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Types of Pathological Jaundice in Newborns

Bakhronova Dilshoda Tokhirovna 1

¹ Student of the pediatric faculty of the Bukhara State Medical Institute

Abstract:

Neonatal jaundice is a visual manifestation of increased bilirubin levels, manifested in full-term newborns with a total bilirubin level of 85 μ mol/L, and in premature newborns - 120 μ mol/L. Hyperbilirubinemia* – an increase in the level of total bilirubin above 342 μ mol/l (20 mg/dl; mg/dL).

Keywords: Newborn, jaundice, hyperbilirubin, bilirubin, hemoglobin.

Pathogenetic classification of neonatal jaundice

Increased bilirubin production:

Hereditary jaundices:

erythrocyte membranopathy (microspherocytosis; elliptocytosis);

erythrocyte enzyme deficiencies (glu-6-ph dehydrogenase, pyruvate kinase);

hemoglobinopathies:

sickle cell anemia,;

M-hemoglobinemia;

globin synthesis defects - thalassemia;

heme synthesis defects - erythroporphyria.

Acquired jaundices:

HDN;

hemorrhages;

swallowed blood syndrome;

polycythemia;

drug-induced hemolysis (oxytocin, vitamin K, sulfonamides);

increased enterohepatic circulation (pyloric stenosis, maternal milk jaundice (Arias' sn-m (pregnane jaundice) CN).

Reduced bilirubin clearance:

Hereditary jaundices:

Defect of bilirubin uptake by hepatocytes (Gilbert syndrome);

Defect of bilirulin conjugation (Crigler-Najjar, Lucey-Driscoll syndromes);

Defect of bilirubin excretion from hepatocytes (Dubin-Jones and Rotor syndromes);

Symptomatic (hypothyroidism, hyperammonemia).

Acquired jaundices:

Hormonal deficiency (hypothyroidism, hypopituitarism);

Hormonal excess (jaundice from breast milk);

Infectious hepatitis;

Toxic hepatitis (sepsis, drug poisoning);

Prematurity;

Total parenteral nutrition.

Obstructive (infantile cholangiopathy):

Hereditary jaundices:

atresia (hypoplasia) of the extrahepatic bile ducts in combination with other malformations;

familial nonsyndromic cholestasis (Bayler, McElfresh);

symptomatic cholestasis in hereditary diseases (cystic fibrosis, histiocytosis X, Niemann-Pick disease, adrenogenital syndrome).

Acquired jaundices:

atresia or hypoplasia of the extrahepatic bile ducts due to perinatal hepatitis;

atresia and hypoplasia of the intrahepatic bile ducts in perinatal hepatitis, primary biliary cirrhosis, sclerosing cholangitis, GVHD;

stenosis or cyst of the common bile duct;

cholelithiasis;

bile thickening syndrome;

bile plug syndrome;

sn-m Alagille.

Mixed genesis with dominance of one of the components: Sepsis.

Classification of neonatal jaundices by the predominant fraction of bilirubin

unconjugated hyperbilirubinemia:

A. Increased bilirubin formation:

1. Hemolysis:

erythrocyte membranopathy: spherocytosis; elliptocytosis; pycnocytosis; stomatocytosis; erythrocyte enzyme deficiencies: glucose-6-phosphate dehydrogenase deficiency; pyruvate kinase deficiency; hemoglobinopathies: alpha- and gamma-thalassemia; immune-mediated hemolysis (positive direct Coombs test): rhesus immunization: AB0 immunization; immunization for rare factors. 2. Increased enterohepatic circulation: intestinal obstruction, pyloric stenosis; ileus, meconium plug, pancreatic cystic fibrosis; breastfeeding (Arias syndrome - pregnane jaundice; not to be confused with Arias syndrome -Crigler-Najjar syndrome type II) 3. Diseases accompanied by blood extravasation (for example, cephalohematoma, intracranial hemorrhage). 4. Polycythemia. B. Decreased hepatic clearance of bilirubin (decreased rate of elimination): 1. Prematurity, including premature babies with late gestation. 2. Hormonal deficiencies: hypothyroidism; hypopituitarism. 3. Impaired bilirubin uptake by the liver: patent ductus venosus (Arantziev's); polymorphism of the SLCO1B1 gene; 4. Impaired bilirubin conjugation: Crigler-Najjar syndrome types I and II; Gilbert's disease. 5. Diseases accompanied by increased enterohepatic circulation: intestinal obstruction, pyloric stenosis; ileus, meconium plug, cystic fibrosis of the pancreas;

breastfeeding (Arias syndrome - pregnane jaundice; not to be confused with Arias disease - Crigler-Najjar syndrome type II) Conjugated hyperbilirubinemia: A. Infectious causes (perinatal cholecystocholangitis): 1. Bacterial and parasitic agents: sepsis; listeriosis; tuberculosis; urinary tract infections; toxoplasmosis; malaria. 2. Viral: CMV; HSV; rubella; reovirus; adenovirus; enterovirus; parvovirus B6; syncytial giant cell hepatitis with paramyxovirus-like inclusions; hepatitis B and E. B. Biliary tract pathology: Biliary atresia. Choledocholar cysts. Spontaneous perforation of the common bile duct. Mucus/bile plug syndrome. Neonatal sclerosing cholangitis. Insufficiency (hypoplasia) of the intrahepatic bile ducts. Alagille syndrome. Caroli disease. Cystic fibrosis. Idiopathic stricture of the bile duct.

Cholelithiasis.

O. cholecystitis.

Chronic cholecystitis.

Acalculous cholecystitis.

O. dropsy of the gallbladder.

Causes of physiological jaundice in newborns

Increased rate of bilirubin formation due to: physiological polycythemia; shorter lifespan of red blood cells containing fetal hemoglobin; catabolic orientation of metabolism and formation of bilirubin due to non-erythrocyte sources (myoglobin, pyrrols, cytochrome). Decreased functional ability of the liver to excrete bilirubin: decreased uptake of bilirubin by hepatocytes; decreased activity of enzyme systems, in particular glucuronyl transferase (reaches the level of adults by 1 - 2 months of life); decreased excretion. Increased re-entry of indirect bilirubin from the intestine into the blood, caused by: high activity of intestinal beta-glucuronidase; entry of part of the blood through the Arantius duct into the inferior vena cava, bypassing the liver; transient intestinal dysbiocenosis.

Treatment of jaundice of breastfeeding and mother's milk

cholekinetics: MgSO4 12.5% ½ - 1 teaspoon x 3 times a day until an acceptable decrease in the NB level;

discontinuation of breastfeeding for 48 - 72 hours (a differential diagnostic sign will be a decrease in the NB level at the end of the specified period);

phototherapy.

Used literature

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