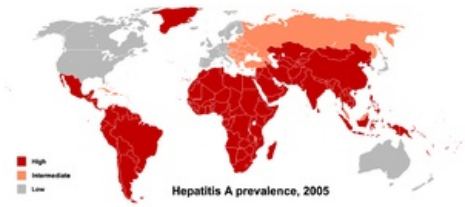
Hepatitis A

The causative agent is HAV, which is an RNA virus from the Picornaviridae family (Enteroviridae, Enterovirus 72), that has a direct cytolytic effect. HAV is a small virus (27-30nm), genetically homogenous, resistant to the external environment. It is an exclusively human pathogen.[1] It spreads by the faecal-oral route (“dirty hand disease”), often by contaminated food and water, rarely parenterally. The entrance gate is the digestive tract and it is excreted in faeces. Transplacental transmission is not possible.[2]

The virus is highly resistant to external influences. It is excreted in faeces as early as 2 weeks before the onset of symptoms and continues for about a week (up to 2 weeks)[2] after the symptoms stop. The patient is the most contagious before the end of the incubation period.

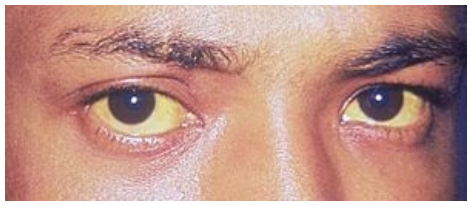
The course of infection



The global prevalence of hepatitis A

The incubation period is 15-48 days.[2] First, the prodromal (“preicteric”) stage begins (dyspepsia, fatigue, fever, weight loss). This is followed by the symptomatic stage (“icteric”) - the leading symptoms are jaundice, dark urine and acholic stool. The course is milder and shorter than in VHB. Cholestatic symptoms are rare and may occur fulminantly. HAV does not cause chronic infections.[1]

Diagnostics


Jaundice: yellowing of the sclera in a patient with viral hepatitis A

Detection of antibodies

Diagnostics is done using anti-HAV antibodies. We perform **the examination of specific IgM in serum** (anti-HAV-IgM), the increase in transaminases and bilirubin and the slight increase in ALP. Negative test in immunocompetent individuals excludes infection. IgM persists in serum for 3-6 months after infection, IgG persists long-term. The infection leaves a long-term to lifelong immunity. The main diagnostic marker.

Electron-microscopic detection of virus in faeces [1]

It can be detected in the second half of the incubation period and shortly after the onset of clinical symptoms.

Detection of antigen and RNA [1]

In stool, similar to microscopy.

Therapy

Treatment is symptomatic - rest, no alcohol, a diet with carbohydrates (possibly glucose) and fat reduction. Corticosteroids only in 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. By clinical examination and liver function monitoring, new cases of infections are identified. Immunolactively administered immunoglobulin (NORGA) is administered to the exposed.

Links

Related articles

- Viral hepatitis
- Jaundice
- Jaundice (icterus)

References

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2. MUNTAU, Ania Carolina. *Pediatrie.* 4. edition. Prague : Grada, 2009. pp. 393-394. ISBN 978-80-247-2525-3.

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