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Perspective

Various Pathophysiological Phases in Jaundice

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ABOUT THE STUDY

High bilirubin levels cause jaundice, also known as icterus, which is a yellowish or greenish discoloration of the skin and sclera. Adults with jaundice frequently have underlying conditions that involve aberrant heme metabolism, liver malfunction, or biliary tract obstruction. While jaundice in neonates is frequent, with an estimated 80% of cases occurring in the first week of life, it is uncommon in adults. Itching, pale faeces, and dark urine are the three symptoms of jaundice that are most frequently seen.

Blood bilirubin concentrations under 1.0 mg/dl are considered normal, while concentrations over 2–3 mg/dl frequently cause jaundice. Unconjugated and conjugated bilirubin are the two kinds of high blood bilirubin.

High levels of unconjugated bilirubin can result from excessive red blood cell oxidation, severe bruises; hereditary disorders such gilbert's syndrome, prolonged fasting, infant jaundice, thyroid issues, or huge bruises.

High conjugated bilirubin levels can result from infections, drugs, or bile duct blockages brought on by conditions like pancreatitis, cancer, or gallstones. They can also be caused by liver conditions like cirrhosis or hepatitis. Jaundice is not the only disorder that can result in yellow skin, such as carotenemia, which can be brought on by taking drugs like rifampin or consuming a lot of foods containing carotene.

The underlying cause of jaundice usually dictates how it should be treated. Surgery is usually necessary if there is a bile duct obstruction; otherwise, medication therapy is used.

Treatment of infectious causes and discontinuation of medications that might be causing the jaundice are possible medical care strategies. When the bilirubin is greater than 4-21 mg/dl, jaundice in neonates may be treated with phototherapy or exchange transfusion depending on age and prematurity.

Ursodeoxycholic acid, draining the gallbladder, or opioid antagonists like naltrexone may all reduce itching. Jaundice is derived from the French word jaunisse, which means "yellow illness."

PATHOPHYSIOLOGY

Jaundice is often brought on by an underlying pathological event that takes place anywhere along the heme metabolism's regular physiological pathway. To grasp the significance of prehepatic, hepatic, and posthepatic categories, one must have a deeper understanding of the anatomical flow of normal heme metabolism. Thus, a description of the pathophysiology of jaundice comes before an anatomical approach to heme metabolism.

Normal heme metabolism

Prehepatic metabolism: Red blood cells break as they move through the reticuloendothelial system when they reach the end of their lifespan (about 120-day) or if they sustain damage, releasing their contents, including haemoglobin, into the bloodstream. Heme and globin are separated from free haemoglobin by macrophage phagocytosis. The heme molecule then experiences two reactions. The microsomal enzyme heme oxygenase catalyses the first oxidation step, which yields biliverdin (a green pigment), iron, and carbon monoxide. The cytosolic enzyme biliverdin reductase then converts biliverdin into the yellow tetrapyrrole pigment known as bilirubin.

Each day, around 4 mg of bilirubin per kilogram of blood are created. The majority of this bilirubin is produced during the process just mentioned when the heme from depleted red blood cells is broken down. The breakdown of other heme containing proteins, such as muscle myoglobin and cytochromes, and inefficient erythropoiesis account for about 20% of the total. The unconjugated bilirubin subsequently circulates *via* the blood to the liver. This bilirubin is carried through the blood attached to serum albumin because it is not soluble.

Hepatic metabolism: Unconjugated bilirubin is converted to bilirubin diglucuronide by the liver enzyme glucuronyl transferase once it has entered the liver (conjugated bilirubin). The liver converts bilirubin into a water-soluble compound that is expelled in the gallbladder.

Posthepatic metabolism: Through bile, bilirubin enters the digestive system. Symbiotic intestinal bacteria in the intestine

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transform bilirubin into urobilinogen. The majority of urobilinogen undergoes conversion into stercobilinogen and subsequent oxidation to become stercobilin. The substance stercobilin, which gives stool its distinctive brown colour, is expelled in faeces. Urobilinogen is briefly reabsorbed into the cells of the digestive tract. Hepatobiliary recirculation occurs in the majority of reabsorbed urobilinogen. The kidneys filter a lesser amount of reabsorbed urobilinogen. Urobilinogen is transformed into urobilin in the urine, giving urine its distinctive yellow hue.

Abnormalities in heme metabolism and excretion

Jaundice pathophysiology can be organized into illnesses that result in either decreased bilirubin elimination or increased bilirubin synthesis (abnormal heme metabolism) (abnormal heme excretion).

Prehepatic pathophysiology: Prehepatic jaundice is caused by a pathological increase in bilirubin production. As a result of increased erythrocyte hemolysis, which raises bilirubin synthesis, more bilirubin is deposited in mucosal tissues, giving the skin a yellow tint.

Hepatic pathophysiology: A severe disruption in liver function causes hepatic cell death and necrosis as well as decreased bilirubin transport across hepatocytes, resulting in hepatic jaundice (also known as hepatocellular jaundice). Between hepatic uptake of unconjugated bilirubin and hepatocellular transport of conjugated bilirubin into the gallbladder, bilirubin transport across hepatocytes may be impeded at any stage. Additionally, the intrahepatic biliary duct becomes mechanically obstructed as a result of subsequent cellular edoema brought on by inflammation. Hepatocellular jaundice is most frequently associated with interferences in all three of the main processes of bilirubin metabolism: absorption, conjugation, and excretion. As a result, both conjugated and unconjugated bilirubin will increase abnormally.

Posthepatic pathophysiology: Obstructive jaundice, also known as posthepatic jaundice, is brought on by an obstruction in the biliary path, which results in higher levels of conjugated bilirubin and bile salts. Complete blockage of the bile duct prevents conjugated bilirubin from reaching the digestive tract, preventing further bilirubin conversion to urobilinogen, and as a result, preventing the production of stercobilin or urobilin. Excess conjugated bilirubin is passed into the urine without the presence of urobilinogen in obstructive jaundice. Urine with conjugated bilirubin (bilirubinuria) has an unusually dark brown colour. Because stercobilin is absent from the stools and conjugated bilirubin is present in the urine, the presence of pale stool and dark urine points to an obstructive aetiology of jaundice.