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DIFFERENTIAL DIAGNOSIS OF JAUNDICE LITERATURE REVIEW

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Abstract
The article describes the characteristics of jaundice, the causes that cause it, and outlines the issues of differential diagnosis. The emphasis is placed on the fact that when carrying out differential diagnosis of jaundice, it is important to determine whether jaundice belongs to one group or another, and then carry out diagnosis within the group. The functional state of the liver was assessed based on the results of biochemical blood tests. It was revealed that with obstructive jaundice, against the background of lipid metabolism disorders and endogenous intoxication, there is an increase in viscosity and aggregation ability of erythrocytes, a decrease in their deformability, coagulation potential and an increase in fibrinolytic activity of the blood. An increase in fibrinolytic activity of the blood indicates the risk of bleeding in the postoperative period, and a decrease indicates thrombotic complications. All these changes are more pronounced in patients of the second group with prolonged obstructive jaundice, when, along with cholestatic processes, cytolytic processes develop and progress. Therefore, in terms of preventing detected disorders and their timely treatment, it is of great importance to identify hidden mechanisms for the development of complications using laboratory studies.

Keywords: jaundice, pathogenesis, differential diagnosis of obstructive jaundice, indicators of homeostasis, endogenous intoxication, hemostasis.

Introduction

The detection of jaundice is not difficult, since it is a well-marked sign that attracts the attention of not only medical professionals, but also the patient himself and those around him. It is always much more difficult to find out its cause, because jaundice is observed in many infectious and non-communicable diseases. Common to all jaundices is the jaundice staining of the skin against the background of an increase in the level of bilirubin in the blood serum. It must be remembered that jaundice staining of the skin can be caused by a number of other reasons, for example, when certain chemicals are deposited in the tissues (when taking akrikhin, occupational hazards). [2-3] Sometimes a peculiar yellowish-orange coloring of the skin is observed with excessive intake of

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carotene from food into the human body or with its insufficient absorption. This condition can often be observed in children with excessive consumption of carotene-containing foods and juices from them (carrots, oranges). In such situations, well-being is not impaired, skin staining occurs gradually. The skin color is not yellow, but yellowish-orange. The most distinct color will be on the skin of the palms, soles, in the chin area, around large joints. The bilirubin content in the blood serum is within the normal range. When it is established that the patient does have jaundice, a more difficult stage of differential diagnosis begins, since there are quite a lot of causes of jaundice and its genesis is ambiguous. It may be due to increased hemolysis of red blood cells, liver damage, cholestasis, or a combination of these factors. [2-4] In the differential diagnosis of jaundice, it is necessary to take into account violations of individual links of pigment metabolism. In a healthy person, about 1% of circulating red blood cells disintegrate daily. When hemoglobin breaks down, bilirubin is formed in the cells of the reticuloendothelium. The resulting bilirubin does not dissolve in water, does not pass through the renal filter into the urine. This is free (indirect) bilirubin (phase I of pigment metabolism). It accumulates in the liver, in the perisinusoid space (Disse) and is actively captured by hepatocytes (phase II of pigment metabolism). In liver cells, free bilirubin is converted into bound bilirubin. With the participation of the enzyme glucuronyltransferase, bilirubin combines (conjugates) with glucuronic acid and turns into bound (direct) bilirubin (phase III of pigment metabolism). The bound bilirubin is soluble in water and can be excreted in urine. Bound bilirubin is excreted through the biliary pole of the hepatocyte into the lumen of the bile capillary (phase IV of pigment metabolism) and enters the lumen of the duodenum through the biliary tract (phase V of pigment metabolism). In the small intestine, bound bilirubin is converted into urobilinogen and sterkobilinogen. Sterkobilinogen is formed from sterkobilin, which turns feces brown. Urobilinogen is absorbed by the intestinal wall and enters the liver again through the portal vein system. Here it is captured by hepatocytes and excreted into the bile capillaries. [5-6] In hepatic insufficiency, urobilinogen is not captured by hepatocytes, but is excreted in urine, oxidizing in air to urobilin. According to the pathogenesis, suprahepatic, hepatic and subhepatic jaundice are isolated. Determining the type of jaundice will be the next stage of differential diagnosis. When conducting differential diagnosis of jaundice, it is important to determine whether jaundice belongs to a particular group, and then diagnose within the group. Suprahepatic jaundice is caused by increased breakdown of red blood cells and, as a result, increased bilirubin formation, insufficient function of bilirubin uptake by the liver. These include various types of hemolytic jaundice — erythrocyte defects, autoimmune hemolytic jaundice, absorbable massive hematomas, and heart attacks. Hepatic jaundice is caused by damage to hepatocytes and, according to some reports, cholangioles. According to the leading mechanism, several variants of liver jaundice can be distinguished. In some cases, hepatic jaundice is associated with impaired bilirubin excretion and uptake, and bilirubin regurgitation. [12-13] This is observed in hepatic cell jaundice, acute and chronic hepatitis, acute and chronic hepatosis, cirrhosis of the liver. In other cases, bilirubin excretion and regurgitation are impaired. This type is noted in cholestatic jaundice, cholestatic hepatitis, primary biliary cirrhosis of the liver, idiopathic benign recurrent cholestasis, and hepatic cell lesions. Jaundice may be based on a violation of conjugation and bilirubin uptake. This is noted in enzymopathic jaundice in Gilbert and Kriegler-Nayyar syndromes. Hepatic jaundice may be associated with impaired bilirubin excretion, for example, in Dabin-Johnson and Rotor syndromes. Subhepatic jaundice occurs as a result of impaired patency of the bile ducts, the leading mechanism in this case is a violation of bilirubin excretion and regurgitation. By the nature of the blockage, subhepatic jaundice is divided into: intracanal (observed when the bile ducts are blocked

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by stones, tumors, parasites, inflammatory exudate); extracanal (caused by compression of the ducts from the outside by a tumor, echinococcus, narrowing by scars). Jaundice of this group develops as a result of increased bilirubin production and insufficient function of its capture by the liver. The main one in the genesis of these jaundices is the increased breakdown of red blood cells (hemolysis), which is why they are usually called hemolytic. The pathology of these jaundices lies mainly outside the liver. As a result of the increased breakdown of red blood cells, a large amount of free bilirubin is formed, which the liver is unable to capture. It is assumed that intracellular pigment transport is also disrupted. Due to the increased release of bilirubin, the content of urobilin bodies in feces and urine increases. The increased bilirubin content in the blood is due to the accumulation of predominantly free (indirect) bilirubin. It should be borne in mind that with massive hemolysis, hepatocytes are not always able to excrete all captured and conjugated bilirubin, as a result, the content of bound bilirubin in the blood increases slightly. [10-11] Thus, the severity of jaundice in this form depends, on the one hand, on the massiveness of hemolysis, on the other — on the functional state of hepatocytes. It is necessary to remember the possibility of a combined genesis of jaundice, which is observed in some infectious diseases (for example, leptospirosis). Hemolysis and damage to hepatocytes may be caused by the infectious agent itself or its toxins. In other cases, hemolysis may be caused by the use of a drug (quinine, sulfonamides), and the defeat of the hepatocyte by an infectious agent (hemoglobinuria fever in malaria). Thus, in some hemolytic jaundice, there may be clinical manifestations of the infectious process. To solve the problem of the suprahepatic nature of jaundice, a set of clinical and laboratory data is used. One of the main signs of overhepatic jaundice is hyperbilirubinemia due to free (indirect) bilirubin. In this regard, the bilirubin coefficient (the ratio of bound bilirubin to its total amount) is not high (less than 50%). An important clinical sign is that there is no acholia, on the contrary, there is a dark coloration of feces and urine. The content of urobilinogens in them is increased. The jaundice of the skin and sclera is moderate, the skin is usually pale (anemia as a result of hemolysis). The liver and spleen may be enlarged, but liver function is not significantly impaired. In the study of peripheral blood, there is a tendency to anemia, an increase in the number of reticulocytes as an indicator of increased erythrocyte regeneration. Sometimes a change in the shape of erythrocytes is detected (macrocytosis, microspherocytosis, sickle-shaped erythrocytes, etc.). When determining the hemolytic nature of jaundice, its type should be clarified.

It is customary to distinguish 3 types of suprahepatic (hemolytic) jaundice

I.Corpuscular hemolytic jaundice. The main causes of such jaundice may be biochemical defects of erythrocytes, for example, hereditary enzymopathy of erythrocytes (lack of glucose-6-phosphate dehydrogenase, Minkowski — Shoffar anemia), hemoglobinopathies (thalassemia, etc.), defects in the membranes of erythrocytes (paroxysmal nocturnal hemoglobinuria, or Markiafava— Mikeli disease, etc.). Jaundice is often repeated. Hemolysis of erythrocytes in individuals with glucose-6-phosphate dehydrogenase deficiency may be triggered by certain medications (quinine, sulfonamides, antipyretics). Such hemolysis is accompanied by an increase in body temperature (hemoglobinuria fever), the release of brown-brown urine with a large sediment, and anemia. This pathology is more often observed in patients with malaria. [8-9]

II. Extracorpuscular hemolytic jaundice. In such situations, increased hemolysis is caused by the action of various factors present in the blood plasma. These may be antibodies (for example, the action of isoantibodies in acute posttransfusion hemolytic anemia as a result of transfusion of incompatible blood), hemolysins of various infectious agents (viruses, leptospira, pathogens of

sepsis). It should be noted that jaundice of this type can also occur under the influence of the pathogen itself (plasmodium malaria) or under the action of hemolytic poisons (arsenic, hydrogen sulfide, etc.).

III. The third type of hemolytic jaundice is caused by an increase in bilirubin production as a result of the breakdown of red blood cells in extensive hematomas, heart attacks, hemorrhages into the abdominal or pleural cavities. Such jaundice, as a rule, does not pose much difficulty in conducting differential diagnosis, since signs of internal bleeding, clinical symptoms of heart attacks (myocardium, lung) come to the fore. Such patients may have a history of various injuries. [7-13] Hemolytic jaundice, as a rule, is caused by non-infectious causes, but sometimes the hemolytic component in mixed jaundice can develop in jaundice forms of leptospirosis, in patients with sepsis, as complications of rubella, measles, mumps. The presence of signs of an infectious disease facilitates differential diagnosis.

Differential diagnosis of hepatic jaundice is one of the difficult moments of medical practice, since the number of diseases occurring with such jaundice is quite large. The cause of liver jaundice can be both infectious and non-communicable diseases. Given the fact that among infectious diseases, hepatic jaundice is most often caused by viral hepatitis, when conducting differential diagnosis of jaundice occurring with signs of an infectious process, it is first of all necessary to exclude viral hepatitis in the patient. To exclude acute viral hepatitis, it is important to compare the severity of fever and other manifestations of general intoxication with the degree of liver damage. Viral hepatitis A is more common in young people, sometimes it occurs in the form of epidemic outbreaks in groups, therefore, it is of great diagnostic importance to identify contacts with hepatitis A patients within the time frame of the incubation period (more often 15-30 days). The duration of the pre-jaundice period is slightly shorter (more often 5-7 days) than in viral hepatitis B (more often 8-10 days). Unlike viral hepatitis B, severe pain is rarely bothered during this period. More often there is a flu—like variant of the pre-jaundice period, less often - dyspeptic and asthenovegetative. During this period, fever, weakness, headache, and decreased appetite are noted. At the end of the pre-jaundice period, the urine becomes dark and the stool discolors. The jaundice period of hepatitis A begins with the appearance of icteric sclera, mucous membranes of the oropharynx, and then the skin. The intensity of jaundice increases throughout the week. Body temperature is normal. There is weakness, drowsiness, decreased appetite, aching pains in the right hypochondrium, some patients may have itching. The liver increases, becomes dense and somewhat painful on palpation. During this period, there may be an enlargement of the spleen. In peripheral blood: leukopenia, neutropenia, relative lymphocytosis and monocytosis. [14-15] The erythrocyte sedimentation rate (ESR), as a rule, remains within the normal range. The content of total bilirubin in the blood is increased, mainly due to direct (bound), the activity of aminotransferases, especially AlAT, is significantly increased, and the parameters of the thymol sample are increased. The jaundice period lasts on average 7-15 days. The period of convalescence is characterized by the rapid disappearance of clinical and biochemical manifestations of hepatitis. Bilirubin content and aminotransferase activity usually normalize by the 20th-25th day after the onset of jaundice. Severe forms of hepatitis A are rare. In viral hepatitis A, the development of chronic forms has not been described. Sometimes there is prolonged convalescence with increased activity of AlAT within 1-2 months after the disappearance of other symptoms of hepatitis. Viral hepatitis B is transmitted in most cases parenterally and less often sexually, therefore, a detailed medical history with an emphasis on parenteral interventions during the incubation period (more

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often 60-120 days) is important for its diagnosis. The pre-jaundice period in viral hepatitis B, unlike enteric hepatitis, is longer. At this time, patients are often bothered by joint pain. In patients with posttransfusion hepatitis, as well as in severe forms, there may be an increase in body temperature at the end of the pre-jaundice period. In general, the severity of all manifestations of the pre-jaundice period is greater than in viral hepatitis A. More often, the pre-jaundice period begins with dyspeptic phenomena (decreased appetite up to anorexia, nausea, vomiting, dull pain in the right hypochondrium). Some patients may experience short-term stool disorder. Arthralgic variant is noted in 30% of patients. In such situations, there are severe aching pains in large joints, bones, and muscles, which increase at night. About 10% of patients with viral hepatitis at this stage of the disease may have an urticular rash At the end of the pre-jaundice period, urine becomes dark, and feces discolor, liver enlargement is noted, the activity of AsAT and, to a large extent, AlAT increases. The jaundice period in acute viral hepatitis B is usually very long and is characterized by the severity and persistence of clinical manifestations, which gradually increase. Jaundice reaches its maximum at 2-3 weeks (with hepatitis A, it disappears by this time). In severe forms, acute liver failure may develop in the first days of jaundice against the background of progressive deterioration of the condition. During this period, patients complain of weakness, malaise, decreased appetite, dull pains in the right hypochondrium. There may be joint pain, itching of the skin. Jaundice increases gradually. At first, jaundice is detected only upon careful examination on the sclera, soft and hard palate, and somewhat later, jaundice staining of the skin appears. Almost all patients have hepatomegaly. [12-13] As the liver recovers, the size of the liver returns to normal. A decrease in the size of the liver with increasing jaundice indicates the possibility of developing acute liver failure. An enlargement of the spleen is often noted. In acute viral hepatitis, the recovery period is longer than in hepatitis A. The condition of patients is gradually improving. As he recovers, jaundice decreases and disappears, appetite appears, the size of the liver decreases, urine lightens, feces stain. Sometimes recovery is delayed, moderate jaundice may appear again in the form of separate waves. After acute viral hepatitis B, asthenization may persist for a long time. The outcome of acute viral hepatitis B can be prolonged and chronic forms. Laboratory diagnostics, and in particular serological determination of markers and polymerase chain reaction, play an important role in the differential diagnosis of acute viral hepatitis. In other infectious diseases, jaundice does not occur in all patients, but only in more severe forms of the disease. In many of these infectious diseases, the appearance of jaundice occurs against a background of high fever and pronounced symptoms of general intoxication. In addition, various organ lesions may appear that are characteristic of a particular infectious disease, but not characteristic of viral hepatitis A and B. Consider the possibilities of differential diagnosis of the most common infectious diseases occurring with jaundice syndrome. The jaundice form of infectious mononucleosis. With infectious mononucleosis, hepatitis almost always develops, often it occurs with severe jaundice. In mononucleous acute hepatitis, there are all the signs characteristic of viral hepatitis, in particular jaundice of hepatic origin, liver enlargement, increased activity of serum enzymes - AlAT, AsAT, alkaline phosphatase, etc. However, with infectious mononucleosis, the syndrome of general intoxication is more pronounced (an increase in body temperature to 39-40 ° C, general weakness, headache, etc.), which persists and sometimes increases after the appearance of jaundice. The main difference lies in the damage to other organs and systems, which is unusual for viral hepatitis. The clinical symptoms of infectious mononucleosis are so characteristic that it allows differentiating this disease with jaundice etiology. [1-3] The main manifestations of infectious mononucleosis, which serve for differential

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diagnosis, are: fever; tonsillitis; generalized lymphadenopathy; hepatosplenomegaly; characteristic changes in peripheral blood. Fever in infectious mononucleosis lasts from 1 to 3 weeks, and sometimes longer. It does not decrease after the appearance of jaundice. Fever often reaches 38-40 ° C. Tonsillitis is an essential component of infectious mononucleosis. Its presence is of great differential diagnostic importance, since it is absent not only in viral hepatitis A and B, but also in other infectious diseases occurring with jaundice, with the exception of the anginous-septic form of listeriosis. Changes in the pharynx in infectious mononucleosis are peculiar. Necrotic changes of the palatine tonsils are characteristic with the formation of films on them, which sometimes resemble fibrin deposits in pharyngeal diphtheria. Sometimes the manifestations of tonsillitis resemble changes in the pharynx in catarrhal or follicular lacunar angina. Changes in the throat do not appear from the first day of the disease, but join fever and other clinical symptoms only on the 2nd-3rd day from the onset of the disease. Generalized lymphadenopathy is a characteristic symptom of infectious mononucleosis and only occasionally occurs in jaundice of another etiology (listeriosis, syphilis), therefore it has great differential diagnostic significance. All groups of lymph nodes are enlarged, which are moderately painful on palpation. Sometimes there is pain in the area of mesenteric lymph nodes. Enlargement of the liver and spleen is characteristic of both viral hepatitis and other infectious diseases associated with jaundice, therefore, the differential diagnostic value of this sign is low. The only thing that attracts attention is a more significant increase in the spleen compared to other diseases. This explains why one of the severe complications of infectious mononucleosis is rupture of the spleen. Such an increase in the spleen, and sometimes more pronounced, is noted only in malaria and recurrent typhus. [2-3]

Toxic hepatitis. Liver damage with the development of hepatic jaundice can be observed in various toxic hepatitis. For the differential diagnosis of infectious and toxic hepatitis, the presence of signs of an acute infectious process (fever, signs of general intoxication, exanthema, epidemiological data, etc.) is of great importance. For the diagnosis of toxic hepatitis, the use of hepatotropic toxic substances (anti-tuberculosis drugs, MAO inhibitors, phenothiazine derivatives), technical liquids (dichloroethane, ethylene glycol), occupational hazards (work with nitric acid-based oxidants, hydrazine, etc.), as well as the absence of manifestations of the infectious process are important.

Acute alcoholic hepatitis it can develop in people suffering from chronic alcoholism after another alcohol abuse, while taking into account the possibility of toxic effects of various alcohol surrogates. Unlike viral hepatitis, with alcoholic liver damage, leukocytosis, increased activity of alkaline phosphatase, higher blood cholesterol and B-lipoproteins are more often noted, bilirubin-aminotransferase dissociation is also noted (the degree of increase in AlAT slightly lags behind the level of hyperbilirubinemia). [12-15]

Drug-induced hepatitis they can be caused by many medications, but more often occur in cases where the drug is prescribed for a long time and in large doses. More than half of drug-induced hepatitis is associated with long-term use of anti-tuberculosis drugs. As a rule, hepatitis develops by the end of the 1st month from the start of the use of medicines. In addition to jaundice and liver enlargement, there may be other manifestations of the drug disease: dyspeptic disorders (nausea, vomiting, heartburn), allergic disorders (itching and burning of the skin, urticaria or hemorrhagic exanthema, lymphadenopathy, eosinophilia). Unlike viral hepatitis, which can overlap with

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tuberculosis, there is no cyclical development of the disease, there are no changes in peripheral blood typical for viral hepatitis (leukopenia, lymphocytosis, decreased ESR). The indicators of the thymol test, unlike viral hepatitis, are negative. Toxic hepatitis can develop not only after taking medications, but also as a result of occupational hazards, poisoning with technical liquids. Nitric acid-based oxidants cause liver damage and jaundice with prolonged exposure (in acute poisoning, signs of respiratory damage prevail). The disease develops gradually, patients develop general weakness, fatigue, decreased performance, decreased appetite, and headache. Against this background, abdominal pain begins, which is localized in the right hypochondrium and in the epigastric region. The liver is enlarged, the bilirubin content in the blood is moderately elevated. The course of hepatitis is relatively favorable. It should be noted that people who work with nitrogen oxides for a long time have yellowish coloring of the skin of the hands, face, and scalp, even with normal bilirubin content in the blood serum. [8-9]

Subhepatic jaundice They are caused by a violation of bilirubin excretion through the bile ducts with its regurgitation, as well as a decrease in bilirubin excretion from hepatocytes. The pathological process is localized outside the liver in the main bile ducts. Subhepatic jaundice can be caused by the following factors:

1) closure (obturation) of the hepatic and common bile ducts from the inside with gallstones, helminths, etc.;

2) compression of the common bile duct from the outside (neoplasm, enlarged lymph node);

3) narrowing of the common bile duct by scars and adhesions (after surgery, after an inflammatory process);

4) atresia (hypoplasia) of the biliary tract.

The development of subhepatic jaundice is caused by a violation of the outflow of normal bile caused by a mechanical factor, hence the name mechanical jaundice. In subhepatic jaundice, the content of bound (direct) bilirubin increases mainly and only to a small extent - free. After the occurrence of obstruction of the bile ducts, bile excretion from hepatocytes continues for the first time, bilirubin released into the lumen of the bile ducts is absorbed through their wall and through communications between the bile capillaries and Disse spaces. An increase in pressure in the biliary tract leads to secondary changes in hepatocytes, as a result, the excretory function of liver cells decreases and paracholia occurs, i.e. the mechanisms inherent in intrahepatic cholestasis are activated. An increase in the level of indirect (free) bilirubin, apparently, is associated with a violation of its capture by hepatocytes from the blood. There is no excretion of urobilin bodies with feces and urine in case of mechanical jaundice. In the group of subhepatic jaundice, the most practical importance is the differential diagnosis of diseases such as the cholestatic variant of viral hepatitis, mechanical jaundice as a result of compression of the bile ducts by neoplasms and mechanical jaundice when the biliary tract is blocked by a stone or helminths. The cholestatic variant of viral hepatitis is observed mainly in viral hepatitis B, but it is also possible in hepatitis C. In this regard, if the patient has clear anamnestic data in favor of hepatitis B (transfusion of blood and its preparations during the incubation period from 45 to 180 days, the appearance of a surface antigen of the hepatitis B virus or antibodies to it in the blood serum), then the manifestations of cholestasis can be interpreted as a cholestatic variant of viral hepatitis B. Viral hepatitis B It is observed in people of any age, whereas neoplasms and gallstone disease are more common in people over 40 years of age. Calculous cholecystitis develops mainly in women. The appearance of obstructive jaundice is sometimes preceded by a violation of the diet. In the

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anamnesis of such patients, signs of cholecystitis can be detected (dull pain in the liver, sometimes short-term jaundice of the sclera, etc.). With neoplasms, the onset of biliary tract obstruction may be preceded by chronic diseases of the stomach, pancreas, weight loss, anemia and other signs characteristic of neoplasms. The onset of the disease also has its own characteristics. [9-10] In viral hepatitis B, as a rule, there is a pre-jaundice period lasting 1-2 weeks, the onset is relatively gradual and usually cyclical. In addition to the cholestatic component, there is always a cytolytic component with moderate signs of intoxication. When the bile ducts are blocked by a stone against the background of moderate manifestations of cholecystitis, a severe pain attack suddenly occurs, after which jaundice soon appears. The pain has a characteristic irradiation in the right shoulder, shoulder blade. Nausea and vomiting may occur during a painful attack. With mechanical jaundice caused by neoplasms, the pain can be very severe, it develops gradually, starting with moderate severity, which then progresses. In viral hepatitis, the pain is moderate, dull, localized in the right hypochondrium, sometimes in the epigastric region. In the pre-jaundice period, arthralgia is characteristic of viral hepatitis B, which is completely atypical for subhepatic jaundice associated with gallstones or neoplasms.

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