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Jaundice: Symptoms, Causes, and Treatments

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ABSTRACT

Jaundice is a complicated illness present in multiple animals and humans of almost all genders and ages. In jaundice, there is yellowing of the skin, eyeball, mucus membrane and a significant increase in bilirubin. Bilirubin is a product synthesized by the breakdown of hemoglobin in red blood cells or we can also say that it is the last product of heme metabolism. Jaundice is characterized in various forms which include pre-hepatic jaundice (because of excessive production of bilirubin), hepatic jaundice (because of a decrease in intake or less excretion of bilirubin and defect in conjugation), post-hepatic jaundice (because of hindrance in extrahepatic biliary passages between liver and duodenum). Symptoms of jaundice include fever, headache, nausea, liver problems, weakness, constipation, and yellowing of mucus membranes. The causes of jaundice may be acquired or congenital. High water intake and a low-fat diet can prevent jaundice. A mixture of immunoglobulins, phototherapy, supportive therapy, and interferons treats jaundice.

1. Introduction:

Jaundice originates from the French word *jaune* which means yellow. Jaundice is characterized by the yellow color of skin, eyeballs, and mucus membranes and excess of bilirubin in body fluids. Excess of bilirubin in body fluids is known as hyperbilirubinemia [2]. Bilirubin is the molecule that is the main reason for the color of jaundice. Breakdown down of red blood cells leads to the formation of bilirubin which is excreted by the liver [15]. It is the end product of the decomposition of hemoglobin molecules. Hemolysis causes a rise in bilirubin formation and results in jaundice [7]. Normal level of bilirubin in body fluid is 5-17mmol/L (0.3-1 mg/dL). If the level of bilirubin in body fluid increases up to 50mmol/L (3mg/dL), then jaundice can be noticeable [8].

2. Symptoms:

Jaundice can strike suddenly or grow gradually over time. Symptoms of jaundice include severe weakness, fever, headache, loss of appetite, bloating, nausea, yellow eyes, skin and urine. Moreover, there is distress around the liver. Internal mouth color is yellow, dark yellow or brown color urine, and clay-colored stools. In more severe cases yellow color skin and sclera of the eye become brown. Jaundice is often accompanied by skin irritation [14].

3. Causes:

There are multiple reasons that cause hyperbilirubinemia which include utilization of medications. Some herbal medications produce harmful metabolites which affect bilirubin metabolism. If dietary supplements taken by body exceeds the level, they lead to liver disorder which ultimately affect bilirubin metabolism. Some recreational drugs promote hemolysis which leads to an increase in bilirubin concentration. Alcohol consumption can stress out the liver. Multiple blood transfusions cause a significant rise in iron concentration which leads to liver damage. After gallbladder surgery, some stone residues still present in the bile duct which are not recognized at the time of surgery can cause jaundice. Some liver disorders disturb the bilirubin metabolism. Bilirubin level rises due to excess hemolysis (breakdown of red blood cells). HIV causes liver function disorder. Revelation to toxic substances can increase the concentration of bilirubin in body fluids [13].

Jaundice is characterized as:

1. Pre-hepatic jaundice
2. Hepatic jaundice
3. Post-hepatic jaundice [1].

4. Pre-hepatic jaundice:

Pre-hepatic jaundice develops before bilirubin enters the hepatocytes. It occurs due to excessive production of bilirubin or hindrance of hepatocyte consumption by some medications. A broad range of extrahepatic disorders might be the result of pre-hepatic jaundice which include hemolysis, ineffective erythropoiesis, blood transfusion, or hematoma absorption [11]. Hemolysis may be extravascular (destruction of RBCs in the reticuloendothelial system) and intravascular (destruction of RBCs directly in blood vessels). An increase in the production of bilirubin is evidence of extravascular hemolysis [6]. Ineffective erythropoiesis is caused due to intramedullary apoptosis of megaloblastic erythroid precursors. Due to aberrant cellular membrane proteins during production, the erythrocytes are more rigid, which reduces the survival time of red blood cells. Jaundice with elevated plasma bilirubin levels arises from this inefficient erythropoiesis [4]. Hematoma absorption causes bilirubin levels to rise significantly. This can overpower the process of conjugation and result in unconjugated hyperbilirubinemia [9].

5. Hepatic Jaundice:

Hepatic jaundice is congenital or acquired hepatocellular disorders, which result in decreased intake of bilirubin from plasma, impaired conjugation, or less excretion. Congenital causes include Gilbert's syndrome, Crigler- Najjar syndrome, Rotors syndrome, and hemochromatosis [12]. Gilbert's syndrome is characterized by a mutation in the Uridine Glucuronosyl Transferase 1A1 (UGT1A1) gene promoter thus limiting the activity of UGT1A1, which permits the excretion of conjugated bilirubin from blood [10]. Crigler- Najjar is associated with biallelic mutations of UGT1A1 which results in partial or total loss of uridine 5'-diphosphate glucuronyl transferase activity which results in unconjugated hyperbilirubinemia moreover there is permanent brain damage [3]. Rotor syndrome is an uncommon, genetic illness that is associated with hyperbilirubinemia [5]. Hemochromatosis is the continuous loading of iron which influences the liver [18]. Acquired hepatocellular disorders include viral hepatitis (e.g. Hepatitis A, B, C, and E), autoimmune disorders, sepsis, alcohol, non-alcoholic fatty liver disease, trauma or malnutrition [12]. Sepsis frequently results in cholestasis, which is infection of the biliary tree or common bile duct that is accompanied by a sharply raised bilirubin level. Hepatocyte dysfunction and hemolysis can also cause hyperbilirubinemia [16].

6. Post-hepatic jaundice:

In post-hepatic jaundice, there is obstruction of the extrahepatic biliary passages between the liver and duodenum, either partially or completely and results in resistance of bile movement. It might be caused due to surgical strictures, Bile stones, Bile duct cancers, or compression of the bile duct. Bile stones block the passage of bilirubin excretion. In addition, bile duct cancers and compression of the bile duct are responsible for the increase in bilirubin levels. [17].

7. Treatment:

Treatment is dependent on the cause of jaundice. The best way to prevent jaundice is a high-water intake and low-fat diet. A blend or mixture of immunoglobulins gives benefits in treatment of pre-hepatic jaundice, as well as phototherapy has the potential to treat increased level of bilirubin in pre-hepatic jaundice. Supportive therapy such as fluids, rest, and pain relief medicines are utilized in the treatment of hepatitis A. Interferons are used in the treatment of chronic hepatitis B and C. A person with post-hepatic jaundice should take a low-fat diet to reduce restlessness from diarrhea and fat ingestion [1].

8. Conclusion:

Jaundice can be identified by the yellowing of skin, eyeballs, and mucus membrane which is caused by the increased level of bilirubin or defect in its metabolism and excretion. Jaundice is classified as pre-hepatic jaundice, hepatic jaundice, and post-hepatic jaundice. The causes of jaundice can be acquired or congenital. Jaundice can be prevented by high water intake and a low-fat diet. Treatment depends upon the cause of jaundice.

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