Neonatal Jaundice: An Analysis of 551 Cases

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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 significant 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 needing 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% (≤ 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
TABLE I - Causes of Jaundice.

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

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.

<table>
<thead>
<tr>
<th>Cause</th>
<th>No.</th>
<th>%</th>
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</thead>
<tbody>
<tr>
<td>Rh isoimmunization</td>
<td>13</td>
<td>9.2</td>
</tr>
<tr>
<td>ABO incompatibility</td>
<td>8</td>
<td>5.6</td>
</tr>
<tr>
<td>G6PD deficiency</td>
<td>24</td>
<td>17.2</td>
</tr>
<tr>
<td>Sepsis</td>
<td>34</td>
<td>24.1</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>50</td>
<td>35.4</td>
</tr>
<tr>
<td>Oxytocin</td>
<td>4</td>
<td>2.8</td>
</tr>
<tr>
<td>Extravasation</td>
<td>2</td>
<td>1.4</td>
</tr>
<tr>
<td>Polycythemia</td>
<td>6</td>
<td>4.2</td>
</tr>
</tbody>
</table>

|               | 141 | 100% |
attributable to ET = 0.8% of ET). A term baby, birth weight 2.3 kg developed cardia
colic 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 patho-
logical 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 hyperbiliru-
binemia were males. A maximal total se-
rum bilirubin of more than 20 mg/dl oc-
curred 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 fre-
cquency has been cited as 77.9%(2) and
82.7%(10). Breastfeeding leads to substan-
tial elevation of bilirubin levels during the
first few days of life.

G6PD deficiency was the second com-
monest 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 ac-
counted for 6.1% and 2.9% of cases in our
study respectively. This is lower than earli-
er reports(2,9,10).

The most common cause of hyperbilirubinemia needing ET was idio-
pathic followed by sepsis and G6PD defi-
ciency. The maximum number of exchang-
es were done for Rh isoimmunization, fol-
lowed by sepsis and G6PD deficiency. Rh isoimmunization, ABO isoimmunization,
G6PD deficiency and sepsis are the report-
ed commonest causes needing ET(2,11).
The incidence of major complications was
1.7 per 100 exchange transfusions com-
pared to 0.95 per 100 procedures reported
by others(12).

Exchange related mortality has been de-
fined as unexplained death during or with-
in six hours of the procedure(13). In the
present study, no death occurred within 6 h
of the exchange although two deaths oc-
curred within 48 h of exchange transfusion
in babies of 29 and 30 weeks gestations,
presumably due to infection and/or bleed-
ing. 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(11). As in the present study, no
death was reported within 6 h of the ET
from Delhi(2).

In conclusion, significant neonatal jaundice 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 signifi-
cant 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