For General Practitioners:

THE PROBLEM OF JAUNDICE IN THE NEWBORN

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In the newborn, jaundice is frequently observed. It may be physiological jaundice or the result of some underlying disease. The two main problems one faces while dealing with a case of neonatal jaundice are, differentiation of physiological jaundice from one due to underlying disease and prevention of kernicterus. The important conditions responsible for jaundice are discussed below.

Physiological Jaundice

Bilirubin is formed by the destruction of red blood cells in the spleen. This pigment is unconjugated and gives an indirect van den Bergh reaction. It is transported to the liver bound to the albumin component of blood proteins. In the liver it is conjugated with glucuronic acid to form bilirubin diglucuronide which gives a direct van den Bergh reaction. For this process of conjugation an enzyme glucuronyl transferase is needed. In the newborn this enzyme is not formed adequately by the liver, resulting in accumulation of non-conjugated bilirubin in blood and therefore jaundice. Since the occurrence of this is seen even in normal newborns and the resulting jaundice is transient, it is termed "physiological." From numerous careful studies the following arbitrary limits are laid down for physiological jaundice. In the majority of cases it appears between the second and the fourth day. In a full term newborn, jaundice within the first twenty-four hours and in the premature that within forty-eight hours, is never physiological. It disappears by the seventh day of life. Visible jaundice in the second week is not likely to be physiological. Serum bilirubin level varies between 2 to 12 mg% with an average of 7 mg%.

Physiological jaundice may be prolonged in the presence of certain disorders, e.g. in cretins, babies of diabetic mothers, hypoxia, hematomas and hypertrophic pyloric stenosis. At times prolonged jaundice may be a valuable clue for suspecting one of these conditions.

Hyperbilirubinemia in some breast-fed newborn babies has been reported. It appears in the second week of life and is due to the secretion of a substance in the milk which interferes with conjugation of bilirubin in the liver. This disappears on stopping breast feeding.

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Iatrogenic Jaundice

Jaundice may be iatrogenic. It has been reported that large doses of vitamin K analogues may increase the serum bilirubin to a dangerous level. This happens through three mechanisms: hemolysis, toxicity on hepatic cells and competition for the conjugating enzyme. If there is an indication for giving vitamin K, the dose should not exceed 2.5 mg and it should be given in its natural form rather than its analogues. Other drugs which have been responsible for hyperbilirubinemia are sulphamethoxine (Madribon), sulphamethoxypyridazine (Lederkyn), sulfasaxazole (Gantrasin) and Novobiocin.

Hemolytic Disorders

Isoimmunisation. The hemolytic process due to isoimmunisation as a result of blood group incompatibilities between the mother and the fetus is well known. The blood group system involved may be Rh, ABO and others like Kell, Duffy and Kidd. Antibodies from the mother cross the placental barrier and enter the fetal circulation where they destroy fetal red blood cells. The hemolysis results in anemia and a raised bilirubin level. The pigment is successfully tackled by the placenta but when the child is born the load falls upon the immature and inefficient liver resulting in hyperbilirubinemia. A history of previously affected siblings, jaundice appearing within 24 hours, anaemia and a palpable spleen should make one suspicious and further investigations should be undertaken.

Spherocytosis. Occasionally hemolysis may be due to spherocytosis. Hemoglobinopathies are not responsible for jaundice at this age.

Infection. Sepsis is frequently associated with jaundice in the newborn. The mechanisms responsible are multiple. From the various studies available it seems that sepsis may be playing a triple role: increased hemolysis, hepatocellular damage and depression of the conjugating mechanism. Jaundice usually appears after the 3rd or 4th day. The infant is lethargic, feeds poorly, anemia is present and liver and spleen are palpable. Fever and leucocytosis may be absent.

Congenital syphilis. Severe involvement of the liver in congenital syphilis is associated with jaundice, anemia and hepatosplenomegaly. Other evidence of congenital syphilis in the form of snuffles, cutaneous lesions, etc. will be present. X-rays of the knees will show evidence of osteochondritis and periostitis and serological tests may be positive.

Toxoplasmosis may manifest itself as jaundice in the newborn. There is hepatosplenomegaly, retinal pigmentation and intracranial calcification. Though rare it has been reported in India.

Virus infections like cytomegalic inclusion disease and herpes simplex hepatitis may present as jaundice. These disorders run an acute course and may be fatal.

Giant cell hepatitis. Jaundice is the chief sign which may be apparent at birth but usually appears after a week. The liver and spleen are palpable. Fever may be present and the child may look ill. Feeds are accepted poorly. Later on, it may present a picture vary much like obstructive jaundice,