Nigerian Journal of Paediatrics, 4 (2), 19

The Incidence of Neonatal Hypoglycaemia in Benin

JACKSON A. OMENE

Department of Child Health, University of Benin, Benin City

Summary

Omene, J. A. (1977). *Nigerian Journal of Paediatrics*, 4 (2), 19. **The Incidence of Neonatal Hypoglycaemia in Benin.** Blood sugar concentrations were evaluated among 1,061 infants admitted into the Special-Care-Baby-Unit, Department of Child Health, University of Benin, over a two-and-half-year period. Seventy infants developed hypoglycaemia, giving an incidence of 6.6 per cent. The incidence for low birthweight infants was 7.9 per cent. Asphyxia was the single most important perinatal factor which predisposed the neonate to hypoglycaemia. Other at-risk infants were those delivered by emergency Caesarean section, low birthweight infants and septicaemic infants.

NEONATAL hypoglycaemia has been extensively studied over the last two decades. It is commonly associated with infants of diabetic mothers (Farquhar, 1959; Cornblath and Schwartz 1966); infants of toxaemic mothers (Cornblath, Odell and Levin, 1959; Cornblath and Reisner 1965; Fluge, 1974), and small-for-gestational-age infants (Lubchenco and Bard 1971). Symptomatic neonatal hypoglycaemia resulting in neurologic sequelae is still a subject of much concern (Haworth and McRae 1967; Beard et al., 1971).

The present study was undertaken in order to determine the incidence of neonatal hypoglycaemia as well as factors that predispose the high-risk infants to the development of hypoglycaemia. High-risk infants in this context include: infants weighing 2500 gm or less (LBW), asphyxiated infants, infants of pre-eclamptic and eclamptic mothers, infants of diabetic mothers and infants delivered by Caesarean section.

Methods and Materials

From April 1, 1974 to September 30, 1976, blood glucose was estimated on all infants admitted into the Special-Care-Baby-Unit, Department of Child Health, University of Benin. Blood sugar concentration was initially evaluated within two hours of admission and subsequently whenever symptoms suggestive of hypoglycaemia were observed. Infant feeding was commenced 4–6 hours after delivery, except in asphyxiated infants with respiratory distress who received only 10 per cent dextrose solution for the first 24 hours. Blood samples for glucose determination were obtained by venepuncture and analysed immediately, using the glucose-oxidase method.

Hypoglycaemia is defined according to Cornblath and Schwartz (1966) as blood sugar level below 20 mg per dl in infants weighing less than 2,500 gm

or less, and below 30 mg per dl in full term infants during the first 72 hours of life, and below 40 mg per dl thereafter.

Hypoglycaemic infants were treated with an intravenous bolus injection of 25 per cent glucose solution (4 ml. per kg. body weight). This was followed by an intravenous drip of a 10-per cent dextrose solution (85 ml. per kg. body weight) during the next 24 hours. If the blood sugar concentration was maintained above 30 mg. per dl, the glucose intake was reduced to 5 per cent during the second day.

Infant remaining symptomatic on the above regime and whose blood sugar values were less than 30 mg per dl were treated with prednisone, (1 mg./kg./day). When the blood sugar levels returned to normal values, prednisone was withdrawn gradually over a period of one week.

Results

The total admission into the Special-Care-Baby-Unit for the period was 1,061, and 392 of these were of low birthweight.

Seventy infants consisting of 39 males and 31 emaesa were hypoglycaemic. Thirty-one of the hypoglycemic infants weighed 2,500 g or less. The overall incidence of hypoglycaemia was 6.6. per cent and that for low birthweight infants was 7.9 per cent. There were 16 deaths associated with neonatal hypoglycaemia.

Table I presents the obstetric and perinatal problems found among the hypoglycaemic infants. Caesarian section was performed as an emergency procedure in 17 of the 27 infants who had this mode of delivery, and 14 of these were also asphyxiated. Asphyxia resulted mainly from malpresentations (breech in particular), cord pathology and uterine dysfunction.

Table II shows the age at diagnosis in the 70 hypoglycaemic infants, and also the age at which symptoms were first observed in the 25 infants who manifested symptoms that are attributable to hypoglycaemia. Fifty-three cases (76 per cent) were diagnosed within the first 24 hours of life, but in 14 infants (20 per cent), including 11 that were referred from peripheral hospitals and maternity homes, hypoglycaemia was not detected until after the age of 72 hours. Similarly, in the symptomatic group, 76 per cent of symptoms occurred within the first 24 hours of life while 20 per cent of symptoms did not manifest until after the age of 72 hours. It is noteworthy, however, that 15 (60 per cent) of the 25 symptomatic infants were also asyphyxiated.

The common presenting symptoms of hypoglycaemia in the present series were jitteriness, apnoea, cyanosis, refusal to feed and limpness

TABLE I

Obstetric and Perinatal Factors Associated with Neonatal Hypoglycaemia in 70 Neonates

Factor	*No. of Cases*	*Per cent of total*
Caesarean Section	27	38.6
Asphyxia (Apgar Score) <6	26	37.1
Pre-eclampsia	6	8.6
Prolonged Labour >24 hours	3	4.3
No Associated Factors	8	11.4
Total	70	100.0

TABLE II

Age at Diagnosis and at onset of Symptoms in 70 Hypoglycaemic infants

Age (Hrs.)	*Age at diagnosis No.*	*Age at onset of symptoms No.*
< 6	40	16
6—24	13	3
24—72	3	1
>72	14	5
Total	70	25

(Table III). These symptoms were promptly relieved by intravenous administration of dextrose solution.

Table IV compares the mean pretreatment glucose level, gestational age, and birthweight, in the asymptomatic and symptomatic groups of infants. The mean glucose value for the symptomatic group was significantly lower than for the asymptomatic group ($P < 0.001$), but both groups did not differ statistically in respect of their gestational age ($P > 0.1$). The mean birthweight of the asymptomatic infants was significantly higher than that for the symptomatic group ($t = 2.1$, $P < 0.05$).

Table V shows the pretreatment blood sugar concentrations in four categories of hypoglycaemic infants. The mean glucose value for the symptomatic vigorous infants (Apgar score > 7) was significantly higher than that of the symptomatic asphyxiated infants ($P < 0.05$). On the other hand, symptomatic asphyxiated infants had a mean blood sugar level of 9.9 mg. per dl which was significantly lower than the value for the asymptomatic asphyxiated infants ($P < 0.001$). Those infants who died within the immediate neonatal period had a mean pretreatment blood sugar concentration of 10.3 mg. per dl. This value was significantly lower than that found among the asymptomatic asphyxiated infants ($P < 0.001$), but corresponded well with the mean level in the symptomatic group ($P > 0.1$).

Twenty of the hypoglycaemic neonates in the present study had a total of 23 associated disorders. These consisted of hyperbilirubinaemia (9 cases), septicaemia (7 cases), hypocalcaemia (6 cases) and polycythaemia (1 case).

Of the 70 infants, seven were lost to follow up. The remaining 63 infants, followed for 3–24 months, remained well except for one infant with delayed milestones. This infant also developed afebrile convulsions at nine months of age and was spastic by 15 months of age.

TABLE III

Clinical Manifestations in 25 Neonates with Symptomatic Hypoglycaemia

Manifestation	*No. of cases*
Tremors (Jitteriness)	14
Apnoea and cyanosis	12
Lethargy and refusal to feed	6
Tachypnoea	2
Convulsior	2
Abdominal distension	1

TABLE IV

Comparison of Blood Glucose, Gestational Age and Birthweights in Symptomatic and Asymptomatic Hypoglycaemic Neonates

Type of hypoglycaemia	*No. of cases*	*+Pre-treatment blood glucose Mean ± ISD*	*≠Gestational age (wks) Mean ± ISD*	**Birthweight Mean ± ISD*
Asymptomatic	45	22.4±3.4	37.1±6	72.8±0.9
Symptomatic	25	12.7±8	36.2±5	2.14±1.4

+P<0.001 ≠P>0.1 *P<0.05

TABLE V

Pre-Treatment Blood Glucose Levels in various Categories of Hypoglycaemic infants

Category of Infants	*No.*	*Pre-treatment blood glucose Mean ± ISD*	
Symptomatic and vigorous (Apgar > 7)	10	17±7.6	P<0.05
Symptomatic and asphyxiated (Apgar <6)	15	9.9±7	P<0.001
Asymptomatic and asphyxiated	11	22.3±6.1	P<0.01
Dead	16	10.3±9.8	

Discussion

The overall frequency of hypoglycaemia among low birthweight infants in the present study was 7.9 per cent. This compares favourably with an incidence of 5.7 per cent reported by Pildes et al., (1967). However, our figure is much lower than that reported by Fluge (1974), who found an incidence of 15.4 per cent among a selected group of low birthweight infants who weighed less than 2,000 gm.A majority of infants in the present study (63.1 per cent) weighed more than 2,500 gm at birth and this may account, in part, for the lower incidence of hypoglycaemia. Furthermore, low birthweight infants in our unit routinely receive intravenous 10 per cent dextrose solution during the first 24 hours of life in addition to oral feeds.

Hypoglycaemia was not observed in infants of diabetic mothers in the present study. This may be because the few diabetic mothers encountered were in a stable metabolic and normoglycaemic state at delivery. Hypoglycaemia was also uncommon in infants of toxaemic mothers in the present series. During the study period 214 infants were delivered to pre-eclamptic mothers, and only 6 of these developed hypoglycaemia. This contrasts with the occurence of hypoglycaemia in over half of infants of toxaemic mothers in other series (Cornblath, *et al.*, 1964; Cornblath and Reisner, 1965). The lower incidence of hypoglycaemia in association with toxaemia of pregnancy observed in the present study may be related to two factors. Firstly, toxaemia of pregnancy occurs less commonly in tropical Africa than in Europe. Secondly, toxaemia of pregnancy among West Africans commonly take the acute form in contrast to the more chronic pattern observed among Europeans; therefore placental insufficiency with the attendant risk of intrauterine growth retardation is less likely to occur (Lawson and Stewart, 1967).

A history of neonatal asphyxia was found in 26 of the hypoglycaemic infants in the present study, and this may be consequent on rapid depletion of hepatic glycogen associated with oxygen deprivation (Evans and Glass, 1976).

The observation by Fluge (1974) that pretreatment blood glucose concentration was significantly lower in symptomatic than in asympto

matic infants has been confirmed by this study. There is no doubt, however, that asymptomatic hypoglycaemic infants require treatment since hypoglycaemia per se represents a potential danger to the developing brain (Beard, *et al.*, 1971).

Of interest is the occurrence of hypoglycaemia in seven septicaemic infants in the present study. The organisms most commonly incriminated are gram-negative bacteria (Yeung, 1970; Yeung, Lee and Yeung 1973). It has also been noted that hypoglycaemia in neonatal sepsis is mainly due to decreased caloric intake by the ill neonate, and also increased peripheral glucose utilization. (Yeung, Lee, and Yeung, 1973).

The present study has confirmed the need for close monitoring of blood sugar levels among infants who are at high risk for hypoglycaemia, particularly asphyxiated infants; infants delivered by emergency caesarean section; low birthweight infants, and infants who show evidence of septicaemia.

References

Beard, A., Cornblath, M., Gentz, J., Kelleum, M., Person, B., Zetterstrom, R. and Haworth, J. C. (1971). Neonatal Hypoglycaemia: A discussion. *J. Pediat.*, **79,** 314–324.

Cornblath, M., Odell, G. B. and Levin, E. J. (1959). Symptomatic neonatal hypoglycaemia associated with toxaemia of pregnancy *J. Pediat.*, **55,** 545–562.

Cornblath, M. and Reisner, S. A. (1965). Blood glucose in the neonate and its clinical significance. *N. Engl. J. Med.*, **273,** 378–381.

Cornblath, M. and Schwartz, R. (1966). Disorders of carbohydrate metabolism in infancy. 1st Edition pp. 41–78, W. B. Saunders, Philadelphia, USA.

Cornblath, M., Wybregt, S. H., Baens, G. S. and Klein, R. I. (1964). Symptomatic neonatal hypoglycaemia. studies of carbohydrate metabolism in the newborn infant VIII. *Pediatrics,* **33,** 388–402.

Evans, H. and Glass, L. (1976). Perinatal Medicine. 1st Edit. pp. 251–257; Harper and Row, Hagerstown, Maryland, New York, San Francisco, London.

Farquhar, J. W. (1959). The child of the diabetic woman. *Arch. Dis. Childh.*, **34,** 76–79.

Fluge, G. (1974). Clinical aspects of neonatal hypoglycaemia. *Acta. Paediat. Scand.*, **63,** 826–832.

Haworth, J. C. and McRae, K. N. (1967). Neonatal hypoglycaemia: A six-year experience. *J. Lancet.*, **87,** 41–45.

Lawson, J. B. and Stewart, D. B. (1967). Obstetrics and Gynaecology in the Tropics and developing countries. 1st Edit. pp. 120–136, Edward Arnold Ltd. U.K.

Lubchenco, L. O. and Bard, H. (1971). Incidence of hypoglycaemia in newborn infants classified by birthweight and gestational age. *Pediatrics,* **47,** 831–838.

Pildes, R., Forbes, A. E., O'Connor, S. M., and Cornblath, M. (1967). The incidence of neonatal hypoglycaemia—a completed survey *J. Pediat.*, **70,** 76–80.

Yeung, C. Y. (1970). Hypoglycaemia in neonatal sepsis. *J. Pediat.*, **77,** 812–819.

Yeung, C. Y., Lee, V. W. Y. and Yeung, C. M. (1973). Glucose disappearance rate in neonatal infection. *J. Pediat.*, **82,** 486–489.