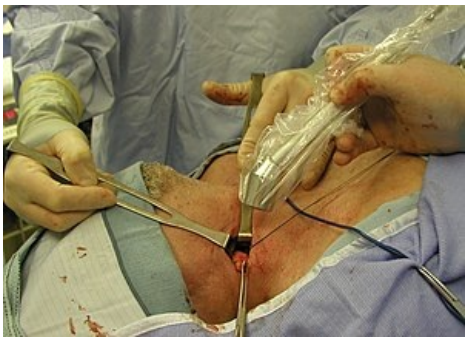
Lymphadenitis

Lymphadenitis is a response to a variety of pathogens. In acute inflammations, they are characteristically enlarged, soaked and hyperemic. Inflammation can be triggered by viruses, bacteria, fungi or protozoa.

Characteristics of lymphadenitis based on the form

Criterion	Acute form	Chronic form	Extinct form
Lymphatic folicules	Enlarged	Wiped structure	Atrophy
Germinal centres	Hyperplastic	Tattered incoherent	Extinct
Lymphocytes	Increased	Reduced	Significantly reduced
Plazmatic cells		Increased	Significantly increased



Enlarged lymph node

Viral lymphadenitis

Infectious mononunucleosis

Infectious mononucleosis is caused by the Epstein-Barr virus. It occurs mostly in **adolescents and young people**. The incubation period of the disease is about **30–40 days**[1]. Its main symptoms include, for example, fever, pharyngitis, enlargement of tonsils and cervical nodes, continuous whitish coatings on the palate tonsils. The main complications are glomerulonephritis and encephalitis.

Diagnosis consists of leukocytosis and atypical lymphocytes (from the 2nd week [1]). In some cases can occur icterus and an increase in liver function tests due to hepatitis.

Histology of nodes shows **hyperplastic follicles and enlarged germ centers**. There is an impact on the paracortical region and proliferation of immunologists. The affected cells have the appearance of a Reed-Sternberg cell.

Cytomegalovirus lymphadenitis

The disease is caused by cytomegalovirus from the herpesvirus family . In nucleuses, we find typical inclusions. A light strip appears around the virion. Most often, the disease breaks out in **newborns or immunudeficitated individuals (AIDS)**.In patients with imuodeficiency, it is manifested by severe and sometimes life-threatening inflammation.

Histology of nodes shows hyperplastic follicles and enlargement of the paracortical region. Immunoblasts are grouped into stripes. Again, the cells have the appearance of a Reed-Sternberg cell.

Lymphadenitis provoked by the herpes simplex virus

Lymphadenitis provoked by the herpes simplex virus exists of two types. **Type HSV-1** is manifested by gingivostomatitis, keratoconjunctivitis and esophagitis. The second type is **the HSV-2 type**, when inflammations arise in the genital area and anus.

As far as histology is concerned, an increase in the paracortex is observable. This will cause compression of the follicles and subsequent atrophy. Deposit-necrotic deposits and intranucleolar inclusions with halo phenomena are formed. Occasionally, there are also Reed-Sternberg cells.

HIV-induced lymphadenitis

Hiv virus has a strong tropism to CD4-lymphocytes, monocytes and dendritic cells. The cause of this phenomenon is the protein gp120, which is located on the surface of lymphocytes. From the histological point of view, three histological images corresponding to the clinical condition are described - acute, chronic and "extinct".

Clinical Picture A is characterized by irregularly enlarged follicles and increased mitotic activity of cells. The interfollicular regions are bleeding.

In **picture B**, the structure of the follicles is wiped off and lymphatic vessels proliferate in them. The amount of lymphacytes is reduced.

The nodules are atrophic in **picture C**, which is also typical for reduced to disappeared follicles. We observe pronounced vascularization and the presence of a large number of plasma cells. Later, the nodule may fibrotize or hyalinize.

Bacterial lymphadenitis

In bacterial lymphadenitis, infection causes acute non-specific or chronic granulomatous inflammation on an immune basis. There is damage to the nodes, which is due to a combination of necrotic, proliferative and sclerosing processes.

Purulent lymphadenitis

Common bacteria (Staphylococci or Streptococci) affect regional nodes during inflammation. In particular, these are superficial nodules, such as cervical, axillary and inguinal nodes. The range of inflammatory changes can have very variable magnitude - from focal abscesses to the destruction of the node by an inflammatory process.

Lymphadenitis in the "cat scratch" disease

The disease is provoked by the bacteria *Bartonella henselae*. From a histological point of view, follicular hyperplasia occurs in the early stages. Focal necrosis occurs first in the affected cortex, then the pulp. Necrosis leads to infiltration of neutrophils and the subsequent accumulation of macrophages around the abscesses.

Lymphadenitis in lymphogranuloma venerum

This type is provoked by the bacterium *Chlamydia trachomatis*. Already a week after infection, the regional nodes are enlarged. In the early stages, it is manifested by follicular hyperplasia. Similar to the disease of the "cat scratch", focal necrosis is formed, which leads to the infiltration of neutrophils and the subsequent accumulation of macrophages around the abscesses. In some cases, the disease can manifest itself even with superficial skin fistulas.

Syphilitic lymphadenitis

Syphilitic lymphadentitis is provoked by the spirochete *Treponema pallidum*. During the first stage of the disease, the ininguinal nodes enlarge. 5-6 weeks[1] after the formation of a hard ulcer, generalized lymphadenitis occurs. The follicles are hyperplastic and have a bizarre shape. Secondary and tertiary follicles are also often present. The bush is ligamentous thickened. T-areas of the nodule are enlarged. Plasma cells in the marrow proliferate. In addition, separate epithelioid granulomas can be observed.

Tuberculous lymphadenitis

This type is provoked by the rod *Mycobacterium tuberculosis*. In this case, inflammation can have the character of caseous fibroproductive lymphadenitis or miliary tuberculosis.

Non-tuberculous mycobacterial lymphadenitis

As the name suggests, non-tuberculous mycobacterial lymphadenitis is provoked by the *Mycobacterium avium-intracellulare*. In this type of disease, the nodules are enlarged, but they have a wiped out architecture. Caseous necrosis are not present. The normal structure is permeated with stripes of large pale histiocytes.

Lymphadenitis in leprosy

Another mycobacteria, this time *Mycobacterium leprae*, causes lymphadenitis in leprosy. There are two forms of this disease. The first form is the **lepromatous** form , when the nodules are enlarged and in the paracortical region many foamy macrophages can be found. The second form is called **tuberculoid**. In this case the nodules are small and have inconspicuous germ centers. Granulomas are present.

Lymphadenitis in Whipple's disease

Lymphadenitis in Whipple's disease is caused by the bacterium *Tropheryma whipplei*. The main symptoms include fever, diarrhea, weight loss, joint pain and damage of CNS. In 90% of cases among these symptoms belongs even malnutrition[1][1]. From a histological point of view, the nodules are enlarged. Foamy macrophages occur. And sometimes even a granulomatous reaction can be present.

Lymphadenitis of a different etiology

Cryptococtic lymphadenitis

The disease is caused by a mycoorganism of yeast type, *Cryptococcus neoformans*. Infection can be passed through contaminated soil. This dieases is widespread worldwide. Most often it affects the lungs, brain and skin. Numerous non-assaying granulomas can be found in the histological sample.

Histoplasmic lymphadenitis

The pathogenic fungus *Histoplasma capsulatum* is the causative agent of histoplasmic lymphadenitis. Territorially, it occurs endemically in the USA. It mainly affects immunosupressed patients. The main manifestations include fever, weight loss and other nonspecific symptoms. Form the histological view: granulomas appear, microbes that are in the macrophage do not perish, on the contrary, they multiply in it. After sufficient multiplication, the macrophage perishes.

Toxoplasma lymphadenitis

The protozoa *Toxoplasma gondii* is the causer of toxoplasma lymphadenitis. Contact with an infected cat or eating uncooked meat is enough for transmission. The infection is mostly asymptomatic. Follicles are hyperplastic and germ centers are enlarged. Groups of scattered epithelioid cells are formed, which do not form granulomatous deposits.

References

1. POVÝŠIL, Ctibor – ŠTEINER, Ivo – BARTONÍČEK, Jan, et al. *Speciální patologie.* 2. edition. Praha : Galén, 2007. 430 pp. ISBN 978-807262-494-2.

Literature

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