

Complication of exchange transfusion at a tertiary care hospital

Author(s): Abdul Wahid Bhat, Churoo BA, Qazi Iqbal, Sheikh MA, Javeed Iqbal, Rubina Aziz

Vol. 15, No. 2 (2011-07 - 2011-12)

Abdul Wahid Bhat(1), Churoo BA, Qazi Iqbal, Sheikh MA, Javeed Iqbal, Rubina Aziz(1)

(1)Department of Transfusion Medicine and Department of Neonatology, Sher-I-Kashmir Institute of Medical Sciences, Srinagar Kashmir, India

Abstract

Although the value of exchange transfusion in the treatment of neonatal hyperbilirubinaemia is recognized, the procedure has some risks and complications. Medical records of 92 newborns who underwent exchange transfusion between September 2006 and August 2008 at Sher-I-Kashmir Institute of Medical Sciences Srinagar Kashmir were studied to determine the cause of jaundice and adverse events associated with neonatal exchange transfusion performed for hyperbilirubinemia. ABO incompatibility was the commonest cause for neonatal hyperbilirubinemia and was found in 25% followed by Rh. incompatibility in 21.3%. None was found to have G6PD deficiency. Complications following exchange transfusion were found in 20.7% neonates. Thrombocytopenia was found in 6%, Hypocalcaemia in 4.3%, Bradycardia, seizures, disseminated intravascular coagulation, Cardio respiratory arrest, hypoxia, necrotizing enterocolitis and apnea were found in 1% each. The overall mortality observed was 2.1%. The majorities of adverse events associated with exchange transfusion were laboratory abnormalities and were asymptomatic and treatable.

Key words: – Hyperbilirubinemia, Exchange transfusion (ET), Neonates
Accepted March 15 2011

Introduction

Jaundice is a common neonatal problem and is seen in 80% of preterm and 60% of term babies [1]. Pathological hyperbilirubinemia in term infants is bilirubin >12 mg% or 10-14 mg% in preterm infants or it appears in first 24-36 hours or beyond the 1st week of life and jaundice is caused by abnormal processes.[2] Hyperbilirubinemia predisposes to the risk of encephalopathy and long term sequel if not managed promptly. However in some neonates serum bilirubin level may rise excessively, which can be a cause of concern because unconjugated bilirubin is neurotoxic and can cause death in newborns and life long sequelae in infants who survive. Incidence of neonatal hyperbilirubinaemia varies with ethnicity and geography [2].

Use of phototherapy, intravenous immunoglobulin and exchange transfusion has resulted in a decline in the rates of significant neonatal jaundice [3,4,5]. Although the value of exchange transfusion in the treatment of neonatal hyperbilirubinaemia is recognized but the level at which ET is indicated remains controversial(3-5)complications attributed to exchange transfusion are common enough hence, should be performed in nurseries prepared to respond to such events. [6] Exchange transfusion (ET) in neonates is commonly used to remove antibody coated red blood cells and or products of hemolysis in various immune or non immune hemolytic anemia's. ET involves incremental removal of blood from the affected infant and replacement with fresh donor blood [7]. Mortality directly attributable to ET is reported to be at least 1% and a mortality of up to 3.2% has been reported in some series published before 1990 [8-14]. This study was undertaken to determine the etiology, rates of adverse events, mortality and morbidity related to exchange transfusion at a tertiary care hospital in Kashmir, India.

Material and Methods:

We prospectively analyzed 92 infants of age below one month who were admitted to the department of neonatology with hyperbilirubinemia and received exchange (ET) transfusion at Sher-I-Kashmir Institute of Medical sciences Soura Srinagar Kashmir over 2 yrs Period from September 2008 to August 2008.

The causes of jaundice were classified in the following ways:- Rh. disease was defined as jaundice in Rh. positive new born from Rh. Negative mothers and evidence of hemolysis and ABO disease was defined as jaundice in newborn with positive combs test against A or B antigens from type O mothers. Whole fresh ABO blood collected in CAPD compatible to both mother and baby were used.

Exchange transfusion were performed using double volume (160ml /Kg) exchange procedure and usually finished in 1- 1½ hrs. All exchange transfusions were performed via umbilical vein by repeatedly removing and replacing small aliquots of blood (5ml / Kg). Base line investigations like blood counts, direct and total bilirubin, and erythrocyte glucose-6-phosphate dehydrogenase (G6PD) level, direct combs test, blood culture, serum, albumin, calcium, sugar, and pre and Post Exchange blood cultures. No intravenous calcium gluconate was used during the exchange transfusion to neutralize the effect of citrate in CAPD. The definition of various complications recorded up to 7 days post ET used were as under: – hypoglycemia if serum glucose was less than 40mg/dl hypocalcaemia if serum calcium was less than 8mg%, thrombocytopenia if platelet count was less than 100000/mm³, bradycardia if heart rate was less than 80 beats per minute, Apnea cessation of respiration for more than 20seconds, seizure any tonic or clonic moment, necrotizing enterocolitis defined as per Bell et al's criteria.

Results:

During the two years period from September 2006 to August 2008, 98 ET in 92 newborn infants full filled the criteria for inclusion into the study. Six infants had received exchange transfusion twice. The infants had a mean gestational age of 38.7 ± 1 weeks and mean birth weight of 2.53 ± 0.52gm. The most common cause (Table-1) of jaundice was ABO incompatibility and was found in 23(25%) followed by Rh incompatibility in 19(20.6%). No patient was found to have G6PD deficiency. The mean total bilirubin was 24.9 ± 3.8mg/dl. And the mean age at presentation was 4 ± 1 days. 11 patients had bilirubin of more than 30mg/dl at admission. Patients with ABO incompatibility had a higher bilirubin level as compared to Rh. incompatibility. Abnormal neurological examination was detected in 11 patients at the first presentation to hospital, which included hypotonia in 5, hypertonia in 4, abnormal cry and movements of body in one each. All the 11 patients with abnormal neurological examination had serum bilirubin more than 30mg% complications occurred in 20 infants. The most common complication was thrombocytopenia and was observed in 6 followed by asymptomatic hypocalcaemia in 4. Bradycardia, seizures, DIC, cardio respiratory arrest, Hypoxia, necrotizing enterocolitis, apnea with need for resuscitation during or immediately post ET occurred in one patient each. Two infants died within 24 hrs of exchange transfusion.

Table 1. Complications of Exchange Transfusion in 92 infants

Complication	No.	Percentage
Thrombocytopenia (plateletcount <100,000/mm ³)	6	6.5
Hypoglycemia	4	4.3
Hypocalcemia	1	1
Bradycardia	1	1
Seizure	1	1
DIC	1	1
Cardiorespiratory arrest	1	1
Hypoxia	1	1
Necrotising enterocolitis	1	1

Apnea	1	1
Death	2	2.1

Discussion

Exchange transfusion ET has been shown to reduce brain damage in severely jaundiced patients. Simultaneously indications for exchange transfusion in jaundice have changed markedly in the past decade in developed countries, following increased use of rhesus anti D and wide spread use of phototherapy in neonatal jaundice [15,16]. Exchange transfusion is associated with serious adverse events including death, and it is likely that the complications of exchange transfusion would increase with amount of blood exchanged. We observed a high rate of adverse events associated with exchange transfusion for neonatal hyperbilirubinaemia that is 20 (21.7%) most of these adverse events were asymptomatic and transient and did not need any intervention especially thrombocytopenia and hypocalcaemia. Although it is evident by different studies that there is a progressive decline over the past decade in the number of neonates who need exchange transfusion for neonatal hyperbilirubinaemia, but still it is needed in 7% of neonates admitted to hospital with hyper-bilirubinaemia. [17].

The commonest cause for hyperbilirubinaemia and exchange transfusion was ABO incompatibility and was observed in 23(25%) of neonates others also have reported the ABO incompatibility as the commonest cause for exchange transfusion in 35.9% and 21.3% of their patients (18). ABO incompatibility had a higher bilirubin level as compared to Rh incompatibility. We did not find any patient having G6PD deficiency. This is in contrast to observed prevalence of 10% and 12.1% and 8.1% by others (19-20). This low prevalence of G6PD deficiency in our group of patients could be due to racial difference with lower prevalence of G6PD found in our population. 6.5% of our neonates needed more than one exchange transfusion and is lower than has been observed by others. (6,13,20) The co morbid factors responsible were seizure, bradycardia, hypoxia, necrotizing enter colitis, DIC, attacks of apnea and were observed in 1% each and corresponds to the 1.5% as observed by other studies [6]. Permanent sequel were not found in any of our patients who underwent single or multiple exchange transfusion while, some have reported it in 1% of their newborn undergoing exchange transfusion [21]. Transient asymptomatic complications in the form of thrombocytopenia and hypocalcaemia were observed in 11% of neonates following exchange transfusion. Bodice-Z has reported this in 6% of his patients. [6] While Patra et al showed such complications in 24% of their Patients [22] Mortality directly attributable to exchange transfusion is reported to be at least 1% and is due to unexplained cardiac arrest, cardio arrhythmias or air embolism [23]. We observed a mortality of 2.1% While, others have reported a mortality of 1.5% and 0.66%. [6,10] Chime reported no death in his study conducted in 1980's [24]. Although the complications of variable severity with potential death in neonate are uncommon in exchange transfusion still, continuous monitoring should be offered to neonates undergo exchange transfusion and all the facilities be kept ready for any emergency event.

References:

1. Abu-Ekteish F, Daoud A, Ramawi H, Kakish, K, Abu-Heija-. Neonatal Exchange Transfusion: A Jordanian Experience. *Ann Trop Pediatrics*. 2000; 20: 57-60.
2. Barbara J Stoll. Jaundice and hyperbilirubinemia in the newborn Kern ictus. In Richard Behram, Robert M. Kleigman, Hal. B. Jensen, eds. *Nelson Text Book of Pediatrics* 17th ed. Saunders, 2004; 592-599
3. Walchko JF, Aski FA. Bilirubin 20mg/dl = Vigin-tiphoia *Pediatrics*. 1983; 71: 660-663.
4. Newman TB, Maisels MJ. Evaluation and treatment of Jaundice in the term newborn: A kinder, gentler approach. *Pediatrics* 1992; 89: 819-831.
5. Jackson JC. Adverse events associated with exchange transfusion in healthy and ill newborns *Pediatrics* 1997; 99: E7.
6. Badice.Z Exchange transfusion in neonatal hyper-bilirubinemia experience in Isfahan, Iran. *Singapore Medical Journal* 2007; 48 (5): 422.
7. Hoontrakoons, Suputtamongkoby exchange transfusion as an adjunct to treatment of Severe Falciparum malaria, *Tropical medicine and international health*. 1998; 3: 156-61.
8. Boggs TR, Westphal MC. Mortality of exchange transfusion *Pediatrics*. 1960; 26: 745-755.
9. Weldon VV, Odell GB. Mortality of exchange transfusion *Pediatrics*. 1968, 41: 797-801.
10. Panagopoulos G, Valaes T, Doxiadis SA. Morbidity E Mortality related to Exchange transfusion *J. Pediatrics* 1969; 74: 247-254.
11. Kitchen WH. Neonate Mortality in infants receiving an exchange transfusion. *Aust. Pediatric*. 1970; 6: 30-40.

12. Hovi L, Siimes MA. Exchange with fresh heparin blood is a safe procedure. Experience from to 69 newborns Pediatric second. 1985; 74: 360-365.
13. Dikshit. SK, Gupta PK, Exchange transfusion in neonatal hyperbilirubinemia Indian Pediatric.1989; 26:1139- 1145.
14. Guran RL, Drew JH, Walkins AM. Jaundice clinical practice 88000 live born infants. Aust NZJ obst. Gynaecol. 1992; 32: 186- 192.
15. Lucey JF. Neonatal jaundice and phototherapy Pediatr Clin North Am. 1972; 19:827-839.
16. Woodrow JC, Effectiveness of Rh prophylaxis. Haema-tologica Budap 1974; 8: 281-290.
17. Funato M, Tamai H, Shimada S. Trends in neonatal exchange transfusion at yodogawa Christian hospital. Acta Pediatr Jp 1997; 39:305-308
18. Sanparvat S. Exchange transfusion and its morbidity in ten year at king Chula longkorn hospital. J. Med. Assoc Thi. 2005; 88: 588-592
19. Madanat F, Karadsheh N, Shamayleh A, Tarawneh M, Khraisha S, Bata M et al. Glucose phosphate deficiency in male newborns. Jordan Med J 1986; 21:205-212
20. Tan KL. Glucose 6 phosphatase dehydrogenase status and neonatal jaundice. Arch Dis. Child 1981; 56:874-877
21. Jackson JC. Adverse events associated with exchange transfusion healthy and ill new born. Pediatrics 1997; 99:E7
22. Patra K, Storfer isser A, Siner B, Moore J, Hack M Adverse effects associated with neonatal exchange transfusion in the 1990s. J. Pediatrics 2004; 144: 626-631
23. Bohggs TR, Westphal MC Mortality of exchange transfusion Pediatrics 1960; 26:745-55
24. Chima RS, Johnson LH, Bhutani VK. Evaluation of adverse events due to exchange transfusion in term and near term newborns Reseach. 2001; 49: 324A

Correspondence to:

Abdul Wahid Bhat

C/O Noorani Medicines

Near Govt. Boys High School Rawalpura

Srinagar 190005, Kashmir, India