INTRODUCTION

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The problem of the jaundiced newborn infant confronts pediatricians all over the world. In the vast majority of cases, jaundice is secondary to a mild and transient disturbance of bilirubin metabolism that does not require special treatment. Increased red blood cell (RBC) destruction has always been considered as the most important factor responsible for neonatal indirect hyperbilirubinemia.[176] Reports concerning the relative contribution of feto-maternal blood group incompatibility among jaundiced babies vary considerably from one locality to another.[42,82,139] In some areas as Western Europe, the prevalence of the Rh negative individuals may be as high as 20% of the total population.[33,144] In such localities and before the introduction of immunoprophylactic therapy, Rh incompatibility was considered as a major cause of hemolytic disease and kernicterus among newborn infants. [33,144] In other areas the Rh problem does not seem to have the same impact as the prevalence of Rh negative individuals there is low. [33,144] The ABO hemolytic disease of the newborn and other feto-maternal subgroup incompatibilities show a similar variability in their prevalence in different parts of the world.[42,82,139,144] In the Newborn Unit of Security Forces Hospital, neonatal jaundice was encountered in 48% of the total admissions during the year 1996-1997.[160] The Rh hemolytic disease was not detected in the jaundiced babies, while ABO feto-maternal blood group differences were found in 17% of them.[160]

Many other hemolytic conditions have been reported in connection with neonatal unconjucated hyperbilirubinemia (Table I).[145] Hereditary

TABLE I

Causes of neonatal unconjugated hyperbilirubinemia[145]

A. HEMOLYTIC DISORDERS

1. Feto-maternal blood group incompatibility:
   "Rh, ABO & others".

2. Genetic disorders:
   a) Spherocytosis
   b) Enzyme defects e.g. G6PD, PK.
   c) Hemoglobinopathies:
      α thalassemia
      Sickle cell disease
   d) Galactosemia

3. Drug induced hemolysis:
   e.g. Vitamin K

B. EXTRAVASCULAR BLOOD

Petechiae, hematoma, haemorrhage, swallowed blood.

C. POLYCYTHEMIA

1. Chronic fetal hypoxia.
2. Materno-fetal or feto-fetal transfusion.
3. Placental transfusion "cord stripping".

D. EXAGGERATED ENTEROHEPATING CIRCULATION

1. Mechanical Intestinal obstruction "atresia, stenosis, meconium plug or ileus... etc.
2. Reduced peristalsis "fasting or underfeeding".

E. DECREASED HEPATIC UPTAKE

1. Persistent ductus venosus.
2. "Y" protein block e.g. abnormal human milk inhibitor (NEFA)

F. DECREASED BILIRUBIN CONJUGATION

1. Glucuronyl transferase deficiency
   a) Familial non hemolytic jaundice
   b) Gilbert's syndrome

2. Enzyme inhibitor
   a) Drugs & hormones "novobiocin, pregnandiol"
   b) Lucey-Driscoll syndrome.

hemoglobinopathies and abnormalities of the red cell membrane or shape contribute little to neonatal indirect hyperbilirubinemia. Red blood cell enzymopathies have been held responsible for variable degrees of RBC destruction and jaundice in new born infants. In many reports, deficiency of the erythrocyte enzyme glucose 6-phosphate dehydrogenase (G6PD) has been considered as the leading enzyme deficiency responsible for neonatal indirect hyperbilirubinemia and even hydrops fetalis.[51,75,121,162,175] It has been estimated that this red cell enzyme disorder affects millions of people throughout the world.[54] The prevalence of the enzyme deficiency, however, shows wide variation from one locality to another.[53,66,149,155,162,175,181] Moreover, increased RBC destruction and jaundice do not occur in every individual with decreased enzyme activity and hence, the reported enzyme deficiency rates are not equal to those related to neonatal hyperbilirubinemia.[53,66,149,155,162,175,181]