SUMMARY AND CONCLUSION

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Exchange transfusion has been performed with increasing frequency over the last few years.

Exchange transfusion is not a procedure without hazards. Complications which had been encountered during exchange transfusion include both general and metabolic complications. Metabolic complications include : hypocalcaemia, hyperkalemia, hypernatremia and rebound hypoglycemia. So, careful attention will avoid most of these hazards of exchange transfusion.

Hypocalcaemia is common due to the complexing of ionized Ca^{+} by citrate in citrated donor blood. The slow administration of 1 ml 10% calcium gluconate after every 100 ml blood exchange counteract the citrate binding and helps avoid occurrence of hypocalcaemia.

Hypoglycemia is probably due to hyperinsulinism resulting from the high glucose load in the donor blood. The mean glucose level decreased gradually to reach 36.24 ± 16.5 mg/dl in full term infants and 26.73 ± 19.16 mg/dl in preterm infants at 2 hours after exchange transfusion, it has to be stressed that these are levels indicating serious severe

hypoglycemia which may cause complications even if the patient does not seem symptomatic. Hypernatremia after the exchange transfusion is due to high sodium content of citrate phosphate dextrose blood (165-170 mmol/L). The danger of hypernatremia is the intracranial hemorrhage especially in very low birth weight infants.

There was a transient hyperkalemia for at least 72 hours noticed in babies exchanged with stored banked blood. On the other hand, serum potassium was transiently depressed in cases transfused with fresh blood. By 72 hours after the procedure fortunately potassium approached normal level in both groups. Therefore we recommended the use of the fresh available blood in every transfusion as well as estimation of potassium level when stored blood is resorted to when there is absolutely no other alternative.

From our study we have noticed that IgG is lower in preterm infants and hence they may be more prone to infections. IgG is known to be transferred across the placenta and therefore the newborn and adult levels are similar and no significant change in IgG level was noticed after exchange transfusion. However in cases with RH incompatibility IgG levels have been noticed to be significantly above normal levels in these newborns due to the extremely high level of IgG in the sensitized mothers crossing the placenta to the baby. This does not occur with ABO incompatibility.

Normal IgM values in newborns are know to be 8-20 mg/100 ml which is 10% of the normal adult values (80-200 mg/100 ml). In our studied newborns the mean IgM levels were significantly higher than the normal levels for newborn infants either due to IgM response to septicemia or presence of naturally occurring isohemagglutinins in the fullterm HDN cases.

The group of septicemic infants have the highest mean IgM level and this probably indicates very early or even in-utero infection. After exchange transfusion the elevation of IgM is due to attainment of the adult levels present in the donors blood.

Exchange transfusion is an effective and promising line of treatment in neonatal septicemia and should not be delayed until severe complications develop such as, sclerema, severe disseminated intravascular coagulation, or neurological sequelae.