Neonatal Jaundice: An Analysis of 551 Cases

Anil Narang
Geeta Gathwala
Praveen Kumar

Jaundice is a common neonatal problem. Since bilirubin is potentially toxic to the central nervous system, early detection and appropriate management of neonatal jaundice (NNJ) is of paramount importance, especially when bilirubin even in physiological ranges may cause permanent neuronal injury. We, therefore, undertook the present study to assess the incidence, severity, causes and therapeutic interventions for jaundice in our inborn babies.

Subjects and Methods

All babies born at Nehru Hospital, Postgraduate Institute of Medical Education and Research, Chandigarh between April 1994 and June 1995, and admitted to the newborn unit with a diagnosis of NNJ irrespective of other associated illnesses were studied. Serum bilirubin was monitored 12 hourly for all babies and after 2 hours and 6 hours following all exchange transfusions. A complete hemogram including reticulocyte count, blood grouping for mother and baby, direct Coomb's test and G6PD estimation were done as a part of jaundice work up for all babies. Cockington's charts were used as guidelines for the therapeutic interventions of phototherapy (PT) and exchange transfusion (ET)(1).

All babies with NNJ and needing either PT/ET were divided into 2 groups: *(i) Group I*-those with total serum bilirubin (TSB) of ≤ 15 mg/dl but needing PT/ET; and *(ii) Group* U-those with TSB > 15 mg/dl and needing PT/ET.

Results

Of 3791 live births, 551 (14.5%) developed NNJ needing therapeutic intervention, *i.e.,* either PT or ET. Three hundred and two (7.9%) of these had a maximum TSB of ≤ 15 mg/dl and 249 (6.56%) had a TSB > 15 mg/dl. Four hundred and eighty two out of 1400, *i.e.,* 34.5% of low birth weight babies (< 2500 g) developed significant NNJ needing PT/ET; 19.5% had a TSB ≤ 15 mg/dl and 15% had a TSB > 15 mg/dl. Among the very low birth weight (<1500 g), 162 out of 247 (65.6%) developed signifi cant jaundice; 48.5% had TSB ≤ 15 mg/dl and 17.2% had a TSB > 15 mg/dl. Of babies < 2500 g, 7.52% developed jaundice need ing intervention; 1.52% had a TSB > 15 mg/dl and 5.9% had a TSB ≤ 15 mg/dl. Amongst preterm babies 440 out of 917 (47.9%) developed jaundice; 26.1% had a TSB ≤ 15 mg% and 21.8% had a TSB > 15 mg%. Of the term babies only *7.7%* (2.5% ≤ 15 mg/dl) developed significant jaundice. Among small for date babies (< - 2SD) 52 out of 124 (42%) developed significant jaundice. There was a male predominance with 56.2% of cases in Group I and 64.2% of cases in Group II being males. The peak TSB was more than 20 mg/dl in 1.5% of live births.

The commonest cause of jaundice in both groups was idiopathic followed by G6PD deficiency and sepsis *(Table I).* These

From the Neonatal Division, Department of Pediatrics, Postgraduate Institute of Medical Education and Research, Chandigarh 160 012.

Reprint requests: Dr. Anil Narang, Head, Newborn Unit, Department of Pediatrics, Post Graduate Institute of Medical Education and Research, Chandigarh 160 012.

Manuscript received: June 14,1996;
Initial review completed: July 15,1996;
Revision accepted: October 22,*1996*

TABLE I - *Causes of Jaundice.*

Cause	Group I (TSB ≤ 15 mg/dl) (n=260) No. (%)	Group II (TSB > 15 mg/dl) (n=242) No. (%)
Rh isoimmunization	7 (2.6)	7 (2.9)
ABO incompatibility	2 (0.7)	15 (6.1)
G6PD deficiency	43 (16.5)	43 (17.7)
Sepsis	31 (11.9)	18 (7.4)
Idiopathic	126 (48.4)	140 (57.8)
Oxytocin	11 (4.2)	2 (0.8)
Extravasation	20 (7.6)	12 (4.9)
Polycythemia	20 (7.6)	5 (2.06)

were also the three commonest causes in SFD babies. Sixty five per cent of the babies with idiopathic jaundice in Group II (TSB > 15 mg/dl) were on breastfeeds. The peak TSB in them was 17.77 ± 2.07 mg/dl.

A total of 141 (3.7% of total live births) babies underwent double volume exchange transfusions (102 in Group II and 39 in Group I). A total of 225 exchange transfusions (5.9 ET per 100 live births) were done. One hundred and sixty two of these were done in Group II and 63 in Group I. There were 56 babies who underwent 2 or more exchange transfusions. *Table II* summarizes the causes for exchange transfusion. Idiopathic jaundice, sepsis and G6PD deficiency were the most frequent causes needing exchange transfusions. Repeat exchange transfusions were needed most frequently for babies with Rh isoimmunization followed by those with sepsis and G6PD deficiency. Donor blood G6PD deficiency was responsible for repeat exchange transfusion in 8 cases.

Mortality due to exchange transfusion defined as death occurring within 6 h of the procedure did not occur in any subject. Four babies (1.7% of ET) were documented to have complications due to exchange transfusion. One of these, a preterm subject of 29 weeks gestation with a birth weight of 1250 g developed apnea, hyperkalemia and a bleeding diathesis, two hours following exchange transfusion done with blood that was 4 days old. Another preterm baby (30 weeks gestation, 1400 g), developed neonatal necrotizing enterocolitis 24 h following an exchange transfusion that was done through the umbilical route. Both these babies died within the next 48 h (mortality

TABLE II- *Causes of Jaundice Needing Exchange Transfusion.*

Cause	No.	%
Rh isoimmunization	13	9.2
ABO incompatibility	8	5.6
G6PD deficiency	24	17.2
Sepsis	34	24.1
Idiopathic	50	35.4
Oxytocin	4	2.8
Extravasation	2	1.4
Polycythemia	6	4.2
	141	100%

attributable to ET = 0.8% of ET). A term baby, birth weight 2.3 kg developed cardiac arrest during the exchange transfusion and was successfully revived. The fourth baby developed pallor and ischemia of the right root during exchange transfusion through the peripheral route (right post tibial artery cannulated). This reversed on removing the arterial line.

Discussion

The 6.5% incidence of pathological hyperbilirubinemia (TSB >15 mg/dl) seen in the present study is in accordance with other reports(2,3). The incidence of pathological jaundice in babies <2500g was 15% and in those ≥2500 g was 5.9%. The same has been reported as 16.6% and 2.9% for Caucasian infants and 7.9% and 1.9% for African-American infants(4). Male gender is a known risk factor for pathological jaundice(5). In our series, 64.2% of those who developed pathological hyperbilirubinemia were males. A maximal total serum bilirubin of more than 20 mg/dl occurred in 1.5% of total live births in the present series. This is similar to the 1.32% reported by others(4).

In more than half (57.8%) of the cases with neonatal hyperbilirubinemia, no cause could be identified. Various reports from our country have revealed that idiopathic hyperbilirubinemia ranges between 8.8 to 57.6%(2,6-9). In the idiopathic group, 65% of babies were receiving exclusive breastfeeding. In earlier reports, this frequency has been cited as 77.9%(2) and 82.7%(10). Breastfeeding leads to substantial elevation of bilirubin levels during the first few days of life.

G6PD deficiency was the second commonest cause of hyperbilirubinemia and accounted for 17.7% of all cases. In a report from Delhi, this figure was 5.1%(2). The higher incidence of G6PD deficiency as a cause of hyperbilirubinemia in our babies may relate to the higher prevalence of G6PD deficiency in the population being catered to by our institution. ABO incompatibility and Rh-isoimmunization accounted for 6.1% and 2.9% of cases in our study respectively. This is lower than earlier reports(2,9,10).

The most common cause of hyperbilirubinemia needing ET was idiopathic followed by sepsis and G6PD deficiency. The maximum number of exchanges were done for Rh isoimmunization, followed by sepsis and G6PD deficiency. Rh isoimmunization, ABO isoimmunization, G6PD deficiency and sepsis are the reported commonest causes needing ET(2,11). The incidence of major complications was 1.7 per 100 exchange transfusions compared to 0.95 per 100 procedures reported by others(12).

Exchange related mortality has been defined as unexplained death during or within six hours of the procedure(13). In the present study, no death occurred within 6 h of the exchange although two deaths occurred within 48 h of exchange transfusion in babies of 29 and 30 weeks gestations, presumably due to infection and/or bleeding. These deaths in very premature babies, though temporally related, may have been due to underlying infection and exchange transfusion was only an associated event. The mortality rate reported earlier was 3.2 per 100 ET(ll). As in the present study, no death was reported within 6 h of the ET from Delhi(2).

In conclusion, significant neonatal juandice occurred in 14.56% of all births with an incidence nearly three times higher in LBW babies compared to babies above 2500 g. Almost half (48%) of all preterms and 42% of SFD babies developed significant jaundice needing ET/PT. Idiopathic, G6PD deficiency and sepsis were the com-

monest ascribed causes of NNJ. G6PD deficiency occurred in over one sixth of the babies. Exchange transfusion in our experience is a safe procedure, except in very small babies with associated serious illnesses. These babies, therefore, need to be closely monitored for jaundice in the first few days of life.

REFERENCES

1. Cockington RA. A guide to the use of phototherapy in the management of neonatal hyperbilirubinemia. J Pediatr 1979; 95: 281-285.
2. Singhal PK, Singh M, Paul VK, Deorari AK, Ghorpade MG. Spectrum of neonatal hyperbilirubinemia: An analysis of 454 cases. Indian Pediatr 1992; 29: 319-325.
3. Maisels MJ. Jaundice. *In:* Neonatology: Pathophysiology and Management of Newborn. Ed. Avery GB. Philadelphia, J P Lippincott Company, 1994, pp 630-725.
4. Hardy JB, Drage JS, Jackson EC. The first year of life: The collaborative perinatal project of the National Institutes of Neurological and Communicative Disorders and Stroke. Baltimore, John Hopkins University Press, 1979, p 104.
5. Maisels MJ, Clifford K, Antle CE. Jaundice in the healthy newborn infant: A new approach to an old problem. Pediatrics 1988; 81: 505-511.
6. Anand VR, Magotra ML. Neonatal jaundice: Its incidence and etiology. Indian Pediatr 1978; 15:155-160.
7. Bajpai PC, Mishra PK, Agarwal M, Engineer AD. An etiological study of neonatal hyperbilirbinemia. Indian J Pediatr 1971; 38: 424-429.
8. Merchant RH, Merchant SM, Babar ST. A study of 75 Cases of neonatal jaundice. Indian Pediatr 1975; 12: 889-894.
9. Mohapatra SS, Menon PSN, Bhan MK, Dhar V. Cockington nomogran as a guide to phototherapy in the management of neonatal hyperbilirbinemia: Evaluation of Indian neonates. Indian Pediatr 1984; 21: 229-233.
10. Maisels MJ, Clifford K. Neonatal jaundice in full term infants: Role of breastfeeding and other causes. Am J Dis Child 1983; 137: 561-562.
11. Dikshit SK, Gupta PK. Exchange transfusion in neonatal hyperbilirubinemia. Indian Pediatr 1989; 26:1139-1145.
12. Hovi L, Siimes MA. Exchange transfusion with fresh heparinized blood is a safe procedure: Experiences from 1069 newborns. Acta Pediatr Scand 1985; 74: 360-365.
13. Boggs W. Mortality of exchange transfusion. Pediatrics 1960; 26: 745-755.