditis or cachectic endocarditis, it is a valve thrombosis, that it is characterized by the formation of **sterile thrombotic vegetation on the valves** (most often the mitral valve). It resembles infectious endocarditis, but there are no signs of inflammation (edema, cellular infiltration, valve vascularization, possibly fibrinoid necrosis). It is usually present **in malignant tumors** as a paraneoplastic syndrome, most often in **adenocarcinomas** of the stomach, pancreas, bile ducts, ovaries. It may also accompany chronic thromboembolic diseases, chronic nephropathy with uremia, COPD etc. A complication is the risk of **thrombus detachment and its embolisation** into the circulation.

Libman-Sacks endocarditis

It is also known as atypical *verrucous endocarditis*. It can accompany **systemic lupus erythematosus**. It is manifested by the formation of **growths** mainly on the wall endocardium and on the ventricular surface of the valves. The growths are larger and flatter than in rheumatic endocarditis. Microscopically, fibrinoid necrosis and inflammatory cellulitis are present . We do not find Aschoff cells, but so-called hematoxylin bodies are present as a residue after LE cells (granulocytes with large basophilic inclusion as a residue of a phagocytosed nucleus).

Cardiac involvement in carcinoid syndrome

A carcinoid is a **tumor derived from cells of the diffuse neuro-endocrine system** (GIT, most often in the appendix, bronchi, islets of Langerhans , etc.). It can be solid, trabecular or solidly trabecular. Cubic to cylindrical cells are present with few mitoses and containing argentafin granules positive for diazoreaction and serotonin. It metastasizes to the lymph nodes and liver.

- Liver metastases are manifested by the development of **carcinoid syndrome** (release of serotonin into the circulation) which leads to **endocardial disruption in the right heart** .
- In the rarer location of the carcinoid in the lungs, the left heart is similarly affected.

Symptoms include **flush** (redness of the skin on the face, chest etc.), diarrhea, asthmatic asphyxiation.

- The **valves** (trikuspidalis, pulmonalis) and **wall of the endocardium** (right atrium and ventricle) are affected.

Thick layers of cellular connective tissue form, the valves are thickened or shortened,stenosis, insufficiency or both may occur. This is followed by hypertrophy and **dilatation** of the right heart, until its **failure**.

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