Nigerian Journal of Paediatrics 1983; **10**: (2) 31–34

Incidence of Neonatal Polycythemia in Ibadan

AH DAWODU* AND A AKINKUNMI**

Summary

Dawodu AH and Akinkunmi A. Incidence of Neonatal Polycythemia in Ibadan. *Nigerian Journal of Paediatrics* **1983; 10**:31. Five hundred consecutive liveborn infants screened for polycythemia revealed an incidence of 3·2% which is close to 3·9% reported among American newborn infants. Intrauterine growth retardation particularly among products of multiple pregnancy, was the major cause in the series. It is suggested that large-for-gestational-age and appropriately-grown infants should also, be routinely evaluated for polycythemia since as many as 45% of the infants with polycythemia may be symptomatic and delay in management may result in serious disability.

Introduction

POLYCYTHEMIA is a recognised haematological problem in the newborn period.[1 2 3] It may be associated with respiratory distress, cyanosis, heart failure and convulsion with resultant brain damage.[2] Hyperviscosity plays a major role in the causation of the disease complex associated with polycythemia and partial exchange transfusion has been found to be a successful line of management.[2] Infants with intrauterine growth retardation have been shown to be at the highest risk of developing polycythemia.[3 4] Other causes of polycythemia include chronic intrauterine hypoxia,[5] delayed clamping of the umbilical cord,[6] and twin-to-twin transfusion[7] or mother to foetus transfusion.[8] In Nigeria as well as in many developing countries, the incidence of low birthweight is high and most of these infants are small for gestational age.[9 10] Furthermore, the incidence of twinning in Nigeria is about five times that reported for the technically developed countries.[11] These factors may predispose infants to neonatal polycythemia.

The present study was undertaken in order to estimate the incidence of polycythemia in Nigerian neonates and to define the risks of such affected infants.

College of Medicine, University College Hospital, Ibadan

Department of Paediatrics
*Senior Lecturer
**Chief Laboratory Scientist

Materials and Methods

Five hundred consecutive liveborn infants delivered at the University College Hospital (UCH), Ibadan, during January and February 1979 were screened for evidence of polycythemia. The umbilical cord was clamped immediately after the birth, i.e. before one minute of life.

Capillary haematocrit was determined in each infant within 8 hours of birth using blood collected from a warmed heel. A repeat venous blood haematocrit was undertaken on any infant with a capillary haematocrit of 70% and above. Forty consecutive infants with capillary haematocrit of between 65 and 69% also had venous haematocrit checked in order to determine if any of these infants with capillary haematocrit within the normal range, ran the risk of polycythemia. A venous haematocrit of 65% and above was regarded as polycythemia.[1] The clinical course of all the infants with polycythemia was carefully noted. The birthweight of each infant was recorded and the gestation calculated from the mother's last menstrual period or by clinical assessment using the Dubowitz scoring system,[12] which has been found to be applicable to infants in the UCH.[13]

Results

Of the 500 infants screened, 42 (8.4%) had capillary haematocrit of 70% and above. Fifteen (33.3%) of these 42 infants were confirmed to have venous haematocrit of 65% and above. In contrast, only one (2.5%) of the 40 infants with capillary haematocrit of 65–69% was found to be polycythemic. Therefore, 16 (3.2%) of the 500 infants were confirmed polycythemic. Seventy-one (14.2%) of the 500 infants were of low birthweight (LBW) and 35 (49.3%) of these LBW infants were small for gestational age (SGA), using Freeman's intrauterine growth chart.[14] Table I summarises the influence of intrauterine growth pattern on the incidence of polycythemia. The incidence of polycythemia was highest among the SGA infants, followed by the large-for-gestational age (LGA) babies.

Among the eight polycythemic SGA infants, four were products of multiple pregnancies. Two of the mothers of the remaining four cases had hypertension. One other mother had sickle cell anaemia; her haematocrit on admission was 20%, but the infant had a venous haematocrit of 69%. The cause of the growth retardation in the fourth case was not obvious. One of the six AGA infants, had Down's syndrome and polycythemia, while one of the two LGA infants was an offspring of a diabetic mother.

Table II summarises the clinical and laboratory features in the 16 babies with polycythemia. Ten (63%) of the 16 babies were plethoric on admission, while seven (44%) were symptomatic. The main clinical features among the symptomatic group included respiratory distress, cyanosis, jitteriness, lethargy, convulsion, jaundice and hypoglycaemia. In four infants, the respiratory distress and central nervous system disturbances were severe enough to warrant partial exchange transfusion using plasma.

TABLE 1

Influence of Intrauterine Growth on the Incidence of Polycythemia

Author	*Intrauterine Growth*	*No. of Cases*	*No. of Infants with Polycythemia*	*Percent of Total*
Present series	Appropriate for gestational age (AGA)*	438	6	1.4
	Large for gestational age (LGA)*	27	2	7.4
	Small for gestational age (SGA)*	35	8	22.9
	Total	500	16	3.2
Wirth et al[3]	AGA	643	18	2.8
	LGA	91	6	6.6
	SGA	56	7	12.5
	Total	790	31	3.9

* AGA=10th-90th centile; LGA=90th centile; SGA=10th centile using Freeman's chart.[14]

TABLE II

Clinical and Laboratory Features in 16 Neonates with Polycythemia

Feature	*No. of Cases*	*Percent of Total*
Plethora	10	63
Respiratory distress	4	25
Jaundice	4	25
Cyanosis	3	19
Jitteriness	3	19
Lethargy	3	19
Hypoglycaemia	3	19
Convulsion	1	6

Discussion

The present study has revealed neonatal polycythemia among Nigerian infants. In the series, the incidence was similar to that reported among American newborn infants.[3] In a previous study of haematological values in Nigerian newborn infants, the range of haematocrit was 42–79% on the first day of life,[15] but venous haematocrit was not estimated in order to determine the incidence of polycythemia among infants with capillary haematocrit of 70% and above. Thus, this is, to the best of our knowledge, the first study on neonatal polycythemia in Nigeria.

It has been suggested that a capillary haematocrit of 70% is a useful cut-off point in identifying infants who require determination of venous haematocrit in order to detect polycythemia.[3] In the present study, 2.5% of infants in whom the capillary haematocrit was less than 70% did have venous haematocrit of 65% and above. This indicates that using a capillary haematocrit of 70% as a cut-off point for further investigation may under-estimate the incidence of neonatal polycythemia. It is therefore, suggested that in a neonate with plethora and symptoms associated with hyperviscosity,[2] a venous haematocrit is mandatory, in order to exclude polycythemia even if the capillary haematocrit is less than 70%.

The commonest cause of polycythemia in the present study and in the study by Wirth, Goldberg and Lubchenco,[3] was intrauterine growth retardation, but the percentage of small-for-gestational-age infants in the present study was higher than that reported by these other workers. Most of the intrauterine growth retardation in our series was associated with multiple pregnancy. Although twin pregnancy may produce polycythemia as a result of twin-to-twin transfusion, this usually arises from monochorionic placentation, a condition which has been found to be uncommon in twin pregnancies in Nigeria.[11] The major factor responsible for the polycythemia among twins in Nigeria seems to be intrauterine malnutrition, rather than twin transfusion syndrome. Relative chronic intrauterine hypoxia probably accounts for the growth retardation and polycythemia in the infant of the mother with sickle cell anaemia. With improved medical care and increasing number of girls with sickle-cell anaemia reaching child-bearing age, some of their offsprings will present with problems associated with intrauterine growth retardation[16] and will need careful monitoring after birth.

Except in the infant with Down's syndrome, a condition which is known to be associated with polycythemia,[17] the cause of polycythemia among the appropriately grown infants was unknown, although it is possible that maternal-foetal transfusion played a role in some of the infants. When the umbilical cord is clamped within one minute, as was the case in the present study, there is no significant rise in the haematocrit.[3] Thus, delayed clamping of the cord probably did not play a significant role in these patients.

The spectrum of clinical problems, probably due to hyperviscosity, in the present series was similar to that reported by other workers.[2]

Although blood viscosity was not determined in these infants, it is known that infants with venous haematocrit of 65% and above have hyperviscosity.[3] The high prevalence of clinical problems emphasizes the need for careful observation of infants at risk, who may require special care soon after birth. Management of infants with symptomatic polycythemia should include partial exchange transfusion with plasma.[18] This modality of treatment has been shown to reduce haematocrit and viscosity and improve blood flow.[19]

Acknowledgements

We are grateful to Dr F Jaiyesimi for useful comments on the manuscript and Mrs E M Oseni for secretarial assistance.

References

1. Kontras SB. Polycythemia and hyperviscosity syndromes in infants and children. *Pediat Clin North Am* 1972;**19**: 919-33.
2. Gross GP, Hathaway WE and McGaughey HR. Hyperviscosity in the neonate. *J Pediat* 1973; **82**: 1004-12.
3. Wirth HF, Goldberg EK and Lubchenco OL. Neonatal hyperviscosity. I: Incidence. *Pediatrics* 1979; **63**: 833-6.
4. Humbert JR, Abelson H, Hathaway WE and Battaglia FC. Polycythemia in small for gestational age infants. *J Pediat* 1969; **75**: 812–9.
5. Gatti RA, Muster AJ, Cole RB and Paul MR. Neonatal polycythemia with transient cyanosis and cardic-respiratory abnormalities. *J Pediat* 1966; **69**: 1063-72.
6. Usher R, Shepherd M and Lind J. The blood volume of the newborn infant and placental transfusion. *Acta Paediat* 1963; **52**: 497-512.
7. Rausen AR, Seki M and Strauss L. Twin transfusion syndrome. *J Pediat* 1965; **66**: 613-28.
8. Micheal AF and Mauer AM. Maternal-fetal transfusion as a cause of plethora in the neonatal period. *Pediatrics* 1961; **28**: 458-61.
9. Effiong CE and Dawodu AH. Neonatal morbidity and mortality in a special-care baby unit in Nigeria. *E Afr J Med Res* 1977; **4**: 25-8.
10. WHO Statistics Quarterly 1980; **33**: 197-224.
11. Nylander PPS. The value of the placenta in the determination of zygosity-a study of 1052 Nigerian twin maternities. *J Obstet Gynaecol Br Commonwlth* 1969; **76**: 699-704.
12. Dubowitz CJ, Dubowitz V and Goldberg C. Clinical assessment of gestational age in the newborn infant. *J Pediat* 1970; **77**: 1-10.
13. Dawodu AH and Effiong CE. Assessment of gestational age in full-term and preterm African newborn infants. *Nig J Paediat* 1977; **4**: 1-5.
14. Freeman MG. Gra ves WL and Thompson RL. Indigent negro and caucasian birth weight gestational age tables. *Pediatrics* 1970; **46**: 9–15.
15. Effiong CE, Taiwo O, Odeniyi OO and Mellits EO. Haematological values in healthyfull-term Nigerian newborn infants. *Biol Neonate* 1976; **28**: 336-46.
16. Usher RH. Clinical and therapeutic aspects of fetal malnutrition. *Pediat Clin N Amer* 1970; **17**: 169-83.
17. Weinberger MM and Oleinick A. Congenital marrow dysfunction in Down's syndrome. *J Pediat* 1970; **77**: 273-9.
18. Oski F. Haematological problems. In: Avery G B. ed. Neonatology—Pathophysiology and management of the newborn. Philadelphia: J B Lippinocott Co 1981: 564-5.
19. Rosenkrautz TS and Oh W. Cerebral blood flow-velocity in infants with polycythemia and hyperviscosity. Effects of partial exchange transfusion with plasmanate. *J Pediat* 1982; **101**: 99-102.

Accepted 24 February 1983