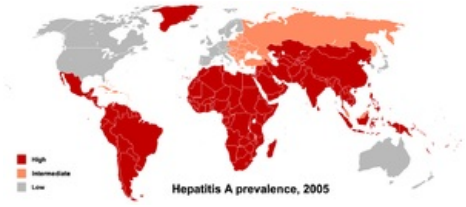
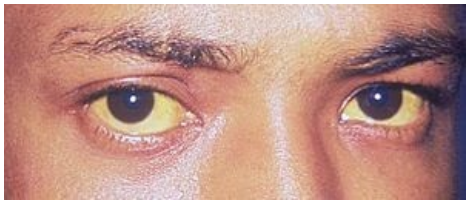
Hepatite A

Template:Infobox - virus





The causative agent is HAV, which is RNA virus from the family "Picornaviridae" ("Enteroviridae", Enterovirus 72), which acts directly cytolytically. HAV is a small virus (27–30 nm), genetically homogeneous, resistant to the external environment. It is an exclusively human pathogen. [1] It spreads by the fecal-oral route ("dirty hand disease"). Often contaminated with food and water, rarely parenterally. The entrance gate is the digestive tract, excreted in the faeces. Transplacental transmission is not possible. [2] The virus is highly resistant to external influences. It is excreted in the faeces as early as 2 weeks before the onset of symptoms and for about a week (up to 2 weeks) [2] after completion, the patient is most contagious before the end of the incubation.

Course of infection

Incubation period is 15-48 days. This is followed by a symptomatic stage ("icteric") - the leading symptom is icterus, dark urine and acholic stool. The course is milder and shorter than with VHB. Cholestatic symptoms are rare and may occur fulminantly. HAV does not cause chronic infections. [1]

Diagnostics

Detection of antibodies

We detect with anti-HAV antibodies. Examination of specific antibodies IgM in serum (anti-HAV-IgM), increase in transaminases and bilirubin u, slightly increase ALP. Negativity of the test in immunocompetent individuals excludes infection. IgM persists in serum 3-6 months after infection, IgG persists for a long time. The infection leaves a long-term to lifelong immunity. The main diagnostic marker.

Electron microscopic detection of stool virus [1] It can be detected in the second half of the incubation period and shortly after the onset of clinical symptoms.

Antigen and RNA detection [1] In stool, similar to microscopy.

Treatment

Treatment is symptomatic - peace of mind, no alcohol, diet with carbohydrates (possibly infusion glucose) and fat reduction. Corticosteroids only for fulminant forms.

Complications

The severity of the infection increases with age (90% are asymptomatic in young children). In 10%, it is a prolonged form, which, however, does not lead to chronicity. Chronic infection and carriers do not exist.

Complications: fulminant liver failure (rare), myocarditis, encephalopathy, cryoglobulinemia, bone marrow hypoplasia, spleen rupture, pancreatitis, Guillain-Barré syndrome. [2]

Prevention

Vaccination with an attenuated vaccine, increased health surveillance at the site of the outbreak. New infections are sought through clinical examination and liver function. The exposed is administered prophylactically immunoglobulin (NORGA).

Links

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