Acute Viral Hepatitis

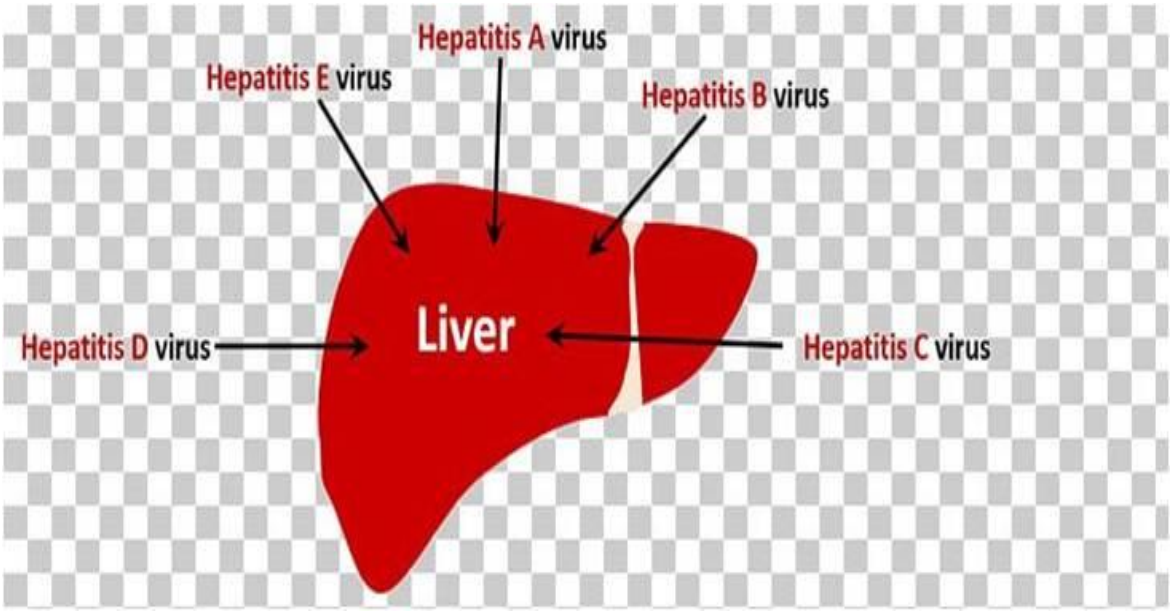
in children

- Acute viral hepatitis is a human-specific infectious disease, a primary infection of the liver, caused by several hepatotropic viruses (A, E, B, C, D, F, G; TTV), clinically manifested by signs of general, digestive and hepatic changes, associated or not with jaundice.
- Acute viral hepatitis is a major public health problem for the following reasons:
- - increased and persistent morbidity, involving children in 55-65% of cases;
- - important potential for chronicity, especially HVC, HVD, HVB.

➢ **With the advent of the B virus vaccine, the incidence of hepatitis B and D has been greatly reduced in children.**

Acute Viral hepatitis

- **Systemic infection which causes inflammation of the liver**
- **Currently 5 recognized types of viral hepatitis: A, E**
- **B, C, D,** F,G,TTV**. All viruses cause similar illness, but have distinct antigenic properties**



Classification of acute viral hepatitis in children:

A. According to the etiology: A, E, B, C, D, F, G, TTV.

B. Depending on the type: 1) typical (jaundice); 2) atypical (anicteric, attenuated, inapparent);

C. Depending on the severity: 1) mild; 2) moderate, 3) severe, 4) malignant;

D. Depending on the duration of the disease:

1) acute (3 months);

2) trenante (3-6 months);

3) chronic (over 6 months).

E. Depending on the evolution:

1) benign, cyclic;

2) with complications: hepatobiliary, intercurrent diseases;

3) with exacerbations and recurrences.

General features of viral hepatitis

Type of Hepatitis

	A	B	C	D	E
Source of virus	feces	blood/ blood-derived body fluids	blood/ blood-derived body fluids	blood/ blood-derived body fluids	feces
Route of transmission	fecal-oral	percutaneous permucosal	percutaneous permucosal	percutaneous permucosal	fecal-oral
Chronic infection	no	yes	yes	yes	no
Prevention	pre/post-exposure immunization	pre/post-exposure immunization	blood donor screening; risk behavior modification	pre/post-exposure immunization; risk behavior modification	ensure safe drinking water

Acute viral Hepatitis

More frequent signs

- Malaise
- Low appetite
- Dark urine
- Nausea
- Abdominal pain
- Scleral icterus
- Vomiting
- Hepatomegaly
- Splenomegaly
- Respiratory symptoms
- Headache
- Fever
- Muscle pain
- Rash
- Joint pain
- Itching

Hepatitis A

is an acute infectious disease with a benign prognosis, without chronic forms, more often in childhood.

Etiology

- ARN-virus
- Picornaviridae family
- Hepatovirus genus
- There is a single viral serotype and a unique antigenic determinant (AgHAV).
- Recently, 7 viral genotypes were identified, with small differences between them (10-25%), but with importance in the development of vaccines.
- HAV is heat resistant, can persist for days, months in contaminated water, is destroyed by boiling at 100 C in 5 minutes, by autoclaving - in one minute.
- Anti-HAV IgM – relates an acute infection
- Anti-HAV IgG – relates an post-infectious immunity or an immunity after vaccination

EPIDEMIOLOGY.

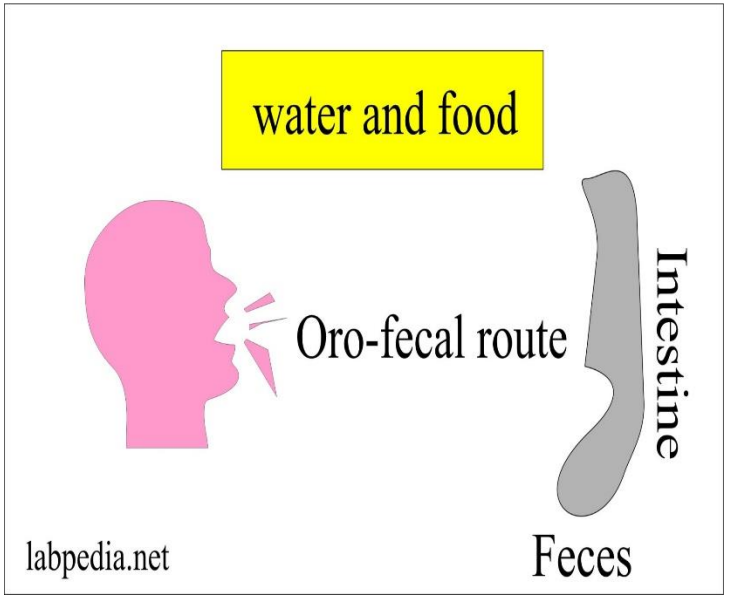
- Viral hepatitis A is the most common etiological form (> 60%) of viral hepatitis. HVA dominates in childhood, where it is responsible for 70-75% of all cases.

Epidemiology

- Source - **sick human** whith diferent forms of deseases, very dangerous are the atypical forms.
- The virus excretion starts in the last days of the incubation period and lasts the whole preicteric period, and the first week of the icteric period.
- VHA is a **typical intestinal infection**
- The highest morbidity is registered in **autumn** and **winter**.

Hepatitis A: Transmission

- Transmission of the infection takes place by fecal-oral route: by direct contact, by ingestion of contaminated water or food, but also indirectly by using recently contaminated objects.
- Transmission of the infection parenterally is possible, but has not been proven.



Receptivity to HVA is general. The disease may have an endemic and / or epidemic character with autumn-winter seasonality. The highest degree of morbidity is in children.

- Inapparent infections or undiagnosed anicteric hepatitis predominate in the young child.
- So; protective antibodies were detected in about 92% of people over 50 years of age.
- The prevalence of HAV is lower in industrialized countries.
- However, its frequency is high, being related to hygiene conditions and the socio-economic environment.

The specific immunity after the disease is lasting maybe even for a lifetime, and the post-vaccine one up to 10 years and more.

- Anti-HAV IgM antibodies appear early in the course of infection and disappear within 3-4 months of onset, and anti-HAV IgG persists throughout life.

PATHOGENY.

- Hepatocytes are the target cells for HAV. After entering the body through the digestive tract, HAV can be seen in the nasopharynx and intestine where several histomorphological changes occur due to its multiplication in epithelial cells in the crypts of the small intestine.
- From this level, through the portal vein, it invades the liver, where replication takes place. Hepatocyte lesions (degeneration, necrosis) are marked by a direct cytopathic effect and inflammatory infiltration of the liver stroma.
- Liver damage may progress clinically or subclinically.
- Inapparent (subclinical) forms are found in ten to one (10:1) to those clinically manifested.

- As a result of the direct lesion of the hepatocytes, there is a decrease in the blood of the substances synthesized by the liver (albumin, coagulation factors), the detoxification function of the liver is reduced.
- Liver cell damage (cytolysis) is expressed by increased enzymes in the blood (ALT, AST, aldolase and a).
- In HVA, foci of hepatocyte necrosis are predominantly limited periportally, usually no acute liver failure develops.
- Viral hepatitis A is characterized by complete healing within a few weeks.
- This is due to the intense immune process in HAV with fast seroconversion from the beginning of the disease, which stops the processes of cytolysis and liver necrobiosis.

Clinical periods

- **Incubation period -** ***is 10-15 days (in average 5-50 days)***
- **Prodromal period (preicteric) -**
- ***(3-10 days)***
- **Icteric period -** ***(1,5-2 weeks)***
- **Convalescence period (reparative, post-icteric)**

Incubation period (15-50 days)

- It has no clinical manifestation
- At the end of the incubation period there is a higher activity of cellular enzymes (ALT, AST)
- The HAV is detected in the last third part of the incubation period.

The prodromal (preicteric) period in children is 3-8 days, with variations between 1-10 days. In about 5% of cases this period may be missing.

- The onset is acute with fever 38-39 ° C (50-80% of cases), headache, asthenia, loss of appetite, nausea, vomiting.
- Epigastric pain often occurs, pain in the right hypochondrium (by relaxing the liver capsule).
- In some children, the pain may radiate to the right lower abdomen, or to the kidneys.
- It is marked by flatulence, constipation, rarely diarrhea.
- 1-2 days before the onset of jaundice, urinary hyperchromia, alcoholic stools are noticed.
- Digestive syndrome occurs in over 70% of children.
- The "pseudo-influenza" syndrome with altered general condition, fever, discrete respiratory manifestations, coryza, pharyngeal hyperemia is observed in 2-4% of cases.

Physical examination causes painful, sensitive hepatomegaly, more common in children than in adults, less often - splenomegaly.

The laboratory examination indicates:

- ✓ liver cytolysis tests (ALT, AST, LDH) much higher than normal.
- ✓ the thymol test is increased from the first days of illness.
- ✓ Although total bilirubin remains normal, the conjugated fraction often occurs.
- Sometimes HVA can start with a clinical picture of an acute abdomen, simulating acute appendicitis, or food poisoning.

Icteric period.

- ✓ Jaundice usually occurs on the 3rd to 5th day of the disease.
- ✓ Simultaneously with the appearance of jaundice, the fever decreases, the patient's condition improves, the toxic manifestations from the prodromal period disappear.
- ✓ The appetite returns and the child feels better.
- ✓ Jaundice is more obvious, it progresses.
- ✓ Depression, bradycardia, and pruritus, commonly seen in adults, are less common in children.
- ✓ In a few days (2-3), jaundice intensifies from subicterus to frank and intense jaundice.
- ✓ In young children, in most cases, jaundice is moderate or discreet.

- Urine during jaundice darkens even more in color ("black beer").
- The chairs become acholic.
- The duration of jaundice is variable - from a few days to 1 month, on average 7-14 days (shorter than in adults), is short in mild forms and can last more than a month in severe and cholestatic forms.
- There is a correlation between the intensity of jaundice and the severity of hepatitis, but jaundice does not always correspond to the degree of severity.
- Hepatomegaly is recorded in 96-100% of cases, in children more pronounced than in adults.
- The liver is consistent and sensitive to palpation.
- Hepatomegaly persists and after the acute period of the disease, the left hepatic lobe is enlarged and sensitive, persisting longer.
- The spleen is rarely palpable, being more common in children than in adults.

- There is no correlation between the degree of hepatosplenomegaly and the intensity of jaundice, as well as the severity of the disease.
- Impairment of the bile ducts of the pancreas is uncommon in young children.
- Cardiovascular system. Bradycardia, hypotension, myocarditis, are rarely seen in children with HAV.
- The kidneys are rarely affected (oliguria, albuminuria, hematuria and discrete leukocyturia).

The period of convalescence

Is characterized by:

- the absence of complaints,
- the disappearance of jaundice,
- normalization of urine and stools,
- maintaining a moderate hepatomegaly.
- The patient feels better.
- In some cases the enlargement of the liver is retained and the liver functional activity is gradually improving.
- Fatigability, asthenovegetative disorders can remain.
- Liver tests return to normal.
- Clinical normalization is earlier than biochemical and histological.
- The duration of this period is 2-3 months, rarely 4-6 months from the beginning of the disease.

Atypical clinical forms

- anicteric,
- attenuated
- subclinical form.

The anicteric form

- Has no icteric skin and sclerae.
- There is a short-time temperature rise, hyporexia, nausea, weakness, abdominal pain, liver enlargement.
- The color of urine and feces may change.
- There is a higher activity of ferments (ALT, AST) and thymol test in blood serum. The bilirubin level is normal.
- The duration of changes are **3-7 days**.

The attenuated form

- subfebrile temperature,
- transitory short-time jaundice (2-3 days),
- dark urine and acholic stool. The attenuated form is termed as “rudimentary” one being a variant of the mild type of the desease.

The subclinical form

- this form has no clinical manifestations,
- but there is higher activity of enzymes (ALT, AST) and IgM antibodies in blood serum.
- the subclinical forms occur more frequently within the VHA outbreak and often are not clinically diagnosed, hence leading to the epidemic process continuation.

DIAGNOSTIC.

- The clinical diagnosis is only presumptive, suggested by the age of the child, the absence of a history of HVA, contact or outbreak data, digestive onset and benign course of the disease.

Laboratory examinations:

- In blood examinations there is leukopenia, lymphocytosis and eosinophilia, while ESR is normal.
- In the biochemical tests:
- high level of ALT, AST
- high bilirubin level from conjugated fraction account
- thymol test increased
- Anti-HAV IgM – relates an acute infection
- Anti-HAV IgG – relates an post-infectious immunity or an immunity after vaccination

Differential diagnosis

- ARVI
- Acute enteric infections
- Acute surgical diseases of abdominal cavity (appendicitis and others)
- Suprahepatic jaundice (hemolytic jaundice)
- Different variants of hepatitis in infectious diseases (yersiniosis, infectious mononucleosis, herpetic infection, salmonellosis)

Hepatobiliary complications (cholecystitis, cholangitis, bile duct dyskinesia) in HAV occur following the activation of endogenous microbial flora, often against the background of bile duct abnormalities.

- Very rarely occur - thrombocytopenia, agranulocytosis.

Viral Hepatitis B (HBV)

- Viral hepatitis B is one of the most important infectious diseases in the world. According to specialized publications, it is estimated that 40% of the world's population has been infected with the hepatitis B virus, and 350 million are patients with chronic viral hepatitis B. This disease has a significant medical and social importance, given the severity of the disease, chronic , disability and primary development of liver cancer.
- Remarkable successes in preventing this disease have been made in recent decades. With the introduction of HBV vaccination and disposable medical instruments, the number of cases of acute HBV has decreased significantly. Vaccination of newborns during 26 years in the Republic of Moldova has contributed to reducing the morbidity of children aged 7-14 years from 62 -58 0/0000 (in the last years of the prevaccination period) to 0.12-0.0170 / 0000 (single cases) in the last years of the vaccination period.

Hepatitis B

“serum hepatitis, post-transfusion hepatitis”

- **Double shelled DNA hepadnavirus**
- **Spread by sex, blood, and body fluids**
- **Severe disease**
- **Prolonged illness**
- **Chronic problems in ~ 10% - 20%**
- **consists of three antigens:**
- **HbsAg - “Australian”,**
- **HbcorAg - nuclear,**
- **HbeAg – infectivity.**

HVB transmission routes:

a) **natural:**

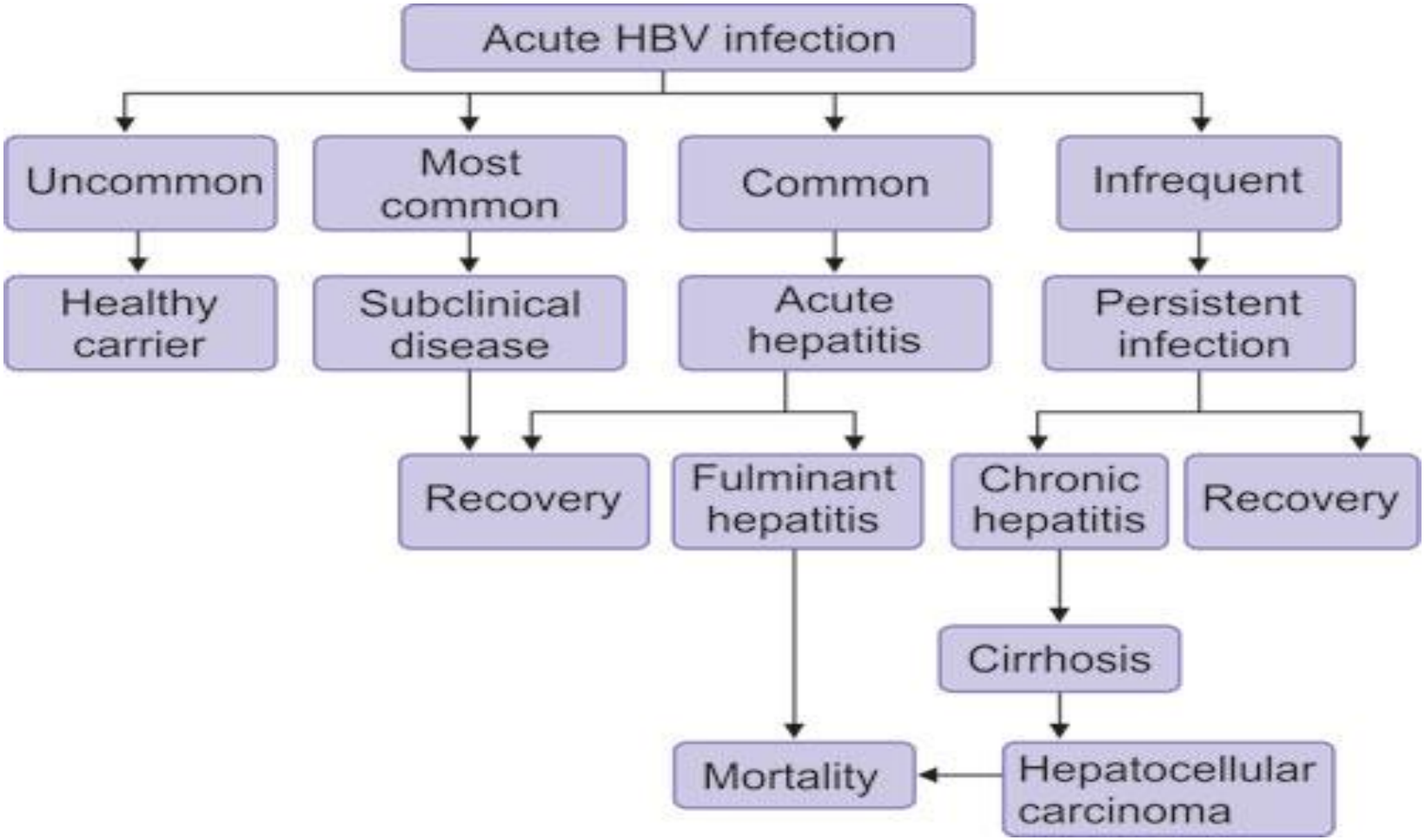
- ***maternal-fetal***: intrauterine (in case of possible acute HBV (HBsAg and HBe-positive) in the last 3 months of pregnancy or the presence of chronic viral active hepatitis in pregnant women) or intranatal- in newborns.

Transmission of HBV from the infected mother to the child in about 95% of cases occurs in labor and only 5-10% of newborns become infected antenatal. The presence of HBsAg in pregnant women is a factor favoring maternal-fetal transmission;

- ***postnatal HBV*** transmission through breast milk is problematic. HBV infection from mother to infant can be transmitted through the blood in the nipple fissures, if the child has lesions of the oral mucosa. The risk of transmitting the infection from mother to child is related to the rate of HBV replication in the mother;

- ***through regular contact with infected people***, through objects of use, dishes.

Evolution of HBV



Periods of the disease

- **Incubative period-(45 – 180 days)**
- **Prodromal period (preicteric)-(1-2 weeks)**
- **Icteric period-(3-4 weeks)**
- **Convalescence period-(reparative, post-icteric)-(2-4-6 months)**

Clinical manifestations

- The disease progresses in stages: there appears weakness, malasiae, patients easily get tired, their workability is low; there is loss of appetite, often the temperature rises.
- Often, these signs are weakly manifested and the disease beginning is manifested with dark urine and jaundice. From the first days some patients have nausea, recurrent vomiting, frequent dyspeptic symptoms.

- The **icteric phase** manifests itself with an intensive jaundice, hepatolienal syndrome (lasting to 30 days), hemorrhages and complications development due to the concomitants bacterial infection.
- The most **severe degree** of the VHB, especially in the infants younger than 12 months, is the **malignant form.**

Viral hepatitis B in infants

- The disease often has an acute onset with a fever of 38-39 ° C.
- The prodromal period is short (3-5 days), manifested by digestive disorders (refusal of food, vomiting, regurgitation, fluid stools).
- The jaundice period is long lasting (up to 3-6 weeks and more). Hepatosplenomegaly is more pronounced compared to older children and persists for a long time.
- Severe and moderate severity forms 2/3 of the cases. Evolution towards chronicity, more frequent cirrhosis of the liver. Bacterial and viral superinfections are common and negatively influence the evolution of HBV.

Congenital viral hepatitis B

- It is caused by hepatocellular disorders with or without jaundice, which are present in newborns in the first days, weeks after birth (up to 2-3 months).
- Fetal HBV often begins with jaundice in the first days of life, progresses slightly, lasts 2-3 weeks-2 months. From the onset of urine hyperchromia, alcoholic stools. The liver is considerably enlarged (4-5 cm). Signs of intoxication are moderate.
- Paraclinical data: moderate bilirubinemia with predominance of the unconjugated fraction, moderate transaminases, increased cholesterol and alkaline phosphatase. Severe forms are common, evolving to chronicity, increased lethality. The positive diagnosis is established by the presence of HBV markers in children and mothers.
- The diagnosis of viral hepatitis B along with the clinical picture, epidemiological data, biochemical tests will be confirmed by determining the HBV markers: HBsAg, HBeAg, anti-HBc IgM and IgG antibodies, anti-HBe, hepatitis B virus DNA.

Hepatitis B: Diagnosis

1. **Symptoms**
2. **Elevated transaminases**
3. **Confirmed by serology**
   - IgM,IgG anti-HBc (IgM or IgG core antibody)
   - HBsAg (surface antigen)
   - HBsAb/anti-HBs (antibody to surface antigen)
   - HBeAg (E antigen)
   - HBeAb/anti-HBe (antibody to E antigen)

Differential diagnosis

- In the preicteric period: acute enteric infections, acute surgical diseases
- In the icteric period: suprahepatic jaundice (hemolytic jaundice), yersiniosis, infectious mononucleosis, herpetic infection, salmonellosis, hepatocellular jaundices of other etiology (B, C, D, E)
- Toxoplasmal, cytomegaloviral hepatitis

- The patients are discharged from hospital when they get their clinical recovery (satisfactory general condition, absence of icterus, decrease of liver syze) and normal functional tests (normal bilirubin level, lowering of transferase activity in blood serum, - it may be higher than normal but only two-three times!) on the 30th-40th day of illness.

Viral hepatitis C

- Is an infectious disease, which is transmitted parenterally and characterized by not severe course and frequent development of chronic forms with following transformation into hepatocirrhosis and hepatocarcinoma.
- Acute viral hepatitis C (HCV) occurs in children of various ages.
- is the most common posttransfusion viral hepatitis

Hepatitis C

"transfusion related non-A, non-B hepatitis"

- Caused by **RNA flavivirus**
- Accounts for 16% acute hepatitis
- Usually asymptomatic or mild disease
- Chronic infection very common
- 20% of community acquired hepatitis
- 90% post-transfusion hepatitis

Clinical manifestations

- The incubation period of viral hepatitis C is approximately from 6 to 8 weeks.
- Preicteric phase is short. Its duration is 5-7 days in average.
- Clinical manifestations of preicteric and icteric phase are like ones in mild form of viral hepatitis B.
- Biochemical changes are typical for all viral hepatitis. Thymol test is abnormally high.
- Fulminant forms do not occur. In spite of favorable course of the acute period of the disease, forming of chronic hepatitis C occurs in 20-50 % of the patients.

Hepatitis C Diagnosis

1. **Symptoms**
2. **Elevated transaminases**
3. **Rule out other causes of hepatitis**
4. **Confirmed by serology**
    - Serologic test detects HCV antibody
    - Positive in chronic cases
    - May not be positive in acute phase
    - Rule out other causes of acute hepatitis

Viral hepatitis D

- is an infectious disease, which is transmitted parenterally and characterized by clinic and laboratory signs of liver lesion, frequent development of fulminant forms of the disease or chronic acute hepatitis with rapid transformation into hepatocirrhosis.

Hepatitis D

- Virus-like particle
- Defective RNA virus
- Requires HBV co-infection to replicate

- **ANYONE WHO IS HBsAg(+) IS AT RISK**

Etiology

- The disease is caused by hepatitis D virus (HDV), which contains RNA.
- In human organism HDV genome is covered by HBsAg. HBV takes the part of the helper virus of HDV.
- In absence of HBV, HDV cannot replicate.
- HDV is characterized by high thermostability and is not inactivated by ultraviolet rays.

Clinical manifestations

- Clinical signs of viral hepatitis D depend on the forms of infection (co-infection or superinfection).
- The incubative period fluctuates from 1.5 to 6 months.
- Duration of preicteric phase is shorter than in viral hepatitis B and this phase has a more acute onset.
- The disease is manifested by low-grade fever (up to 38°C), severe pain in hepatic region, arthralgias of large joints.
- In icteric phase low-grade fever remains, and keeps for 7-12 days. Toxemia and pain in right hypochondrium increase. Changes of liver are similar to those in hepatitis B, but urticarial rash and splenomegaly is found more frequently.

- The typical sign of hepatitis D is double-phase course of the disease: on 15-32 day from the onset of the icteric phase appearance of weakness, body temperature, arthralgias, increasing of serum transaminases and serum bilirubin levels, and jaundice are found. The second phase is caused by influence of HDV (whereas the previous changes have been caused by HBV).
- Biochemical tests are like the ones in viral hepatitis B.

- The outcome of co-infection is less favorable, than viral hepatitis B: in 70 % of the patients the disease has a severe course, fulminant form of hepatitis occurs in half of them. Probability of forming chronic hepatitis is the same as in hepatitis B because the main pathogenetic mechanism of its development is inability of immune system to eliminate HBsAg from the human body.

- Superinfection has the incubative period from 1 to 2 months.
- Preicteric phase is short, its duration is 3 to 5 days only. The signs of toxemia are severe. The patient main complains are the pain in right hypochondria, arthralgia. Hepatosplenomegaly is found in all of the patients. Dimensions of spleen are enlarged significantly.
- Transition of the disease into icteric phase is not accompanied by improvement of patient's general condition, like in hepatitis co-infection. Clinical manifestations are similar to other types of viral hepatitis. It is accompanied by fever, nausea, anorexia, pain in the hepatic region, increasing jaundice. The exacerbation may have a severe course and signs of acute hepatic failure may occur.
- Biochemical findings in acute phase of the illness are similar to those in hepatitis B.

Diagnosis Of Hepatitis D Virus (HDV) Infection

	Acute HDV/HBV coinfection	HDV superinfection of HBV carrier	Chronic HDV infection
HDAg	Early and short-lived	Early, but soon masked by anti-HDV	Undetectable (masked by antibodies)
Anti-HDV, IgM	Transient and delayed	High-titer and long-lasting	High-titer
Anti-HDV, IgG	Late, increasing titers	Late, increasing titers	High-titer
HDV RNA	Early and sensitive marker	Early and sensitive marker	Usually high-level
IgM anti-HBc	Positive	Negative	Negative

HDV, hepatitis D virus; HBV, hepatitis B virus; HDAg, HDV antigen; IgM, immunoglobulin M; IgG, immunoglobulin G; anti-HBc, hepatitis B core antibody.

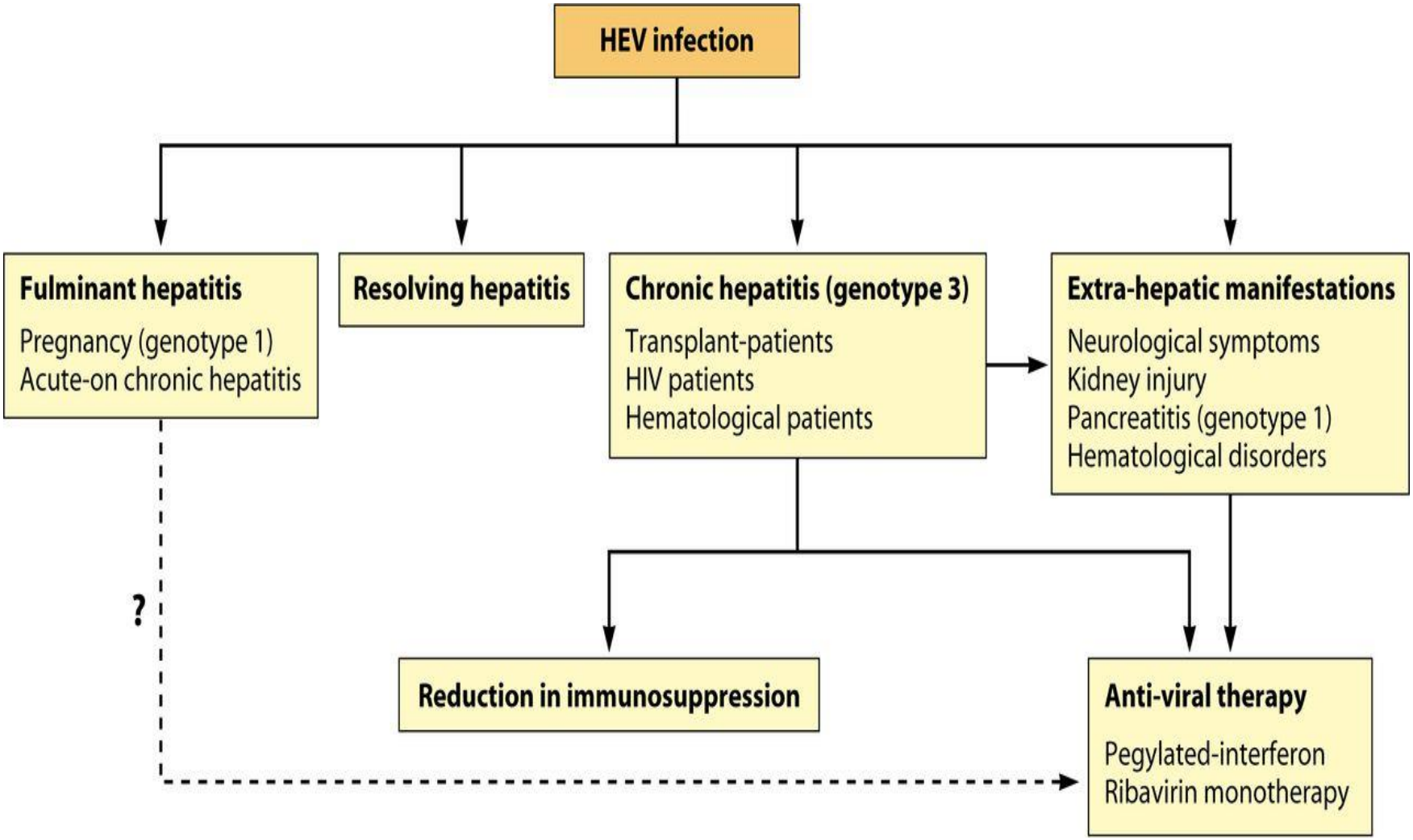
Acute viral hepatitis E(HEV)

- is an infectious and contagious disease caused by the hepatitis E virus (HEV) with a clinical and histological picture similar to viral hepatitis A with self-limiting evolution, without chronicity.

Epidemiology

- Hepatitis E spreads widely in tropical and subtropical countries.
- The source of infection is the patient with various clinical forms of VHE.
- The infection is spread by fecal-oral route. Large epidemics and outbreaks can occur. They are connected with the usage of contaminated water. Susceptibility to the infection is general.

Possible evolutions of HEV



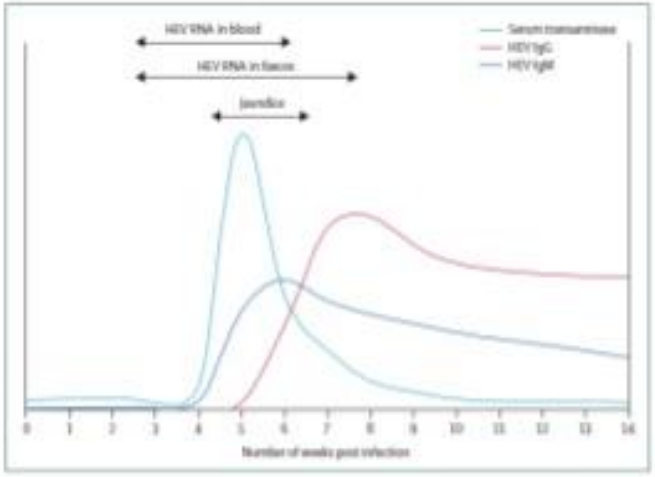
Hepatitis E

- Produces symptoms mostly in 15-40 year olds
- Children usually have sub-clinical infection
- Outbreak potential
- 15-60 day incubation period, usually 5-6 weeks
- No chronic carriers: self-limited
- Severely affects pregnant women with a the mortality rate of 10-20%
- Overall mortality rate is 0.5-3%

- A special sign, which has been discovered in viral hepatitis E, is hemoglobinuria. Its appearance is connected with hemolysis of erythrocytes. It is found in patients with severe forms of hepatitis E only.
- Hepatitis E has especially severe course in pregnant women in 2nd and 3rd trimester of pregnancy. The disease is characterized by high frequency of abortions and premature births.
- In 1-3 days the hepatic failure occurs in mother. Lethal outcomes in pregnant women fluctuate from 20 % (the 20th-21 st weeks of pregnancy) up to 75 % (the 40th week of pregnancy).
- Hemorrhagic syndrome is a dangerous complication of the disease. It is manifested by gastromenia, enteromenia, epistaxis. Acute renal failure occurs in half of the patients with severe form of hepatitis E.

Diagnosis of Hepatitis E

- Clinical presentation - acute hepatitis
  - Jaundice, anorexia, hepatomegaly, abdominal pain/tenderness, nausea/vomiting, fever etc.
- Biochemical markers
  - ↑ Alanine/aspartate aminotransferase, bilirubin, alkaline phosphatase
- Confirmation of HEV infection
  - Detect virus or components
    - Particles (EM – stool)
    - RNA (blood, stool)
    - Ag (blood, stool)
  - Detect anti-HEV
    - IgM
    - IgG
    - IgA (?)



Kamar *et al.*, 2012 Lancet

TREATMENT OF ACUTE VIRAL HEPATITIS.

It includes, first of all, **bed rest** in the first 2-4 weeks of illness and **diet**. The diet should be adapted to the stage of the disease, its Severity and the age of the child. It is recommended to ensure a Sufficient fluid intake.

In mild HV forms, treatment is only hygienic and symptomatic.

In moderate and severe forms, in case of gastric intolerance the fluids are administered intravenously (slow infusions with glucose solution of 5-10%, sol.Ringer lactate.

Drug treatment is not indicated, except for vitamins of the B complex (B1, B2, B6), as well as C, E, K.

In the absence of effective etiotropic therapy, treatment is pathogenic, supportive, symptomatic.

Antiviral therapy is indicated in HBV at risk of chronicity: persistence of markers of viral replication and hypertransaminase.

- **In malignant form** or when "alarm" signs appear, it is recommended:
- Corticotherapy (Prednisolone at a dose of 5-10-15 mg / kg / 24 hours, 4-6 times a day i / v, short course).
- Detoxification therapy with nictemeral volume equal to the physiological requirement of fluids, orally and i / v. Intravenous is administered alone. Glucose 5-10%, sol.Sodium chloride 0.9%, sol.Ringer lactate, sol.Albumin 10-15%.
- Oral antibiotic therapy to prevent endogenous superinfection.
- Antiproteolytic (Aprotinin), 5% aminoacapronic Ac); treatment of cerebral edema with diuretics, control of hemorrhagic syndrome, correction of acid-base and hydroelectrolytic balance, bacterial superinfections, etc.

PROPHYLAXIS.

- **Prevention in HVA and HVE** are the usual hygiene measures for fecal-oral infections (individual and collective hygiene, ensuring sufficient amounts of drinking water and its chlorination according to the rules).
- **Preventive measures in viral hepatitis B, C, D, G** consist in:

✓ prevention of infections by inoculation (correct sterilization of medical and non-medical instruments),

✓ testing of blood donors and bone marrow grafts at HV markers.

Specific HAV prophylaxis is given with Havrix vaccine from the age of 2 in closed institutions for children or individually.

Specific prophylaxis of HBV and HVD includes the administration of recombinant HBsAg vaccine to children at birth (in the first 24 hours of life), at 2-4-6 months.

- Revaccinations can be performed every 5-10 years.
- It should be noted that the VB vaccine is indicated for premature babies with a body weight> 2 kg.
- If the newborn weighs less than 2 kg, the vaccine will be given in 1 month.

- **If the mother is HBs-positive** and the newborn's body mass is **more than 2 kg**, the hepatitis B vaccine will be given with the **anti-HBV immunoglobulin in the first 12 hours after birth.**
- **Revaccination -** at the age of 1 and 6 months.
- If the body **weight of the newborn is less than 2 kg**, the **HBV vaccine** is indicated **in the first 12 hours after birth.** **Revaccination -** at the age of 1, 2 and 6 months.
- If the hepatitis B immunoglobulin is not available, the hepatitis B vaccine is given at 0, 1 and 2 months of age.
- Additional dose between 9 and 12 months.