DIFFERENTIAL DIAGNOSIS OF JAUNDICE IN CHILDREN

Recommendations for V–VI students
Higher medical education institutions
of the III–IV accreditation levels studying in English

МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
Харківський національний медичний університет

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ДИФЕРЕНЦІЙНА ДІАГНОСТИКА ЖОВТЯНИЦЬ У ДІТЕЙ

Методичні вказівки для студентів V–VI курсів вищих медичних закладів освіти III–IV рівнів акредитації, що навчаються англійською мовою

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Упорядники Маргарита Олександрівна Гончарь
Тетяна Сергіївна Маліч

В методичних вказівках для студентів V–VI курсів вищих медичних закладів освіти III–IV рівнів акредитації, що навчаються англійською мовою, наведено головні моменти етіології та патогенезу, а також основні сучасні погляди на діагностику, диференційну діагностику жовтяниць у дітей різного віку.

Методичні вказівки можуть бути використаними студентами медичних факультетів для підготовки до практичних занять з навчальної дисципліни «Педіатрія».

Differential diagnosis of jaundice

Introduction

Hyperbilirubinemia of different genesis in infants and newborn period is still one of the leading places in the structure of neonatal pathology. The causes that lead to their development are diverse, and jaundice of the skin and mucous membranes can be a symptom of many other diseases, which often makes their differential diagnosis very difficult. Finding out the causes of jaundice in newborns allows timely diagnosis and adequate therapy, which is one of the important conditions for preventing such a serious complication as the development of bilirubin encephalopathy and subsequent disability of children

Jaundice (Icterus) – the jaundice staining of the skin and the visible mucous membranes of the eyes and oral cavity, is observed in 40–50 % of newborns. The leading cause of jaundice is hyperbilirubinemia. The clinical importance of the latter is very great because it leads to the development of deep metabolic disorders in the body of the newborn, changes in the functional state of the liver and to cerebral disorders. In the diagnosis of jaundice, it is necessary to evaluate all stages of destruction and transport of hemoglobin: from cells of the reticuloendothelium to liver cells, excretion of bile into the intestine and, finally, excretion by the kidneys, as well as to take into account special functional criteria. Currently, several variants of neonatal jaundice classifications are used for practice.

Jaundice of newborns (pathogenetic classification)

1 Hereditarily

Increased production of bilirubin

1. Erythrocytic membranopathies (microspherocytosis, elliptocytosis, etc.).

2. Erythrocyte enzyme deficiency (glucose-6-phosphate dehydrogenase, pyruvate kinase, etc.).

3. Hemoglobinopathies – defects in structure (Minkowski-Schofar's disease , M-hemoglobinemia, etc.) and synthesis of hemoglobin (thalassemia), heme (congenital erythroporphyria).

2 Acquired:

1. Hemolytic disease of newborns, as a result of alloimmune conflict between mother and fetus.

2. Hemorrhage (cephalogematoma, aponeurosis, into the brain, internal organs), abundant pyechiae and ecchymoses, etc.

3. Syndrome of swallowed blood.

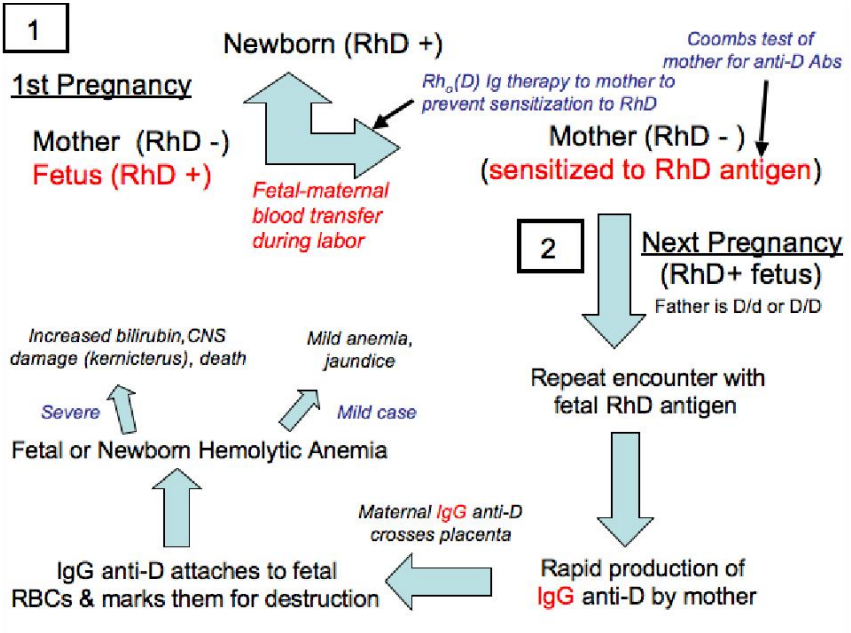
4. Polycythemia.

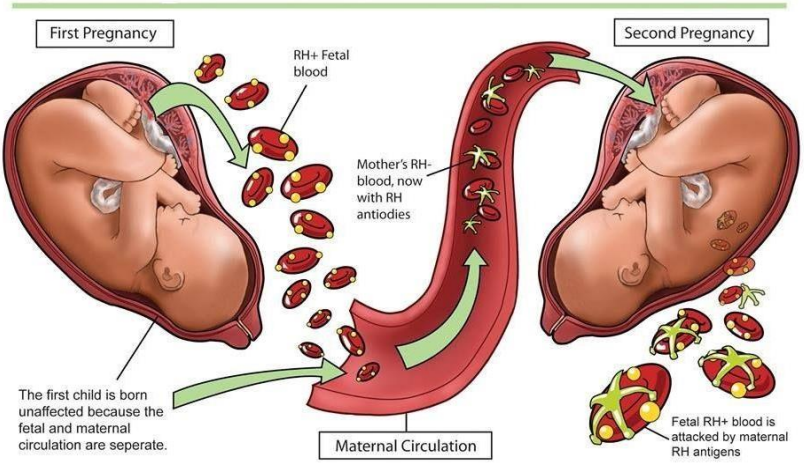
5. Medicinal hemolysis (vitamin K, sulfonamides; maternal oxytocin, etc.).

6. Increased enterohepatogenic circulation of bilirubin (pilorostenosis, jaundice from breast milk, bowel obstruction, etc.)

7. Vitamin E-deficiency anemia and neonatal pycnocytosis.

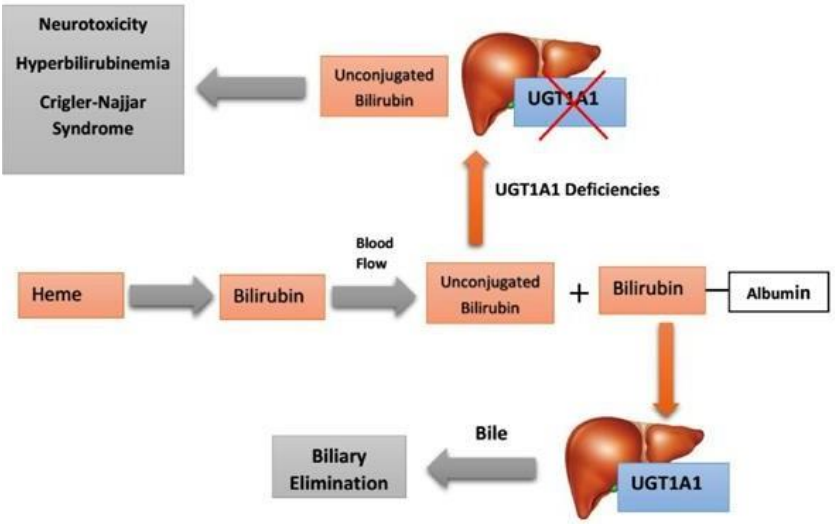
Pathogenesis of hemolytic disease





Pathogenesis of hemolytic disease

Reduced clearance of bilirubin (liver jaundice)



Pathogenesis of Crigler-Najjar Syndrome

Reduced clearance of bilirubin (liver jaundice)

1. Defect of capture of bilirubin by hepatocytes (Gilbert's disease).

2. Defects in the conjugation of bilirubin (Krigler-Najjar syndromes type I and II, Lutz-Driscoll).

3. Defects of bilirubin excretion from hepatocytes (Dubin-Jones syndrome, Rotor).

4. Symptomatic in hypothyroidism, galactosemia, fructosemia, hyper-methioninemia, hyperammonemia, etc.

5. Deficiency of hormones (hypothyroidism, hypopituitarism), or their excess (jaundice from breast milk

Parenchymal jaundice
Infectious hepatitis.
Toxic hepatitis (sepsis, poisoning, medicinal).
TORCH-infection

Obstructive (mechanical) jaundice (obstructive pediatric cholangiopathy)

1. Atresia or hypoplasia of the extrahepatic bile ducts of the fetal type –- syndromatic abnormalities of the biliary tract in combination with other malformations (Alagillilla syndrome, "Norwegian cholestasis", trisomy on the 13th, 18th, 21st pairs of autosomes).

2. Familial, non-syndromic cholestasis (Bayler, McElfresh, etc.)

3. Symptomatic cholestasis in hereditary disease s – cystic fibrosis, alpha-1-antitrypsin deficiency, hemochromatosis, histiocytosis X, Niemann-Peak disease , glycogenic disease , hepatic-cerebrorenal syndrome with beta-dihydroxyhydroxysterohydroxysteroid deficiency.

4. Cholestasis with enlargement of the biliary tract (Caroli's disease , polycystic disease , congenital liver fibrosis).

5. Atresia or hypoplasia of the extrahepatic biliary tract due to perinatal hepatitis, probably caused by type 3 reoviruses.

6. Intrahepatic atresia and hypoplasia of the biliary tract in perinatal hepatitis of different etiology, as well as in primary biliary cirrhosis, primary sclerosing cholangitis, transplant-versus-host reaction, rejection of transplanted liver.

7. Stenosis of the common bile duct or his cyst.

8. Cholelithiasis.

9. Tumors and other entities.

10. Bile syndrome, bile duct syndrome.

11. Jaundice of mixed genesis with the dominance of one of the components.

12. Transient jaundice of newborns.

13. Neonatal jaundice of prematurity.

14. Sepsis.

Clinical and laboratory classification of neonatal jaundice

I. Jaundice with indirect hyperbilirubinemia:

1. Hemolytic anemia:

a) hemolytic disease of newborn;

b) hereditary membranopathies, hemoglobinopathies and erythrocytic fermentopathies;

c) acquired (infectious, medicinal, microangiopathic).

2. Polycythemia.

3. Hematomas and syndrome of swallowed blood.

4. Children from mothers with diabetes.

5. Hereditary:

a) defects in bilirubin clearance;

b) symptomatic in hypothyroidism and other endocrinopathies, galactosemia, fructosemia and other metabolic abnormalities.

6. Reduced release of bilirubin from the intestine and increased intestinal and hepatic circulation of bilirubin:

a) jaundice from breast milk;

b) jaundice with pylorik stenosis;

c) jaundice with meconial ileus;

d) jaundice with bowel obstruction.

II. Jaundice with direct hyperbilirubinemia (dominated by BDG serum) but with a normal color stool:

1. Hepatitis (viral, bacterial, parasitic, fungal, toxic).

2. Hereditary metabolic abnormalities (galactosemia, fructosemia, tyrosinemia, Dubin-Jones syndrome, Rotor, Byler, glycogenosis, cystic fybrosis, accumulation disease s).

III. Jaundice with direct hyperbilirubinemia and with discolored stools of varying severity (cholestatic jaundice):

1. Complete intrahepatic atresia of the biliary tract (without or with hepatitis):

a) normal extrahepatic biliary tract;

b) hypoplasia of the extrahepatic biliary tract.

2. Complete extrahepatic atresia of the biliary tract:

a) normal number of biliary tract;

b) reduced number of biliary tract.

3. Hypoplasia of the extrahepatic biliary tract:

a) normal number of biliary tract;

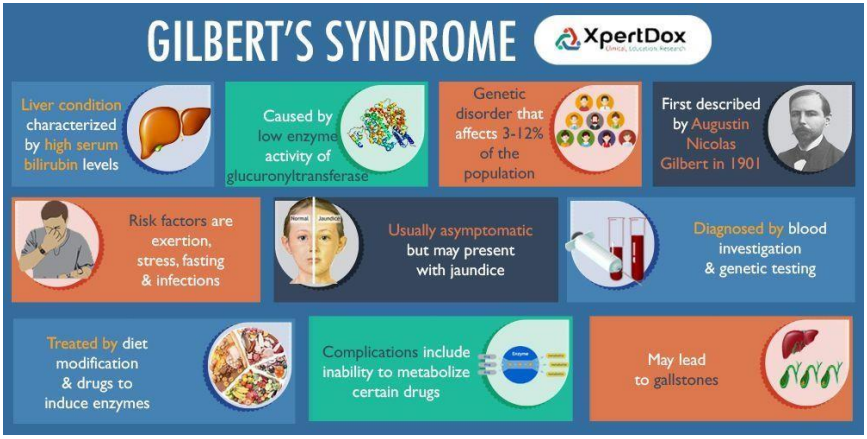
b) reduced number of biliary tract.

4. Hepatitis without bile duct abnormalities.

5. Bile syndrome (bile duct syndrome), cholelithiasis.

6. Cyst of the bile duct or compression outside the biliary tract.

7. Cystic fibrosis and alpha-1-antitrypsin deficiency.

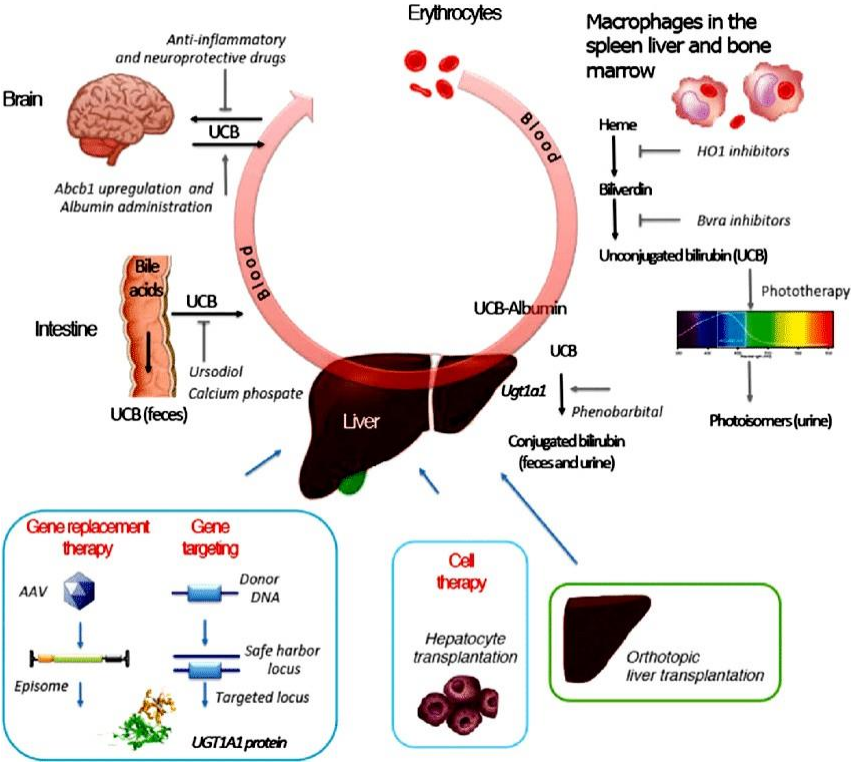


A special difficulty in the diagnostic plan is the differential diagnosis of hereditary defects of bilirubin clearance in newborns. In practice, you can use the following criteria:

Jaundice (Gilbert-Maylegrapht syndrome) Has ancestral origin (autosomal dominant type of transmission) and is characterized by hepatic

dysfunction, a slight decrease in the conjugation of indirect bilirubin. Therefore, hyperbilirubinemia is negligible. It rarely occurs in the neonatal period, more often in the pre- and pubertal periods with considerable physical and nutritional activity. Treatment is usually not needed. Sometimes glucose, phototherapy is prescribed. The outlook is always favorable.

Krigler-Najjar syndrome



Krigler-Najjar syndrome is a family jaundice caused by unconjugated, non-hemolytic hyperbilirubinemia.

There are two variants of Krigler-Najjar syndrome: type I and type II. The genetic substrate for Krigler-Najjar syndrome is a mutation of the UGT1A1 gene encoding the enzyme uridine diphosphate glucuronyltransferase (UDFGT) in the liver. The most common is the Gly71Arg mutation. It is found in about 20 % of the population of the Asian region. The result of these mutations is either the complete absence of the enzyme (type I syndrome) or a decrease in its activity (type II syndrome).

Type I syndrome was described in 1952 by American pediatrician J. F. Crigler and V. A. Najjar. In type I syndrome, the genetic defect is manifested in the complete absence of UDFGT. Type I syndrome is manifested by neonatal unconjugated hyperbilirubinemia – the level of unconjugated bilirubin in the serum can reach 855 μkmol / l at birth. This leads to the appearance of severe jaundice in the first hours of life and bilirubin encephalopathy, which is the cause of disability of the patient. Phenobarbital therapy is ineffective. In rare cases, patients with type I Kriegler-Najjar syndrome survive to adolescence.

Type II syndrome, known since 1962 in Krigler-Najjar type II, a genetic defect is manifested by a decrease in the activity of the UDFGT enzyme. Type II syndrome can manifest from birth, more often to the third day of life. In many patients with type of Krigler-Najjar II type jaundice appears only in adolescence or no clinical manifestations of the disease. In rare cases (infections or stress conditions), bilirubin encephalopathy can occur. In this variant of the disease there are lower levels of serum bilirubin. In patients with type II syndrome, the level of unconjugated serum bilirubin can reach 340 μkmol/l. The life expectancy of patients with type II syndrome is higher than that of patients with type I syndrome.

The clinical symptoms in patients with type I and II type Krigler-Najjar syndromes are different. This is due to differences in the pathogenetic mechanisms of the two variants of the disease (with type I enzyme UDFGT in the liver is completely absent; with type II, there is a decrease in its activity) and, accordingly, with different expressiveness of unconjugated hyperbilirubinemia. Genetic testing is important in diagnosis.

At the molecular level, the genetic defect in Krigler-Najjar syndrome is the presence of mutations in the UGT1A1 gene encoding the enzyme uridine diphosphate glucuronyltransferase (UDFGT) in the liver. The most common is the Gly71Arg mutation.

To illustrate the above, we provide our own observation of a child with a diagnosis of Krigler-Najjar II type. For the first time, a mother turned to a district pediatrician for complaints about the jaundice of the skin and mucous membranes of her child at the age of 10 days.

From the anamnesis of the disease and life it is known that the boy was born from the first pregnancy, which took place without peculiarities, in the gestation period of 38 weeks, from the first physiological childbirth, with an Apgar score of 7–8 points, weighing 3250, g 52–30–32 cm Mother has 0 (I) Rh + blood type, baby 0 (I) Rh + blood type. The condition after birth was satisfactory. At 3 days of age appeared jaundice color of the skin and mucous membranes, which was regarded as the appearance of physiological jaundice and a child in a satisfactory condition for 3 days of life was discharged from the hospital.

From the family history, it was revealed that the parents are young, healthy. There is Gilbert's syndrome along the line of the maternal grandfather.

In somatic status, the general condition of a child of moderate severity, active, loudly screaming, is in natural feeding, for the first month added 1000 g. The intense bright orange color of the sclera and skin, including the palms and soles, was noticed. At the age of 3 months, the baby was admitted to the CBCL, the mother complained of skin yellowness and mucous membranes, moderate symptoms of intoxication. When examining the child, the bright saffron-jaundice color of the skin and mucous membranes was noticed, but there were no changes in somatic status. In physical and neuro-mental development the child met the age standards. In a complete clinical biochemical examination of the child, changes in bilirubin metabolism were detected only. The level of total bilirubin ranged from 180–300 μkmol/l due to the indirect fraction (150–270 μkmol/l). Given the peculiarities of bilirubin metabolism, which can be impaired in different ways of its formation, transformation and excretion, differential diagnosis was made between 4 types of jaundice: 1) conjugated 2) hemolytic; 3) parenchymal; 4) mechanical. After careful comprehensive clinical and biochemical studies, hemolytic, parenchymal and mechanical genesis of jaundice in this child were excluded. Differential diagnosis was made between different types of conjugative jaundice, caused by impaired processes of conversion of indirect bilirubin into direct. The cause of this condition is the insufficient activity of the enzyme UDF-glucuronyltransferase, which is used to convert indirect bilirubin into direct, resulting in the accumulation of indirect bilirubin in the blood of the child in the absence of signs of hemolysis.

Eliminating the acquired causes of conjugation jaundice in a child, there was a need to make a differential diagnosis between hereditary variants of bilirubin disorders: type Krigler-Najjar I and II type, Gilbert-Meylengraht syndrome. The child was consulted at the medical-genetic center. On full examination, no changes in protein, fat, carbohydrate, electrolyte metabolism were detected. Molecular genetic testing was carried out and the following conclusion was made: in a sample of a child's DNA, a mutation in the UGTIAI gene was performed by direct automatic sequencing. In the promoter region of the UGTIAI gene was registered an increase in the number of "1A" repeats in one of the chromosomes, which leads to a decrease in the functional activity of the protein UDP-glucosyltransferase I. In exons 2 and 4 of the UGTIAI gene mutations were detected p.1176 + 2, 1176 + 5 delta and with .1130G> T (Gly377Val) in the heterozygous state. Among the 50 individuals in the population sample, the Gly377Val mutation was not found. The obtained molecular genetic data confirm the diagnosis of type II Kriegler-Najjar syndrome.

Currently, a child 6 years old, grows and develops satisfactorily, the subjectivity of the skin and mucous membranes only on the face and trunk, the level of bilirubin ranges from 100–180 μkmol/l, in the neurological status there is a syndrome of tonic disorders in the lower extremities. It is constantly monitored and treated by the staff of the department.

Thus, the rare case of detection and timely treatment of hereditary pathology of bilirubin clearance in a child almost from the moment of birth allowed to avoid a severe neurological complication - bilirubin encephalopathy.

Hemolytic jaundice

Hemolytic jaundice is also common. They are typical for them

1) normochromic anemia with hyperregenerative manifestations (reticulocytosis);

2) enlargement of the liver and spleen;

3) growth of free indirect bilirubin;

4) normal color of urine and feces.

Hemolytic disease of newborns is caused by the incompatibility of the mother and the fetus with erythrocyte antigens (rhesus factor, blood groups, rare factors, and about 100 of them) and is diagnosed in 0.5–1 % of newborns. The basis of this disease is enhanced hemolysis of the baby's erythrocytes under the influence of antibodies produced in the body of the mother, relative to the various antigens of the Rh system (Rh) and group antigens. The number of maternal antibodies penetrating the fetus during pregnancy and childbirth (especially intensive), the degree of hemolysis of erythrocytes, the severity of the disease is directly dependent on the previous sensitization of the woman (blood transfusion in the past, previous pregnancies and childbirth, pathology of pregnancy). It should be remembered that sensitizing the body of the woman (increasing the potential for the production of antibodies to antigens of the erythrocytes of the fetus) is not childbirth, and pregnancy, in particular, aborted artificially or spontaneously. That is why it is important to have a firstborn baby without interruption of pregnancy with antigenic incompatibility of red blood cells in spouses.

Clinical manifestations of disease depend on the degree of hemolysis of erythrocytes, and, consequently, on the massiveness of antibodies (the degree of sensitization of the pregnant woman, the permeability of the vessels of the placenta). In this regard, there are 3 vital variants of the diseases:

1) the most frequent – jaundice;

2) rarely – anemic;

3) edema.

The jaundice is severe. The baby is born urgently with normal body weight, more often with unchanged skin. Jaundice appears in the first hours of life, in the second half of the first day, at the beginning of the second and further intensifies. Physiological erythema masks the disease and it can be diagnosed with delay. Therefore, it is necessary to inspect the sclera (skin color and sclera becomes saffron). Especially severe course of this form of disease is that the child is born with jaundice. Amniotic fluid, first-grade grease and fruiting membranes are yellow. This means that the disease begun prenatally and its prognosis is very serious.

The Disease is characterized by enlargement of the liver and spleen (foci of extramedullary hematopoiesis, degenerative changes, swelling occur), rarely – point hemorrhage, mellitus, bleeding from injection sites. Blood hemoglobin sharply decreases (up to 80–60 g/l), there are erythropenia (3–2×1012/l and below), reticulocytosis, normomegaloblastosis, anisocytosis, poikilocytosis, neutrophilosis with left shift, increase in ESR. Premature changes are more pronounced.

However, the severity of disease and the choice of therapeutic measures depend on the amount of free bilirubin in the umbilical cord serum (normal – 6–30 mg/l; 10.3–51.3 μkmol/ l) and peripheral blood, the dynamics of its growth. Critical levels of free bilirubin at birth – 30–35 mg/l (51.3–59.8 μkmol/l) and more, in the first day – 100–120 mg/l (171.0–205.2 μkmol/l), the second – 150–170 mg/l (256,5–290,7 μkmol/l), the third – 180–200 mg/l (307,8–342,0 μkmol/l). These figures indicate the need for a replacement blood transfusion. To confirm disease, it is important to determine the increase in serum bilirubin concentration. Hourly increase of bilirubin in healthy children – from 0.2 to 3.4 μkmol/l/h (from 0.1 to 2 mg/l/h), and in patients – from 5.1 to 17.1 μkmo/l/h (from 3 to 10 mg/l/h).

Factors that contribute to the occurrence of bilirubin encephalopathy:

• damage to the blood-brain barrier – hemorrhage into the brain, neuroinfection, seizures, arterial hypotension, SDR;

• decrease in the ability of albumin to bind indirect bilirubin – prematurity, hypoalbuminemia, acidosis, hypoxia, infections, competition in the use of medicines - antibiotics, diuretics, sedatives, etc;

• increased sensitivity of neurons to the toxic effects of indirect bilirubin – prematurity, severe asphyxia, hypoglycemia, anemia, fasting;

An increase in indirect bilirubin in the serum above the critical numbers causes CNS damage, manifested as bilirubin encephalopathy (bilirubin intoxication or jaundice), which consists of the following phases:

• Asphyctic (hours) – hypotension, sharp suppression of physiological reflexes (especially Moro and sucking), lethargy, bradypnoe, prolonged apnea, asphyxia, cyanosis, stagnant wheezing;

• Spastic – a piercing "brain" cry, extensible hypertonus, stiff neck, spasm of a glance, a "sunset" symptom, seizures and more.

• Imaginary, false well-being – reduced spasticity, hypertension, the impression of the reverse development of the disease .

• Formation of neurological complications – cerebral palsy, athetosis, paralysis, paresis, mental retardation.

Hereditary microspherocytic hemolytic anemia (Minkowski-Schafar)

Hereditary microspherocytic hemolytic anemia (Minkowski-Schafar) also belongs to the group of hemolytic jaundice. The disease is transmitted by the dominant type. Its basis is a defect in the form of erythrocyte and its enzyme systems. It is manifested by periodic crises of erythrocyte hemolysis at

any age, in particular in the first days of life. It is characterized by a triad: anemia, jaundice, splenomegaly. Laboratory parameters are as follows: reduction of osmotic resistance of erythrocytes, presence of microspherocytes, increase of indirect bilirubin level, absence of group and resultant incompatibility.

Treatment is carried out in the period of hemolytic crisis: prednisolone is prescribed, and in the absence of effect - splenectomy. With timely treatment, the prognosis is favorable.

Hereditary jaundice due to insufficient glucose-6-phosphate dehydrogenase

Hereditary jaundice due to insufficient glucose-6-phosphate dehydrogenase (G-6-FD) and erythrocytes is rare. It is described in the section on hemolytic anemia. Clinically manifests a crisis at any age under the influence of medicines and chemicals. It also has a triad (jaundice, anemia, splenomegaly).

The diagnosis is confirmed by the determination of G-6-FD deficiency in erythrocytes. In the treatment of the most important is the exclusion of the factor that caused hemolysis. The outlook is favorable.

Neonatal hepatitis

Neonatal hepatitis refers to many forms of liver dysfunction that affects fetuses and neonates.[1] It is most often caused by viruses or metabolic disease s, and many cases are of an unknown cause.

Signs and symptoms:

The infant with neonatal hepatitis usually has jaundice that appears at one to two months of age, is not gaining weight and growing normally, and has an enlarged liver and spleen. Infants with this condition are usually jaundiced. Jaundice that is caused by neonatal hepatitis is not the same as physiologic neonatal jaundice. In contrast with physiologic neonatal jaundice, infants with neonatal hepatitis present with dark urine. Infants may also present with delayed growth

Causes

The cases of neonatal hepatitis are many. Viruses that have been identified include cytomegalovirus, rubella virus, hepatitis C and B viruses, herpes simplex viruses, coxsackievirus, echovirus, and paramyxovirus.

Metabolic and immune disorders can also cause neonatal hepatitis.

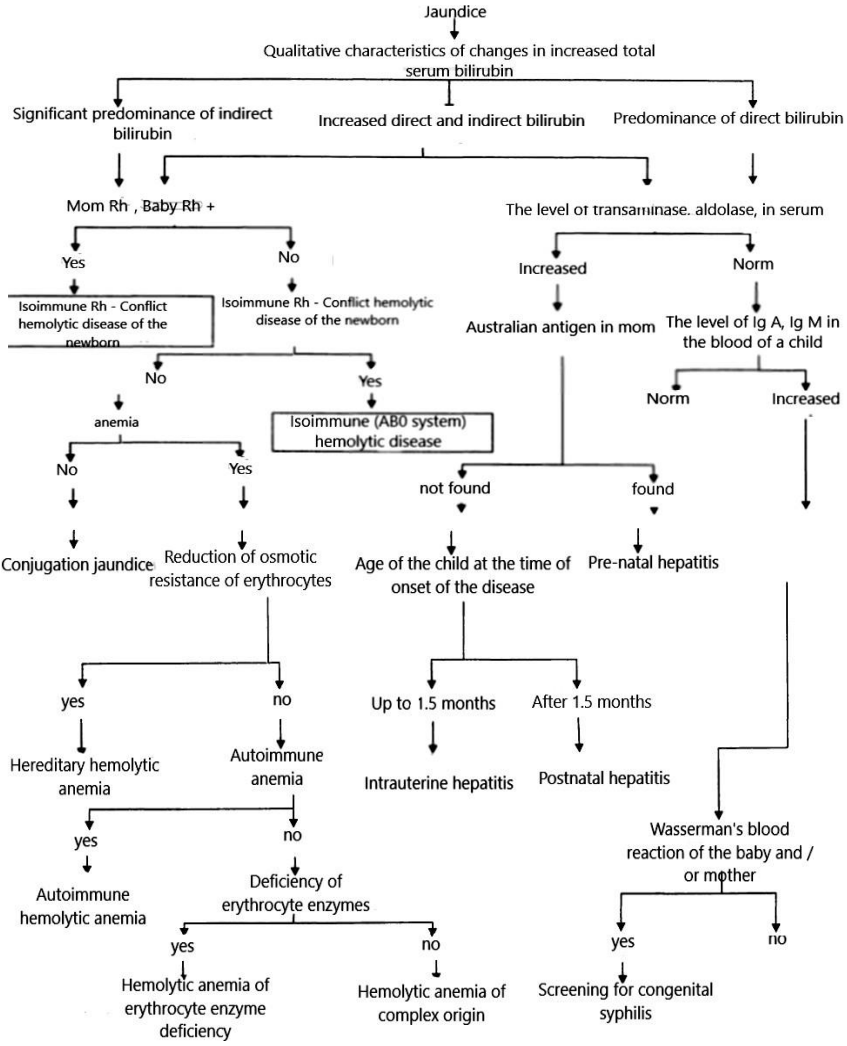
Giant cell transformation throughout the parenchyma is common.

Differential diagnosis

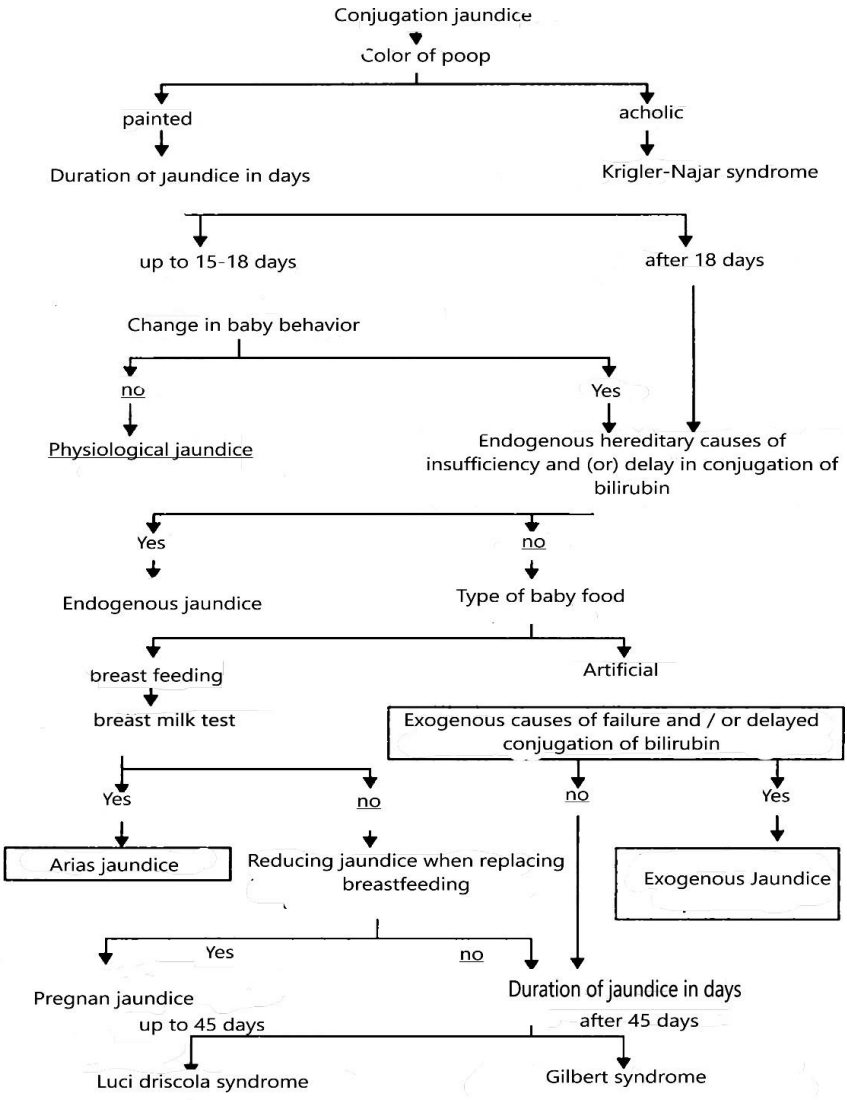
Conditions that can present similarly include galactosaemia, hereditary fructose intolerance, cystic fibrosis, and biliary atresia.

To facilitate differential diagnosis between jaundice of different genesis, we add the algorithms presented in Schemes 1–4.

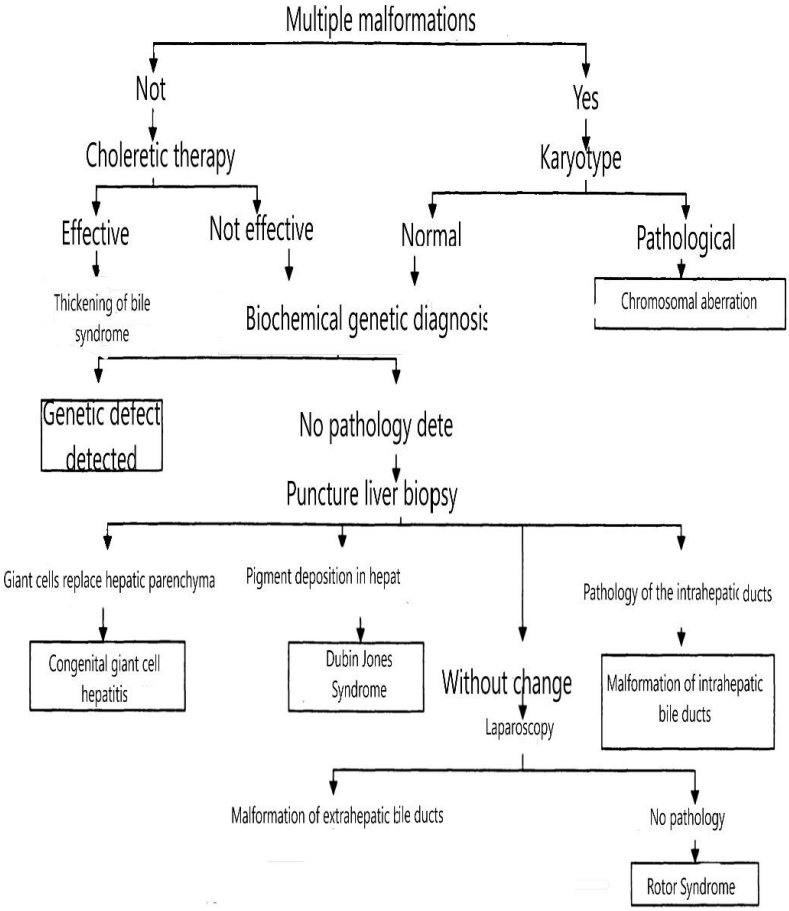
Algorithm of action when the symptom of jaundice:



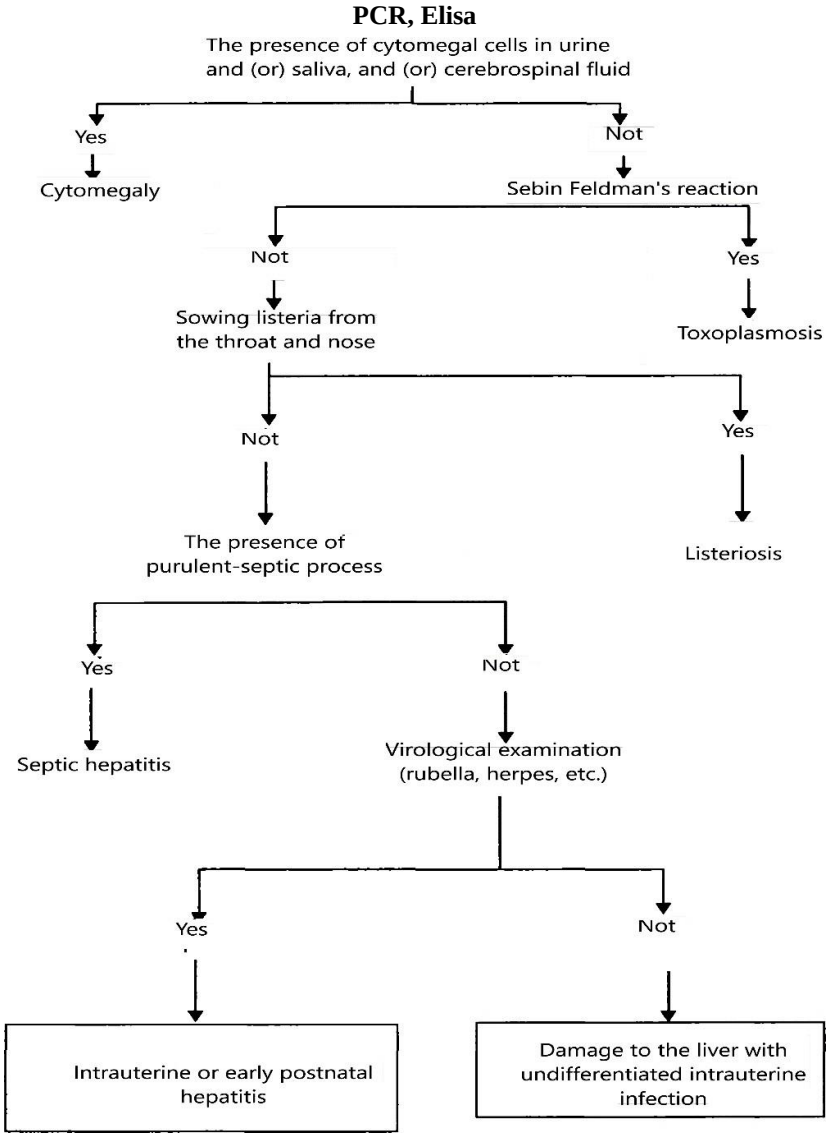
Algorithm for differential diagnosis of conjugation jaundice:



Algorithm of differential diagnostics of mechanical devices:



Algorithm for differential diagnosis of parenchymal jaundice:



TEST CONTROL:

1. At the child for 3 days of life the skin is yellow. Birth weight – 3200 g, body length 52 cm. Active. Over the lungs puerile breathing. BH – 36 per min. Heart tones are rhythmic. HR – 130/min. The abdomen is soft. The liver protrudes from under the costal arch 1.5 cm, the spleen does not palpate. Meconium emptying. Most likely diagnosis:

A. Hemolytic disease of the newborn.
B. Physiological jaundice.
C. Sepsis of newborns.
D. Minkowski-Schofar anemia.
E. Atresia of the biliary tract.

2. At 21 days of age, the skin is yellow. The baby was born with a weight of 3200 g, body length 52 cm. Sluggish. Over the lungs puerile breathing. Breathing – 40 per min. Heart tones are rhythmic. HR – 158/min. The abdomen is soft. The liver protrudes from under the costal arch 4 cm, the spleen does not palpate. Emptying yellow. Most likely diagnosis:

A. Physiological jaundice.
B. Early jaundice.
C. Prolonged jaundice.
D. Late jaundice.
E. Nuclear jaundice.

3. A newborn 20 hours of life has a yellow skin color and sclera. Most likely diagnosis:

A. Physiological jaundice.
B. Early jaundice.
C. Prolonged jaundice.
D. Late jaundice.
E. Gallbladder atresia.

4. Newborn 3 days. Weight at birth 3 450 g, body length – 51 cm Breastfeeding. She yells loudly, actively sucking. On objective examination: the skin is ecteric. Overall condition is satisfactory. Internal organs without pathological changes. Mud, urine of ordinary color. What is the limiting condition of the baby?

A. Newborn baby syndrome.
B. Transient hyperbilirubinemia.
C. Hemolytic disease of the newborn.
D. Atresia of the bile ducts.
E. Syphilis.

5. The cause of hemolytic disease of the newborn is:

A. Immaturity of glucuronyltransferase of the liver.
B. Hemoglobinopathy.
C. Isoimmune hemolytic anemia.
D. Pre-natal infection.
E. Autoimmune hemolytic anemia.

6. A preterm baby born with body weight 1 900 g at 34 weeks gestation age was diagnosed with conjugated jaundice. For the treatment of jaundice use:

A. Phototherapy.
B. Phenobarbital.
C. Infusion therapy.
D. Exchange blood transfusion.
E. All answers are correct.

7. A newborn baby is diagnosed with hemolytic disease, a conflict in the ABO system. At what indicators of umbilical cord blood and hourly gain will you start exchange blood transfusion

A. 50 μkmol/l and 3.5–4 μkmol/l/h.
B. 80 μkmol/l and 10.0 μkmol/l/h.
C. All of the above is incorrect.
D. Everything listed is correct.
E. 100 μkmol/l and 7 μkmol/l.

8. A full-term neonate from I pregnancy of a timely normal delivery within an hour after birth appeared jaundice. In the mother, the blood group is 0 (I) rhesus-negative, in the child A (II), rhesus-positive. Blood bilirubin total 61.3 μkmol/l, indirect 60.3 μkmol/l. An hour later, an indirect 85.3 μkmol/l. Which of the following is paramount?

A. Infusion therapy.
B. Phototherapy.
C. Exchange blood transfusion.
D. Induction of liver enzyme systems.
E. Enterosorbents.

9. A newborn baby is treated conservatively for hemolytic disease, phototherapy sessions. Determine the increase in the daily amount of fluid during phototherapy.

A. 10–20 %. B. 30–40 %. C. 35–40 %. D. 40–45 %. E. 5–7 %.

10. Nuclear jaundice develops as a result of hemolytic disease in the newborn. Late signs of nuclear jaundice include

A. Apnea.
B. Cramps.
C. Sunset syndrome.
D. Opistotonus, bringing the arms to the torso.
E. All answers are correct.

Response standards:

1	2	3	4	5	6	7	8	9	10
B	C	B	B	C	A	B	C	A	E

CONTROL TASK

1. A woman with blood group B(III) Rh(+) gave birth to a full-term healthy boy. Examination on the 3rd day of the infant's life shows him to have icteric tint to his skin. The child has no problems with suckling, sleep is nondisturbed. The abdomen is soft, the liver protrudes by 2 cm from under the costal margin. Complete blood count: hemoglobin – 200 g/L, erythrocytes – 5.5×10^{12}/L, total bilirubin – 62 mcmol/L, indirect bilirubin – 52 mcmol/L. What condition can be suspected?

A. Physiological jaundice.
B. Congenital hepatitis.
C. Hemolytic disease of newborn due to Rh incompatibility.
D. Biliary atresia.
E. Hemolytic disease of newborn due to ABO incompatibility.

2. A 3-day-old infant with hyperbilirubinemia (428 mcmol/L) developed disturbances manifesting as periodical excitation and convulsions against the background of inertness, hypotension, hypodynamia, and inhibition of unconditioned reflexes, convergent strabismus, rotational nystagmus, and setting-sun eye phenomenon. What is the most likely cause of such symptoms?

A. Bilirubin encephalopathy.
B. Craniocerebral injury.
C. Brain tumor.
D. Hydrocephalus.
E. Infantile cerebral paralysis.

3. A baby was born at 36 weeks of gestation. Delivery was normal, by natural way. The baby has a large cephalohematoma. The results of blood count are: Hb – 120 g/l, Er – 3,5 × 10^{12}/l, total serum bilirubin – 123 mmol/l, direct bilirubin – 11 mmol/l, indirect – 112 mmol/l. What are the causes of hyperbilirubinemia in this case?

A. Erythrocyte hemolysis.
B. Intravascular hemolysis.
C. Disturbance of the conjugative function of liver.
D. Bile condensing.
E. Mechanical obstruction of the bile outflow.

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Навчальне видання

ДИФЕРЕНЦІЙНА ДІАГНОСТИКА ЖОВТЯНИЦЬ У ДІТЕЙ

Методичні вказівки для студентів V–VI курсів вищих медичних закладів освіти III–IV рівнів акредитації, що навчаються англійською мовою

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