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Effects of Exchange Blood Transfusion on Serum Electrolytes, Calcium and Phosphorus among Neonates with Jaundice

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Summary

Akarakiri TAF and Laditan AAO. Effects of Exchange Blood Transfusion on Serum Electrolytes, Calcium and Phosphorus among Neonates with Jaundice. *Nigerian Journal of Paediatrics* **1983; 10: 89.** The effects of exchange blood transfusion (EBT) on serum electrolytes, calcium and inorganic phosphorus were studied in 30 infants with neonatal jaundice. The study revealed a higher serum sodium, potassium and phosphorus in the donor blood samples than in the samples obtained from all the infants at the beginning, during and at the end of EBT ($p < 0.001$). There were also significantly lower ($p < 0.001$) chloride and bicarbonate in the donor blood than in the infants' blood during the procedure. During the transfusion, no abnormal electrolyte pattern was observed, although there were changes in the serum concentration of calcium and phosphorus due, perhaps, to calcium gluconate which was administered routinely during the procedure. It is suggested that administration of calcium gluconate during EBT is unnecessary and may, in fact, be injurious.

Introduction

The effectiveness of exchange blood transfusion (EBT) in the management of severe neonatal hyperbilirubinaemia is now well established. The increasing number of newborns with neonatal jaundice (NNJ) in Ibadan has led to an extensive use of EBT. The procedure is not without hazards,[1-5] although most babies tolerate it quite satisfactorily. One serious possible complication which may arise from the procedure is electrolyte disturbance. The present study was carried out to assess the effects of EBT on serum electrolytes, calcium and phosphorus.

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Materials and Methods

The subjects comprised 30 consecutive out-patient cases of NNJ who required EBT in the Children Emergency Room (ChER), University College Hospital (UCH), Ibadan, during the period, June–August 1982. The criteria for EBT and the procedure are as previously described,[6] but briefly, full term babies were exchanged if the total bilirubin level exceeded 340 µmol/L (20mg/

100ml), while pre-term babies had EBT done at the level of 255 mmol/L (15mg/100ml). Twice the blood volume was exchanged using warm blood of less than seven days old and preserved with bufferred citrate-phosphate-dextrose. In addition to obtaining a sample of donor blood, blood samples were obtained from the newborn infant before the start of the procedure, halfway and at the end. One millilitre of 10% calcium gluconate was administered intravenously after every 100ml of donor blood had been transfused. All the babies were full-term except two who were pre-term weighing 1.0 and 1.9kg, respectively.

Results

The 30 infants (21 males and 9 females) were aged, between one and six days (mean age, 3 days). The causes of the jaundice are listed in Table I. Glucose-6-phosphate dehydrogenase (G-6-PD) deficiency either alone or in combination, was the commonest cause of the jaundice.

Table II summarizes the mean±SEM serum electrolytes, urea, calcium and phosphorus in the donor blood as well as in blood samples obtained during the EBT. It will be observed that the donor blood contained a high serum sodium 155±2mmol/L (155±2 mEq/L) and potassium 10.3±0.7mmol/L (10.3±0.7 mEq/L) and low chloride, 76±1mmol/L (76±1 mEq/l) and bicarbonate, 14±1mmol/L (14±1mEq/L). Serum urea and calcium were within normal range, while serum phosphorus was higher than normal 4.9±0.2 mmol/L (15.2±0.7 mg/100ml). There was a significant difference ($p<0.001$) between the levels of sodium, potassium, chloride, bicarbonate and phosphorus in the donor blood and those in blood samples obtained from the babies before the onset of the transfusion. This difference persisted throughout the procedure. The calcium concentration rose progressively throughout the transfusion, while serum phosphorus showed some rise between the onset and the end of transfusion.

Serum calcium concentration in the donor blood samples ranged from 1.6 to 2.9 mmol/L (6.4 to 11.5mg/100ml). It ranged from 1.6 to 2.0 mmol/L (6.4–8.0 mg/100ml) in donor blood given to five babies, from 2.0 to 2.2mmol/L (8.0–8.8mg/100ml), in the blood for seven babies and from 2.3 to 2.9 mmol/L (9.2–11.5mg/100ml) in the blood for 18 babies. The high levels of the calcium in the donor blood did not appear to affect any of the infants since none of them developed cardiac arrythmia.

Two infants died within 24 hours of the EBT; both had severe septicaemia.

Discussion

Although disturbances of electrolytes and acid balance such as acidosis, hyperkalaemia, citrate intoxication and hypocalcaemia are thought to be responsible for the various complications of EBT,[7–10] recent studies[11 12] including the present one, have revealed no abnormal serum electrolytes in the blood samples obtained from the babies. In other words, the gross abnormal electrolytes in the donor blood in the present study did not appear to have affected corresponding levels in the infants. The successful adaptation of donor blood by the babies may have been due to some hormonal regulations involving such hormones as calcitonin, parathyroid hormone, vitamin D and aldosterone. Furthermore, the normalisation of electrolytes, calcium and phosphate levels in the neonates could have been achieved by the fact that all the babies except two, were full-term and healthy babies with normal renal function.

The routine administration of calcium gluconate during EBT is questionable because serum calcium concentration was low in only a small number of donor blood. For instance, only five of the donor blood samples had calcium levels which ranged between 1.6–2.0 mmol/litre (6.4–8.0 mg/100ml). Thus, the progressive rise of serum calcium in the patients, which did not correlate with the levels in the donor samples, were due to

TABLE I

Causes of Neonatal Jaundice in 30 Infants

Cause	*No. of Cases*
G-6-PD deficiency alone	8
G-6-PD deficiency + infection	5
G-6-PD deficiency + ABO incompatibility	2
G-6-PD deficiency + Rh incompatibility	1
ABO incompatibility alone	3
Infection alone	2
Low birthweight alone	2
Unknown	7

G-6-PD = Glucose-6-Phosphate dehydrogenase

TABLE II

Mean ± SEM Serum Electrolytes, Calcium and Inorganic Phosphorus in 30 Infants requiring EBT

	Donor Blood	*Exchange Blood Transfusion*		
		Onset	*Halfway*	*End*
Sodium	155 ± 2	138 ± 1*	139 ± 1*	140 ± 1*
Potassium	10.3 ± 0.7	4.7 ± 0.2*	4.1 ± 0.2*	4.1 ± 0.2*
Chloride	76 ± 1	100 ± 1*	98 ± 1*	97 ± 1*
Bicarbonate	14 ± 1	20 ± 1	18 ± 1	19 ± 1
Urea	4.3 ± 0.7	7.7 ± 0.8	6.6 ± 0.7	5.5 ± 0.6
Calcium	2.2 ± 0.1	2.6 ± 0.1	2.8 ± 01*	3.2 ± 0.1*
Phosphorus	4.9 ± 0.2	2.6 ± 0.2*	3.3 ± 0.2*	3.0 ± 0.2*

* $P < 0.001$ (difference between donor and babies' blood samples)
All values are in mmol/L

Conversion: SI units to Traditional units
For sodium, potassium, chloride and bicarbonate:

	1mmol/L	=	1mEq/L
For calcium:	0.25mmol/litre	=	1mg/10 ml
For phosphorus:	0.32 mmol/litre	=	1mg/100ml
For urea	0.166mmol/litre	=	1mg/100ml

the administered calcium gluconate during EBT. It is, therefore, not unlikely that calcium gluconate administration may sometimes be responsible for cardiac arrythmias and arrest. The hazards of EBT are probably not the result of changes in the concentration of sodium, potassium, chloride, bicarbonate, calcium and phosphorus. They are possibly related to the clinical state of the baby at the time of EBT and the aetioloy of NNJ such as Rhesus incompatibility and infection.

Regretably in the present study, both the serum citrate and albumin levels which might have influenced the serum calcium and phosphate concentrations were not determined. It is hoped however, that in future studies on this subject, not only will these two biochemical variables be assessed but some of the hormones that are known to regulate serum calcium values will also be determined.

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References

1. Boggs TR Jr and Westphal MC Jr. Mortality of exchange transfusion. *Pediatrics* 1960; **26:** 745-55.
2. Taylor WC. Mortality and morbidity of exchange transfusion. *Can Med Ass J* 1962; **87:** 1267-70.
3. Panagopoulos G, Valaes T and Doxiadis SA. Morbidity and mortality related to exchange transfusions. *J Pediat* 1969; **74:** 247-54.
4. Hashemi-Tasab AR and Ziai M. Mortality of exchange transfusion and risks associated with hyperbilirubinaemia. *Indian J Pediat* 1971; **38:** 11-14.
5. Laditan AAO, Effiong CE and Antia AU. Morbidity and mortality from exchange blood transfusion in neonatal jaundice. *Nig J Paediat* 1975; **2:** 42-6.
6. Effiong CE and Laditan AAO. Neonatal jaundice in Ibadan: A study of cases seen in the out-patient clinic. *Nig J Paediat* 1976; **3:** 1-8.
7. Miller G, McCoord AB, Joos HA and Clausen SW. Studies of serum electrolyte changes during exchange transfusion. *Pediatrics* 1954; **13:** 412-18.
8. Bolande RP, Traisman HS and Philipsborn HF. Electrolyte considerations in exchange transfusion for erythroblastosis foetalis. *J Pediat* 1956; **49:** 401-6.
9. Farquhar JW and Smith H. Clinical and biochemical changes during exchange transfusion. *Arch Dis Childh* 1958; **33:** 142-59.
10. Taylor WC, Griodale LC and Stewart AG. Unexpected death from exchange transfusion. *J Pediat* 1958; **52:** 694-700.
11. Pierson WE, Barrett CT and Oliver TK Jr. The effect of bufferred and non-bufferred ACD blood on electrolyte and acid base homeostasis during EBT. *Pediatrics* 1968; **41:** 802-14.
12. Tan KL and Tan IK. Plasma potassium, sodium and chloride levels during and after EBT. *Austr Paediat J* 1975; **11:** 165-8.

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