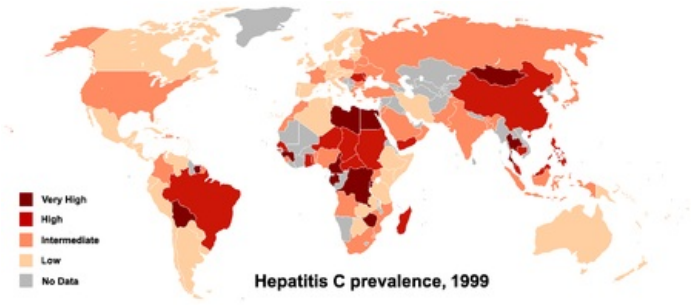
Hepatitis C

The causative agent is the HCV-RNA virus (*Flaviviridae*). We differentiate its genotypes and their subtypes, the virus is easily subject to mutations. There are 6 types of viruses and a large number of subtypes (genotype 1b occurs most often in the Czech Republic). Spread parenterally (blood derivatives – haemophiliacs, i.v. drug addiction, hemodialysis, sexual transmission, perinatally from mother to fetus, transplant grafts), it is about 100 times less contagious than VH

Acute HCV has an ICD code of B171, chronic B182.



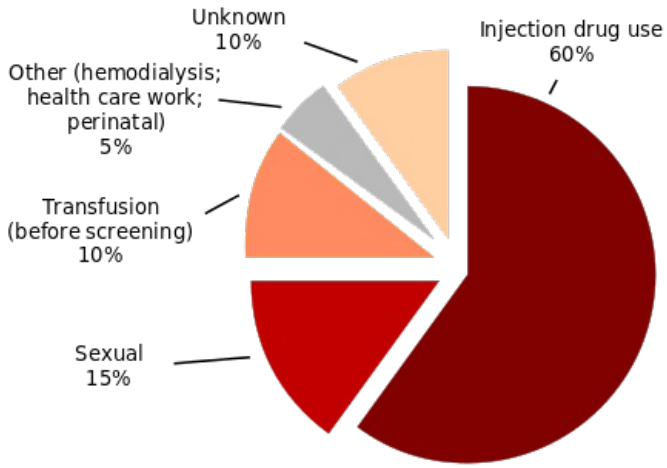
Global prevalence of hepatitis C

The course of infection

The incubation period is 15–160 (most often around 50) days[1].

1. **Acute infection** (asymptomatic or icteric form), in 15% spontaneous elimination, in 85–90% it becomes chronic;
2. **chronic infection**.

The infection is often asymptomatic, or it takes the form of vague dyspeptic problems. It is **usually without jaundice**. It may manifest itself clinically after years as liver cirrhosis (or its complications) or hepatocellular carcinoma. Jaundice occurs more often in the elderly. Hepatic failure is rare. The development of liver cirrhosis is slow, accelerated by HBV and alcohol. Asymptomatic carriers are rare.



Sources of hepatitis C infection

Diagnostics

We determine serologically - **anti-HCV** antibodies (not only in infected persons but also in those who eliminated the virus spontaneously or by antiviral treatment). PCR detection of **viral RNA** is an indicator of active infection. The rise in Anti-HCV is seen about 3 weeks after exposure, it has no preventive effect against reinfection. It is characterized by a small link between biochemistry and histology (even slightly increased ALT, large changes). For this reason, we have to resort to liver biopsy more often.

Ig production may be delayed; if acute HCV is suspected, the test should be repeated. Chronic hepatitis C requires a biopsy with staging (advanced liver fibrosis) to show a risk of progression to liver cirrhosis. The virus cannot be grown on tissue cultures, so **PCR or Ig anti-HCV is detected.**

Treatment

At the moment (2020) the treatment consists of **directly acting antivirals** (**DAA**) in interferon-free mode, and often in ribavirin-free mode. This prevents serious side effects of pegylated interferon (PEG-IF alpha) and ribavirin.

Genotyping of the virus is important before starting.

Treatment has **minimal** side effects.

Efficiency is close to **100 %**.

Drugs block the multiplication of the virus in the hepatocyte. According to the place of operation, we divide them into 3 groups of antivirals :

1. viral protease blockers, cleaving viral proteins
2. NS5A replication complex inhibitors (assembling viral particles)
3. viral polymerase inhibitors

Efficacy is assessed in **12 and 24 weeks** after the end of therapy.

2 Negative HCV PCR tests performed 12 weeks apart indicate the elimination of the virus from the body (the patient achieved a so-called sustained virological response).

DAA: Directly Acting Antivirals (Most Important Fixed Combinations)

Genotype specific:

Sofosbuvir (400 mg) + **ledipasvir** (90 mg): 1-0-0, standard 12 weeks. Treatment for genotypes 1 and 4.

Grazoprevir (100 mg) + **elbasvir** (50 mg): 1-0-0, standard 12 weeks. Treatment intended for genotypes 1 and 4 (in the Czech Republic only for 1a and 1b with low viremia).

Pangenotypes:

Sofosbuvir (400 mg) + **velpatasvir** (100 mg): 1-0-0, standard 12 weeks. Treatment for HCV genotypes (1-6).

Glecaprevir (100 mg) + **pibrentasvir** (40 mg): 3-0-0, standard 8-12 / 16 weeks. Treatment for HCV genotypes (1-6).

Sofosbuvir (400 mg) + **velpatasvir** (100 mg) + **voxilaprevir** (100 mg): 1-0-0, standard 8-12 weeks. Treatment for HCV genotypes (1-6).

This treatment may or may not be combined with **Ribavirin** (according to the genotype of the virus, severity: liver cirrhosis) and the therapy lasts for a maximum of **12** weeks. The side effects are quite mild but the treatment is very expensive (the price is still falling).

Unlike previous therapy, there is a success rate of up to 97%.

The ultimum refugium is then a liver transplant.

Prophylaxis

There is no effective vaccine against HCV (due to the high variability of the virus). Reduction of the risk of infection: in health care, in high-risk populations (MSM).

Screening examinations of at-risk individuals.

Links

Related articles

- Viral hepatitis
- Jaundice
- Jaundice (icterus)

Sources

- PASTOR, Jan. *Langenbeck's medical web page* [online]. [cit. 2010]. <http://langenbeck.webs.com>.

- BENEŠ, Jiří. *Studijní materiály* [online]. [cit. 2010]. <http://jirben.wz.cz>.

External links

- Hepatitis C (Czech wikipedia)
- https://www.tribune.cz/clanek/45676-terapie-chronicke-hepatitidy-c-v-roce
- https://www.who.int/news-room/fact-sheets/detail/hepatitis-c
- https://www.infekce.cz/DoporVHC17.htm
- https://www.ikem.cz/cs/hepatitida-c/a-3619/
- https://www.internimedicina.cz/pdfs/int/2016/05/03.pdf

References

1. Doporučené postupy pro praktické lékaře. *Virové hepatitidy*. 2001. reg. č. o/020/016. Autoři: Stanislav PLÍŠEK a GALSKÝ Jan. Available from <http://www.cls.cz/dokumenty2/postupy/r016.rtf>.

Literature used

- HAVLÍK, Jiří, et al. *Infektologie.* 2. edition. Prague : Avicenum, 1990. 393 pp. ISBN 80-201-0062-8.

- LOBOVSKÁ, Alena. *Infekční nemoci.* 1. edition. Prague : Karolinum, 2001. 263 pp. ISBN 80-246-0116-8.