However, the clinical implications of PMN G6PD deficiency still appear unclear. Rodey *et al(6)* in their study of PMN G6PD levels and bactericidal activity concluded that the susceptibility to infection was probably not a function of the total quantity of the enzyme. Further the increased susceptibility to sepsis may be due to increased iron concentration caused by erythrocyte hemolysis(4). The clinical profile of infections in G6PD deficient neonates include: *(i)* A male predominance; *(ii)* Late sepsis (sepsis >72 hours of age): the explanation for this lies in the fact that there is normally a decline in G6PD activity corresponding to the fall in the leucocyte count around the first week of life. This is accentuated in babies with deficiency(4); and *(iii)* Infection with catalase positive organisms, *viz.,* klebsiella, enterobacter, *E.coli* and *S. epidermidis(1).*

The above features were classically seen in our patient who was a male and developed catalase positive (Klebsiella) septicemia on the seventh day of life. A study of this case and review of relevant literature suggests that G6PD deficiency is a risk factor for severe infections with catalase positive organisms in the newborn period. It thus seems imperative that infants with G6PD deficiency be aggressively treated for these infections.

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Etiology of Neonatal Jaundice at Shimla

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The etiological factors responsible for neonatal jaundice are likely to be affected by the population studies(1), gestational

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age, sex(2), maternal medication(3), type of feeding(4) and the altitude(5,6). In a number of cases, however, even the most sophisticated investigations fail to reveal any etiological factors and these cases are then labelled as 'idiopathic'. The frequency of such cases varies depending upon the investigative facilities available. Recently, an association has been reported between the increased incidence of neonatal hyperbilirubinemia and high altitude, in studies conducted at height of 3100 meters in Colorado. The present study was undertaken to evaluate the etiological factors responsible for neonatal jaundice in Shimla (altitude 2000 meters above sea level).

Material and Methods

We studied 164 neonates admitted to the Department of Pediatrics, Indira Gandhi Hospital, Shimla and the Neonatology Unit at Kamla Nehru Hospital, Shimla. Out of these, 109 (66.4%) were boys. Each baby was observed at least twice a day in diffuse daylight for detection of jaundice. Serum bilirubin estimation was done once jaundice was clinically observed. A detailed clinical examination was done and antenatal, natal and family history obtained. Jaundice was considered to be physiological when serum bilirubin was less than 12 mg/dl in fullterm and less than 15 mg/dl in preterm neonates with jaundice appearing on second or third days of life and subsiding in 7-10 days. Bilirubin level above this was labelled as hyperbilirubinemia. Breast milk jaundice was considerd to be present when in the breast fed babies, unconjugated bilirubin increased between 4-7 days of life in the absence of any other identifiable cause and jaundice subsided when breast feeding was stopped. Investigations included estimation of level of serum bilirubin, hemoglobin, total and differential leucocyte counts, reticulocyte count, peripheral smear examination, G-6-PD assay, direct Coomb's test and blood grouping of baby and mother. Anti-D antibody titre estimation, cultures of blood, X-rays, liver biopsy and serology for *Toxoplasma* and *Treponema pallidum* were done wherever required.

Results

Of 164 neonates, 105 (64%) developed clinical jaundice. Hyperbilirubinemia was present in 38 (23.1%) cases only. Out of 105 jaundiced babies, 68 (64.8%) were boys and 37 (35.2%) girls. Eighty five (81%) were fullterm and 20 (19%) were preterm.

The etiological factors incriminated are summarized in *Table I.* Physiological jaundice was seen in 63.8% cases. Of these 19.4% were preterm and 80.6% were fullterm neonates. Sepsis associated with neonatal jaundice was found in 11 (10.5%) of our cases. Out of these, jaundice was the presenting complaint in only 4 (36.7%) cases. The remaining were brought with complaints of lethargy, poor feeding, fever, difficulty in respiration and convulsions. Total serum bilirubin levels in these babies

TABLE I- *Etiological Factors in 105 Cases of Neonatal Jaundice*

	Number (n=105)	Percentage
Physiological jaundice	67	63.8
Sepsis	11	10.5
ABO incompatibility	5	4.7
G-6-PD deficiency	3	2.9
Breast milk jaundice	3	2.9
Rh incompatibility	2	1.9
Cephalhematoma	2	1.9
Idiopathic	12	11.4

ranged from 11.4 mg/dl to 20.0 mg/dl with conjugated bilirubin being 7.2 mg/dl to 12.6 mg/dl. The jaundice on an average lasted for 13.6 days in these babies with the duration of jaundice being longer in preterm (14.5 days) than in term (12.3 days) babies. Seven (63.6%) cases were blood culture positive. Jaundice was attributable to ABO blood group incompatibility in 5 (4.8%) cases. History of previous still births was present in 2 (40%) of the mothers. Onset of jaundice varied from 18-48 hours after birth and the peak was seen on 5-8 (mean 6.2) days of life. Blood group O-A incompatbility was observed in 3 (60%) and O-B incompatibility in 2 (40%) of the mother child pairs. Predominantly un-conjugated hyperbilirubinemia with serum bilirubibin ranging from 16 mg/dl to 26 mg/dl was seen and the jaundice lasted from 11-18 (mean 13.8) days in these neonates.

Jaundice because of G-6-PD deficiency was found in 3 (2.9%) cases. All of them were males. Onset of jaundice varied from 24-60 hours (mean 44 hours) after birth. Serum bilirubin in these babies varied from 16.0-21.2 mg/dl with peak of jaundice being attained on 6-8 days of life and jaundice disappeared by 11-14 (mean 12.3) days of life.

Breast milk jaundice was found in 3 (2.9%) of the babies in the present series. Two of them were jaundiced upto legs and one upto trunk. Jaundice was first detected 172-196 hours after birth. Predominantly unconjugated hyperbilirubinemia with peak serum bilirubin levels of 14.7-16.0 mg/dl was seen. The jaundice in these babies lasted for 15-16 (mean 16.3) days of life.

Rh incompatibility accounted for 2 (1.9%) of our cases. History of previous still births was present in one of the mothers. Jaundice developed within 24 hours after birth in both the cases. One neonate with serum bilirubin 28 mg/dl died within 12 hours of hospitalization.

Large cephalhematoma leading to neonatal jaundice was found in another 1.9% of our cases. Jaundice appeared 48-96 (mean 72) hours after birth in these cases and peak serum bilirubin values of 16 mg/dl and 12.9 mg/dl were observed.

Despite intensive investigations, no cause responsible for jaundice could be established in 12 (11.4%) of our cases. Peak serum bilirubin values ranged from 12.7-16.7 mg/dl, being 14.4 mg/dl in term and 15.7 mg/dl in preterm neonates. The jaundice in these neonates lasted for 8-14 days of life.

Discussion

The overall incidence of neonatal jaundice as reported by various Indian workers varies from 54.6% to 77%(2,7,8) which is similar to the present study. In the previous studies (Jone in Lucknow(7), Bombay(9) and Pune(2), the incidence of physiological jaundice was observed to be 57%, 25.3% and 47.2%, respectively as against ours of 63.8%. However, our study can not be compared directly with other two reports(2,9) as both of these studies are based upon a selective group of hyperbilirubinemic neonates.

Recently, Moore *et al.* (5) and Leibson *et al.* (6) have reported a high incidence of hyperbilirubinemia in neonates born at 3100 meters in Colorado. They have found it to be four times the incidence of hyperbilirubinemia at sea level and twice the incidence at 1600 meters altitude. However, we could not establish a similar relationship. Controlled comparative, multicenter studies are further required before such a relationship between high altitude and the incidence of neonatal jaundice is established.

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Cord Blood Cholesterol in Term and PreteTrm Newborns

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Increasing awareness about the origins of the atherosclerosis in early life has renewed interest in determination of various lipid fractions in the pediatric age group. Atherosclerotic cardiovascular diseases are the major causes of morbidity and mortality in adult population of the industrialized societies. Several investigators believe that the atherosclerotic lesions may have its genesis in the childhood(l). Studies(2) have suggested that hypercholesterolemia can be diagnosed at birth by estimation of total cholesterol or low density lipoproteins (LDL) in umbilical cord blood.

Cord blood cholesterol estimation is logistically feasible because of the ease with which cord blood can be obtained at birth and its simple method of detection(3). The present study was undertaken to determine the normal values of umbilical cord blood cholesterol in the local population and its

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