Angina

Angina (*acute tonsillitis*) is an acute inflammation of the tonsils, most often the palatine tonsils.

Tab. Distribution of angina

microbiological	pathological	anatomical	pathogenetic		
			separate angina (purulent)	**symptomatic angina (associated)**	**secondary angina**
bacterial	catarrhal	palatal	angina catarrhalis	angina in scarlet fever	angina agranulocytotica
viral	lacunar	retronasal	lacunaris	angina monocytotica	angina leukemica
fungal	folicular	lingual	follicularis	angina vesiculosa (herpangina)	
	vesiculosis		pseudomembranacea	angina in oropharyngeal tularemia	
	pseudomembranous		ulceromembranacea	angina gonorrhoica	
	ulceromembranous		retronasalis	diphteria	
	phlegmonous		lingualis		
	gangrenous				

Etiology and pathogenesis

Bacteria

90% of angina is caused by the pathogen *Streptococcus pyogenes* (β-hemolytic streptococcus). It is transmitted by droplets and the course is usually turbulent. Other, less common bacteria include: pneumococcus, staphylococci, Hemophilus, which are usually milder.

Human is often a carrier (the mucosa is colonized by bacteria, when the immunity decreases, the disease develops). Streptococcal cultivation is poorly established because it is often part of the physiological flora.

Viruses

Common agents include influenza, herpes viruses, coxsackie (herpangina), EBV. The infection often crosses the tonsillar tissue.

Mycosis

Rather, they accompany chronic tonsillitis, immunosuppression, diabetes mellitus and AIDS.

Angina often occurs during periods of climatic breakdown (sudden cooling) and under the influence of impaired immunity or stress.

Purulent (separate) angina

The most often on palate tonsils – *angina palatina*.

Angina lacunaris

The most common form of angina is usually caused by *Streptococcus pyogenes*. The main inflammatory process takes place in the lacquer. Fibrin is exuded, which mixes with the pus and flows from the lacuna to the surface. There he coagulates around the entrance to the lagoon, creating a yellowish-white target. The coating is only attached to the lacuna and can be peeled off. ⚠

Angina lacunaris

The highest incidence is in school children and early adulthood. Relapses are frequent.

Clinical picture and diagnostics

Common symptoms include **fever above 38 °C** (up to a degree more in children), often initial **chills**, lethargy, abdominal pain and vomiting in children. Bilateral **sore throat**, worsening when swallowed, which may radiate to the depths of the ear (common innervation n. V., IX., X.). *Foetor ex ore*, hypersalivation and painful **swelling of the nodes** in the trigonum caroticum also appear. On the tonsils, we can find targets of purulent

fibrinous exudate that extend beyond the tonsils. The soft floor and arches are movable.

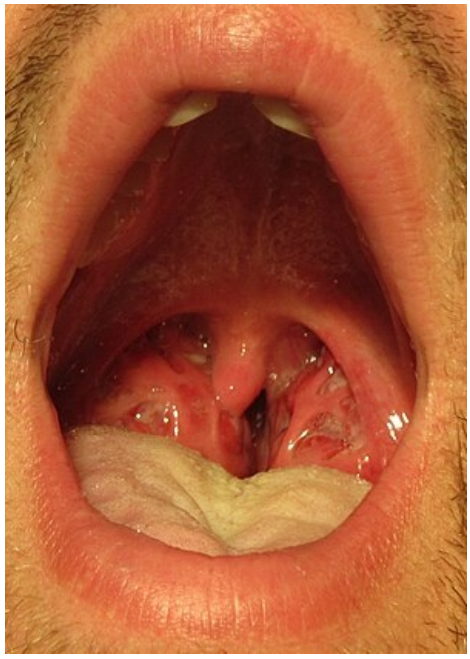
Therapy

If there are no signs of general disease, we think of *Streptococcus pyogenes*. We serve **antibiotics** (phenoxymethylpenicillin) – for 7 days, and finally depot penicillin (procaine benzylpenicillin), or 1st and 2nd generation cephalosporins, macrolides, clindamycin. ⚠

Together with antibiotics, we use **symptomatic treatment** including antipyretics, gargles, braces, liquid food and rest. At the end of the treatment, it is advisable to check the inflammatory parameters, possibly proteinuria.

Angina catarrhalis

It is a form of acute tonsillopharyngitis, which is usually part of **influenza diseases** or occurs independently as the first stage of purulent tonsillitis. It is manifested by redness and rapid tonsil enlargement up to twice in 24 hours.



Enlarged tonsils in streptococcal angina

Angina follicularis

A rarer form that can take place covertly with the lacunar form. The main pathology takes place in the follicles, which turn into small, translucent abscesses. The clinical picture and treatment are identical to lacunar angina. As the infection goes deeper, there is more scarring, which increases the risk of chronicity.

Angina pseudomembranacea

Angina pseudomembranacea, typical of diphtheria (rare today). The infection destroys not only the tonsils but also the surroundings. The surface of the tonsil with fibrin exudation forms a pseudomembrane, which cannot be wiped off as easily as the coating of lacunar sore throat. Bleeding occurs when the pseudomembrane is removed. Sometimes at this stage, lacunar angina may pass, but it does not extend beyond the tonsils (in true diphtheria the pseudomembrane exceed the edge of the tonsils).

Therapy

Prokainpenicillin, erythromycin.

Petechiae in streptococcal angina

Angina ulceromembranacea (Plaut-Vincent angina)

Secondary angina is caused by opportunistic microflora – anaerobic *Bacillus fusiformis* (fusospirosis) and *Borrelia vincenti*[1]. Analogous to ulcerative gingivitis. Today it is rare, it occurs in people with poor oral hygiene, malnutrition and vitamin deficiency.[1] (advanced alcoholics, homeless people, drug addicts)

Clinical picture and diagnostics

On one of the tonsils, there is a necrotizing process, visible as a cratered, dirty gray coated ulcer, which is usually accompanied by *foetor ex ore*[1]. Sometimes we can see a similar process on the gums. The finding may resemble a tonsil tumour or granulomatous inflammation.

Therapy

We rub tonsils with hydrogen peroxide.

Angina retronasalis

It affects children of preschool and school-age. It is a disorder of the pharyngeal tonsils, which is affected less often, which is attributed to the self-cleaning ability of the tonsils (it is covered by the respiratory epithelium with goblets and has many salivary glands in the area).

Clinical picture and diagnostics

Similar to palatal angina, the pain is felt deep behind the nose, and increases when swallowed (by rubbing the soft palate). It is usually accompanied by nasal obstruction, pathological secretion and tenderness. The pharyngeal tonsil is enlarged, reddish, sometimes with coatings. Flowing mucus from the nasopharynx causes purulent rhinitis. When the mouth of the Eustachian tube is affected, the middle ear ventilation is disturbed. It may cause transient hearing loss, inverted eardrums or catarrhal otitis media. Swollen nodules are present behind the upper third of the edge of m. sternocleidomastoideus.

Therapy

Similar to palatal angina, we give nasal drops with antibiotics.

Angina lingualis

Rare involvement of the tongue and tonsils (there is a salivary gland with a cleansing ability at the bottom of each lacuna). It occurs more often after tonsillectomy.

Angina pseudomembranacea

Symptomatic angina

Acute tonsillitis is part of the overall infectious disease[1]. These are local symptoms of a general infectious disease with bacteremia or viremia. They occur as catarrhal forms (more like pharyngotonsillitis) - in influenza, measles and poliomyelitis.

Angina in scarlet fever

The causative agent is usually β-hemolytic *Streptococcus pyogenes*, which produces Dick's erythrogenic toxin, the release of which into the bloodstream is responsible for the accompanying symptoms. It typically occurs in smaller epidemics in children's groups (mostly aged 4-12 years)[1].

Clinical picture and diagnostics

Bluetongue to lacunar angina, raspberry tongue, erythema of the face with circumoral fading (Filat's symptom). Swelling of the regional lymph nodes is usually present. General symptoms include vomiting, abdominal pain, headache, sleep rash (mainly in the lower abdomen, chest and inner limbs - embolization predilection) and small papules in the nail beds and on the auricles (Šrámek's symptom)[1].

🔎 *For more information see Angina in scarlet fever.*

Angina monocytotica

Angina in infectious mononucleosis, caused by EBV or, more rarely, CMV. In the past, the disease was predominant in adolescents, today it occurs mainly in children. In children, the incubation period is about 2 weeks, in adults even longer.

We distinguish between the **abdominal form**, in which hepatosplenomegaly is pronounced, and the **lymphonodal form**, in which symptoms on the tonsils and nodes predominate.

Clinical picture and diagnostics

Exhausting fever with morning and evening maximum, chills, sore throat and headaches. Gray-green pseudomembranes are present on the tonsils together with *foetor ex ore*. Periorbital edema (Bass phenomenon) and rash on the palate may be present. The course of the disease is highly variable. Generalized swelling of the lymph nodes is usually present, mostly on the palatal ones. We find atypical lymphocytes in the blood count. In 90 % of cases, liver tests are positive. It may resemble sepsis in angina, purulent sore throat or listeriosis.

Therapy

Mostly symptomatic, which includes relief of fever and pain. Adherence to a strict liver diet is important. In case of complications from bacterial inflammation, we give antibiotics.⚠

Angina vesiculosa (herpangina)

Summer viral disease of preschool and school children. Diseases are caused by Coxsackie group B viruses and ECHO viruses[1] (similar to HSV or VZV). It often manifests as tonsillopharyngostomatitis.

Clinical picture and diagnostics

High fever, anorexia, dysphagia, vomiting, headache, abdominal pain. On the throat, on the almonds and the arches, we find 1-2 millimetres large papules or painful blisters with a red border. It turns into circular ulcerations within 2 days. It resolves spontaneously in about 7-10 days[1].

Therapy

Symptomatic treatment including local treatment (gentian violet).

🔎 *For more information see Herpangina.*

Secondary angina

They occur during the collapse of the immune system (mainly of the cellular immunity) - acute haematological diseases such as **agranulocytosis** (*angina agranulocytotica*), **AML**, **ALL** (*angina leukemica*).[1];

It manifests as ulcerative to necrotic tonsillitis, often asymmetric, and stomatitis is also present. The absence of swollen descending lymph nodes is noticeable[1]. The condition is severe, the patient is generally prone, and there is a risk of developing sepsis. Treatment belongs to hematologists.

Therapy of tonsillopharyngitis according to etiology[2]

Etiology	1st choice treatment	2nd choice treatment
adenoviruses	symptomatic	–
EBV, CMV	symptomatic	–
St. pyogenes	penicillin V or procain-G-PEN	macrolides, lincosamides
Arcanobacterium haemolyticum	macrolides	doxycycline
Cor. diphtheriae	antitoxin + PEN G	antitoxin + macrolid
N. gonorrheae	ceftriaxon/cefepim once	ciprofloxacin once

A common mistake is the administration of ATB in viral etiology, unnecessary administration of macrolides without a positive allergological history instead of penicillin, or indication of ATB without microbiological examination.

Links

Related articles

- Differential diagnostics of coating angina
- Chronic tonsillitis
- Complications in tonsillitis

Sources

- BENEŠ, Jiří. *Studijní materiály* [online]. ©2007. [cit. 2009]. <http://jirben2.chytrak.cz/materialy/orl_jb.doc>.

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2. BENEŠ, Jiří, et al. *Infekční lékařství.* 1. edition. Galén, 2009. vol. 651. pp. 411. ISBN 978-80-7262-644-1.

Used literature

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