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Case Series

Double volume exchange transfusion in neonates with severe jaundice in the era of light emitting diode phototherapy

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ABSTRACT

Neonatal jaundice, though a common clinical condition, severe hyperbilirubinemia necessitating exchange transfusion remains a concern due to acute bilirubin encephalopathy and long-term neurodevelopmental sequelae. Exchange transfusion has drastically reduced after Light emitting diode-intensive phototherapy. This study analyzed the clinico-etiological profile of jaundiced neonates who underwent double-volume exchange transfusion in a tertiary care center. This hospital-based descriptive cross-sectional study was conducted on neonates who underwent exchange transfusion from May to October 2024 over six months. Data obtained by structured proforma was analyzed with descriptive statistics. Among 9 neonates who had double volume exchange transfusion, 7 were boys and 5 were outborn. Their mean gestational age was 37.1 weeks and birth weight ranged from 1350 to 3720 gm. The etiologies identified include Rh incompatibility (1 case), prematurity (1 case), unknown (4 case) and 3 cases with multiple etiology (one with G6PD deficiency, OB incompatibility and Gilbert syndrome, one baby with G6PD deficiency and OB incompatibility, another baby with late onset neonatal sepsis with G6PD deficiency). Their peak serum bilirubin levels ranged from 19 to 45 mg/dl. Two neonates required multiple exchange transfusions. Post-exchange transfusion, 5 neonates had thrombocytopenia. Maximum modified bilirubin-induced neurological dysfunction score was 10/12 in one neonate with G6PD deficiency and OB incompatibility. This study reemphasizes severe neonatal jaundice requiring exchange transfusion still exists. We recommend community-level implementation of mandatory routine screening, early identification, and timely referral for neonatal jaundice by ASHA workers.

Keywords: Jaundice, Neonatal, Phototherapy, Exchange transfusion

INTRODUCTION

Neonatal jaundice, though a common clinical condition, severe hyperbilirubinemia necessitating exchange transfusion remains a concern due to acute bilirubin encephalopathy and long-term neurodevelopmental sequelae.[1] Double volume exchange transfusion (DVET), once common in the neonatal intensive care unit (NICU), has become rare due to advances in preventing haemolytic disease of the newborn and improvements in the detection and management of hyperbilirubinemia.[2] Exchange transfusion has reduced drastically nowadays post light emitting diode (LED) intensive phototherapy. We observed that the number of Double volume exchange transfusions was increasing in our unit. Hence, we analyzed the clinical-etiological profile of neonates who underwent exchange transfusions for severe neonatal jaundice.

CASE SERIES

This hospital-based descriptive cross-sectional study was carried out in the neonatal intensive care unit of a tertiary care center, on neonates who underwent exchange transfusion over six months from May to October 2024. Out of 215 NICU admissions, a total of 9 neonates underwent DVET. The age of onset of jaundice, postnatal age of NICU admission, postnatal age of performing

double volume exchange transfusion, cause of hyperbilirubinemia, an indication of exchange transfusion, post-procedure complications, modified bilirubin-induced neurological dysfunction (M-BIND) scoring, and duration of hospitalization were obtained from the discharge records.

All patients received phototherapy before and after the procedure. Data obtained by structured proforma was analyzed with descriptive statistics.

The demography and etiology were detailed in the following Tables 1 and 2 respectively.

Table 1: Demographic features of babies who underwent double volume exchange transfusion.

Demographic variables		Frequency (n=9)
Birthplace	Inborn	4
	Out born	5
Gestation	Term	8
	Moderate preterm	1
Sex	Boy	7
	Girl	2
Birth weight (gm)	2500-3999	7
	1500-2499	1
	1000-1499	1

Course

Figure 1 depicted below shown the onset of jaundice, severity, and the interventions done. The minimum age at which DVET was done at 36 hours in one baby who had multiple etiology and the maximum at 8 days of life. Two neonates required multiple exchange transfusions and one neonate had a maximum of 3 exchange transfusions. The peak serum bilirubin levels ranged from 19 to 45 mg/dl.

Table 2: Etiologic features of babies who underwent double volume exchange transfusion.

Etiology		Frequency, (n=-9)
Rh Incompatibility		1
Prematurity		1
Multiple etiology	Gilbert syndrome, glucose 6-phosphate dehydrogenase (G6PD) deficiency, OB incompatibility	1
	G6PD deficiency, OB incompatibility	1
	G6PD deficiency, Late onset neonatal sepsis	1
Unknown		4

Complications

Post-exchange, 5 neonates had thrombocytopenia, which was managed conservatively. Three neonates developed acute bilirubin-induced encephalopathy, confirmed with neuroimaging. One baby had a maximum M-BIND score of 10/12. This baby had persistent abnormal sensorium and tone, despite three DVET, and during follow-up found to have abnormal neurological examination with hypertonia and hearing impairment.

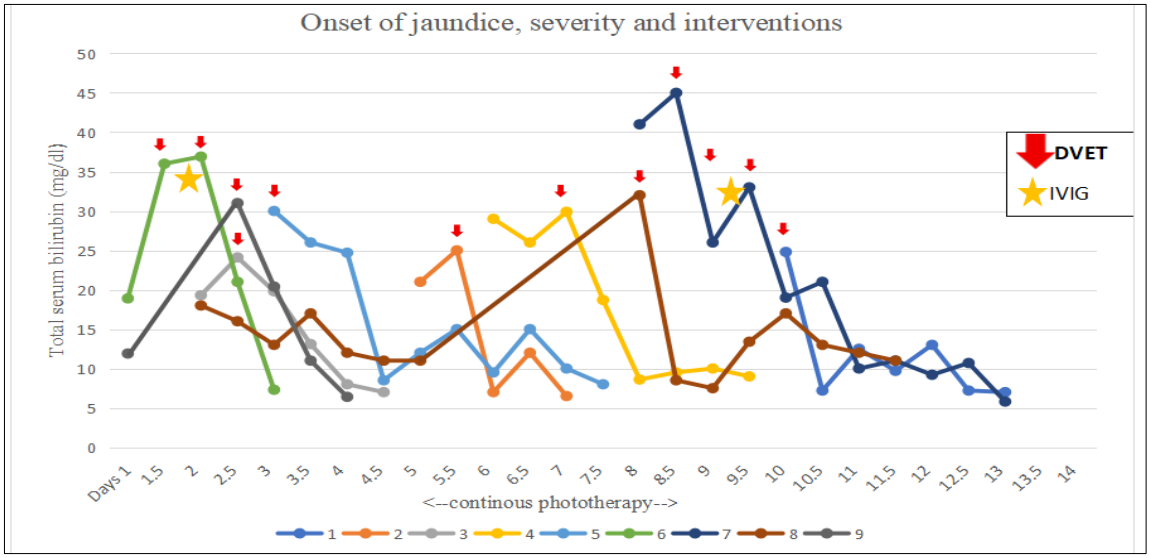

Figure 1: Onset of jaundice, severity, and the interventions done.

DISCUSSION

As per our unit protocol, we screen for neonatal jaundice before discharge. We observed that the number of DVETs seems to be higher in our unit. Hence, we analyzed the data for all neonates who underwent DVET during the six-month period, in contrast to all other studies, whose data span are over a period of eleven to twenty years and nearly

a decade earlier.[3-5] During the study period, out of 215 NICU admissions, 146 neonates had neonatal hyper-bilirubinemia requiring phototherapy and nine neonates (4.19 % of total NICU admissions) underwent 12 double volume exchange transfusions. In a retrospective study in India, observed over eleven-year period, out of 39,217 admissions, 1816 exchange transfusion procedures were done on 1575 neonates (4.01% of total NICU admissions).[4] The average age for exchange transfusion (ET) was 5 days for inborn babies and 7 days for outborn babies. In a single-center experience in northern India, Kakkar et al. highlighted that the average age for exchange transfusion is 9.8 days.[3] But in a large multi-center cohort study by Wolf et al, the median postnatal age at the time of ET was 1 day (0, 3 days) notably more than one-quarter of infants receiving an exchange transfusion on the day of birth; median postnatal age at discharge was 11 days.[5] In our study, the age of presentation was earlier than the study done by Kakkar et al but delayed as compared Wolf et al. Only one baby (11%) was moderate preterm with very low birth weight, all others were born at term gestation, however in a study by Kim et al, out of 23 babies who underwent exchange transfusion, 11 (48%) were preterm.6 Our study showed male preponderance (7 out of 9 babies), in contrasts to the study by Dey et al, where female preponderance is seen (56% of 41 babies).[1]

On evaluation, the etiology of neonatal jaundice we found were, OB incompatibility, Rh incompatibility, late-onset sepsis (methicillin-resistant *staphylococcus aureus*), prematurity, G6PD deficiency and Gilbert syndrome. Though the DCT was negative in one baby, but the elution test demonstrated blood group incompatibility. Dey et al in 2021 found that out of 41 babies, Rh incompatibility was observed in 33 cases, which accounts for 80% of the total neonates who underwent DVET.[1] In 2020, Kim et al found that the most prevalent cause for exchange transfusion was haemolytic anaemia due to non-ABO maternal blood group discrepancies.[6] Gilbert syndrome is a benign condition that results in occasional jaundice due to mild unconjugated hyperbilirubinemia, often triggered by stress, dehydration, or illness. It is generally asymptomatic in neonates, does not lead to significant hyperbilirubinemia, and typically does not require treatment.[7] Literature has shown an association between G6PD deficiency and Gilbert syndrome.[8-10] We have also seen a similar association in one inborn neonate, the baby developed jaundice within 24 hours of life not responding to LED phototherapy, and required an exchange transfusion at 36 hours of life. This early presentation may be secondary to the association with G6PD deficiency and OB incompatibility. The baby had persistent hyperbilirubinemia even after two exchange transfusions and required intravenous immunoglobulin (IVIG) infusion for ongoing haemolysis, after which the bilirubin level declined.

Another baby with multiple etiology was outborn and presented to us on day 8 of life with features of acute bilirubin encephalopathy and had maximum bilirubin level of 45 mg/dl. In a study by Alken et al, they found maximum bilirubin levels ranging from 30 to 50mg/dl with a median of 32 mg/dl.[11] The etiology of such high serum bilirubin in our case was found as G6PD deficiency with OB incompatibility and the baby required 3 times exchange transfusion with one IVIG infusion after which the bilirubin level decreased. Because of the irreversible neurological insult with maximum M-BIND score of 10/12, despite three ET, this baby presented with abnormal neurodevelopment and hearing impairment on follow-up.

Acute bilirubin encephalopathy refers to the acute condition caused by severe hyperbilirubinemia. Signs and symptoms may include decreased feeding, lethargy, abnormal muscle tone (either hypotonia or hypertonia), a high-pitched cry, retrocollis, opisthotonus, the setting-sun sign, fever, seizures, and possibly death.[12] Modified bilirubin-induced neurological dysfunction score (M-BIND) was developed to provide a more comprehensive identification of BIND in neonates with high bilirubin levels. It consists of eye movement, muscle tone, mental status, and cry patterns.[13] In our study, three out of nine neonates developed acute bilirubin-induced encephalopathy, with a maximum M-BIND score of 2,7 and 10. In a study by Radmachar et al, M-BIND score ≥10 was seen in seventeen babies out of 333 newborns with jaundice.[13] Currently, T1-weighted MRI images are used to discriminate acute bilirubin encephalopathy and non-acute bilirubin encephalopathy neonates with hyperbilirubinemia, In this study, T1 weighted magnetic resonance imaging (MRI) showed hyperintensity in basal ganglia in 3 babies.[14] Post-exchange, 5 neonates (55%) had asymptomatic thrombocytopenia with a minimum platelet count of 18,000/mm^3, which was managed conservatively, Sabzehei et al, in their study, found thrombocytopenia in 33 % of cases.[15] Another study had shown thrombocytopenia(less than 1 lakh/mm^3) in 57 % of cases of ET (81 out of 202 neonates) secondary to post-exchange transfusion with severe thrombocytopenia in 17% of cases (less than 50,000/mm^3).[16]

Limitations

Limitations of the study were single-center experience with a small sample size and long-term follow-up is yet to be done.

CONCLUSION

This case series reemphasizes severe neonatal jaundice requiring exchange transfusion still exists. Reducing preventable causes of permanent neurological damage by screening for severe neonatal jaundice is necessary. Recommendations include community-level implementation of mandatory routine screening, early identification, and timely referral for neonatal jaundice utilizing home-based newborn care visits by an ASHA worker using a transcutaneous bilirubin meter with a single cut-off value ≥13 mg/dl or gestational age-specific color-coded graphs in future studies.

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