Incidence, Predictors, and Outcomes of Hypoglycemia Among At-Risk Neonates

Praneet Kumar, Shakal Narayan Singh, Shalini Tripathi*, Mala Kumar

Abstract

Background and Aim: Hypoglycemia is less studied in healthy at-risk neonates compared with hospitalized neonates, especially in the first few days of life. Hypoglycemia can have serious neurologic implications later in life. This study was conducted to assess the incidence, predictors, and outcomes of hypoglycemia among at-risk neonates and its neurologic implications during the first few months of life.

Materials and Methods: At-risk neonates were screened for hypoglycemia at 2, 6, 12, 24, 48, and 72 hours of life. They were subjected to neurodevelopmental assessment at 3 and 6 months of life and were evaluated based on the Rashtriya Bal Swasthya Karyakram developmental scale.

Results: Of the 483 neonates, 65 (13.5%) neonates had hypoglycemia. Low birth weight, small for gestational age, maternal hypothyroidism, maternal chorioamnionitis, delayed first feed, feeding of milk other than breast milk, low frequency of feed, > 4 hours of interval between night feeds, lack of support from health worker for early feeding initiation, and maternal sedation were predictors of hypoglycemia. At the 3-month follow-up, 4 of the 55 neonates showed a developmental delay, which was observed at 6 months too. Vision (3/4) was the most commonly affected domain.

*Correspondence

Dr Shalini Tripathi
Additional Professor
Department of Pediatrics
King George's Medical University
Lucknow 226003, Uttar Pradesh
India

E-mail: shalini.avasthi@rediffmail.com

Conclusion: The incidence of hypoglycemia in at-risk neonates was comparable with that in hospitalized neonates. Maternal risk factors and faulty feeding habits increased the hypoglycemic episodes. Neonates who developed hypoglycemia need to be monitored for developmental delay, especially for vision-related problems.

Key Words: Blood glucose level, Rashtriya Bal Swasthya Karyakram developmental scale, low birth weight, small for gestational age, maternal hypothyroidism, gestational diabetes mellitus, chorioamnionitis, jitteriness

Introduction

Hypoglycemia is a frequently encountered metabolic problem in neonates.[1,2] After birth, neonates lose the continuous supply of transplacental glucose. A high brain-to-body weight ratio along with developmental immaturity of adaptive mechanisms such as gluconeogenesis and glycogenolysis further increase the chances of hypoglycemia in neonates. Symptoms of hypoglycemia are nonspecific and include feeding difficulties, tachypnea, hypotonia, abnormal cry, jitteriness, apnea, coma, and convulsions. Prolonged and repeated episodes of hypoglycemia are associated with poor neurologic outcomes.[3,4] The at-risk groups—neonates who are born premature, small for gestational age (SGA), have low birth weight (LBW), and whose mothers have diabetes—should be monitored for their blood glucose level (BGL) routinely, especially, in the early hours of life.[5-7] The incidence of hypoglycemia is variable and depends on the criteria used in different studies.[6,8,9] According to the National Neonatal-Perinatal Database, in India, the incidence of hypoglycemia was 0.6% among intramural neonates and 3.2% among extramural neonates.[10] The effect of neonatal hypoglycemia is high in developing countries, where neonatal mortality accounts for 50% to 60% of neonatal deaths.[11] The proportion of SGA neonates is higher in the developing countries than that in the developed countries. As universal screening of BGL may not be possible, there is a need to identify neonates who are at high risk of developing hypoglycemia, especially in resource-limited countries such as India.[3]

Aim

To assess the incidence and predictors of hypoglycemia among at-risk neonates and monitor their neurodevelopment at 3 and 6 months of life

Materials and Methods

Study design

This prospective, observational study was conducted in the neonatal unit of King George's Medical University (Lucknow, Uttar Pradesh, India), a tertiary care teaching hospital, from September 2016 to August 2017. A total of 483 healthy neonates were enrolled into this study. Ethical clearance was obtained from the institutional ethics committee, and informed consent was obtained from the parents.

Inclusion criteria

Neonates of mothers with diabetes, large-for-gestational age neonates (birth weight > 90th centile), SGA neonates (birth weight < 10th centile), preterm neonates (< 37 wk of gestation), and LBW neonates (weighing < 2500 g) were considered for this study.

Exclusion criteria

Neonates with major congenital malformations, hypoxic–ischemic encephalopathy stage 3, meningitis, and acute bilirubin encephalopathy were excluded. Neonates whose parents did not consent for the study

and were not willing to attend follow-ups, and hospitalized neonates were excluded.

Sample size calculation

Sample size was calculated based on the study by Harris et al.[12] To calculate a 95% confidence interval for a proportion with a margin of error not more than 5%, the sample size has to be 384. It was increased by 25% to compensate for loss to follow-up.

Study procedure

Mothers were counseled about the neonatal feeding schedule. Feeding was initiated in the first hour of life and thereafter every 2 to 3 hours or as required. The BGL in neonates was estimated at 2, 6, 12, 24, 48, and 72 hours of life. Neonates were monitored for clinical symptoms for a minimum of 72 hours, and those with on-going risk were monitored thereafter too. Hypoglycemia was defined as BGL < 45 mg/dL (< 2.2 mmol/L) according to the WHO's criterion.[13] Severity of hypoglycemia was graded as mild, if the BGL was 40 to 44 mg/dL; moderate, if the BGL was 30 to 39 mg/dL; and severe, if the BGL was < 30 mg/dL.[5]

Detailed data of all the enrolled neonates including weight, gestational maturity, sex, feeding details (ie, time of initiation of the first feed, type, duration, frequency of feeding, and night feeds), clinical symptoms, treatment details, any complications, duration of hospital stay, and outcome were recorded. Maternal data including age, parity, socioeconomic status, antenatal care, blood group, and metabolic conditions such as diabetes and thyroid disorder were recorded. Data about pregnancy-related conditions including gestational diabetes mellitus, antepartum hemorrhage, pregnancy-induced hypertension, antenatal corticosteroid status, chorioamnionitis (ie, maternal fever, foul smelling discharge, color of liquor, uterine tenderness, maternal tachycardia, fetal tachycardia, and leukocyte count), and intrapartum details such as evidence of fetal distress, prolonged labor, obstructed labor, any medications received, and mode of delivery were also recorded. After delivery, information about pain and sedation in the mother and availability of a lactation counselor was also recorded. Neonates who developed hypoglycemia were treated according to the unit protocol.

The BGL was measured using glucometer (On Call Plus Glucometer, SJV Scientific Company, Coimbatore, Tamil Nadu, India) and glucose oxidase method. In case of low BGL, the blood samples were collected in fluoride vials and assessed using reflectance spectrophotometry.

Developmental assessment of the neonates was done at 3 and 6 months using the Rashtriya Bal Swasthya Karyakram (RBSK) developmental scale.[14] Accordingly, each neonate was assessed for 6 domains: gross motor, fine motor, hearing, speech, vision, and cognition and socialization. A delay in the development was noted when an infant failed to achieve the age-specific domain characteristics.

A brain magnetic resonance imaging (MRI), brainstem evoked response audiometry (BERA), visual evoked potential (VEP), and electroencephalography (EEG) were done in neonates with symptomatic hypoglycemia.

Statistical analyses

Data were summarized as mean (SD) for quantitative/continuous data and proprotion/percentages for qualitative/categorical data. Categorical data were analyzed using the χ^2 test or Fisher's exact test, and continuous data were analyzed by *t* test or Mann–Whitney *U* test. A *P* value of ≤ .05 was considered statistically significant. Relevant variables ($P < .05$) were included in the multivariate logistic regression model to identify independent predictors of hypoglycemia. Statistical analysis was done using SPSS version 23 (IBM Corp, Armonk, NY, USA).

Results

Of the 483 enrolled neonates, 65 (13.5%) were hypoglycemic. Of these 65 neonates, 9 (13.8%) had severe, 53 (81.5%) had moderate, and 3 (4.7%) had mild hypoglycemia. A single episode of hypoglycemia was noted in 40 (61.5%) of the 65 neonates, whereas 25 (38.5%) of them had recurrent episodes. The number of neonates who developed hypoglycemia at 2 hours of life were 35/65; at 6 hours, there were 19/65 (6 neonates had new episodes); at 12 hours, there were 15/65 (10 had new episodes); at

24 hours, there were 11/65 (10 had new episodes); at 48 hours, there were 8/65; and at 72 hours, there were 7/65. Of the 65 neonates, 11 (16.9%) neonates were symptomatic and 54 (83.1%) neonates were asymptomatic. Jitteriness was the most frequent symptom.

Table 1 presents a comparison of the neonatal and maternal baseline and clinical characteristics between neonates with and without hypoglycemia.

Table 1. Baseline and Clinical Characteristics of the Mothers and the Neonates

Characteristic	Hypoglycemia, *n* = 65	No Hypoglycemia, *n* = 418	*P* Value
Birth Weight, g (Mean ± SD)	2329 ± 629	2466 ± 647	.119
Weight for Gestational Age, *n* (%)			
SGA	36 (55.4)	122 (29.2)	< .001
AGA	21 (32.3)	250 (59.8)	
LGA	8 (12.3)	46 (11.0)	
Gestational Category, *n* (%)			
Preterm	32 (49.2)	172 (41.1)	< .001
Term	33 (50.8)	246 (58.9)	
Sex, *n* (%)			
Male	33 (53.8)	255 (61.4)	.181
Female	32 (46.2)	163 (32)	
Parity, *n* (%)			
Uniparous	38 (58.5)	235 (56.2)	.735
Multiparous	27 (41.5)	183 (43.8)	
Socioeconomic Status, *n* (%)			
Low	26 (40)	197 (47.1)	.56
Middle to upper	39 (60.0)	221 (52.9.0)	
Mode of Delivery, *n* (%)			
Vaginal	30 (46.2)	256 (61.2)	.021
LSCS	35 (53.8)	162 (38.8)	
Maternal Education, *n* (%)			
Uneducated	25 (38.5)	130 (31.1)	.609
Educated	40 (61.5)	288 (68.9)	
ANC Visits, *n* (%)			
Incomplete	34 (52.3)	203 (48.6)	.574
Complete (> 4)	31 (47.7)	215 (51.4)	
Maternal Gestational Characteristics			
Mother with hypothyroidism, *n* (%)	6 (9.2)	7 (1.7)	.004
Clinical chorioamnionitis, *n* (%)	15 (23.1)	15 (3.6)	< .001
GDM detection, wk (Mean ± SD)	28.9 ± 2.1	30.9 ± 3.7	.003
GDM, *n* (%)	30 (46.2)	128 (30.6)	.013
Prolonged labor, *n* (%)	4 (6.2)	3 (0.7)	.008
Fetal distress, *n* (%)	12 (18.5)	41 (9.8)	.038

AGA, appropriate for gestational age; ANC, antenatal care; GDM, gestational diabetes mellitus; LGA, large for gestational age; LSCS, lower segment cesarean section; SGA, small for gestational age.

The proportions of SGA neonates (55.4% vs 29.2%; $P < .001$) and preterm neonates (49.2% vs 41.1%; $P = .01$) were significantly higher in the hypoglycemia group compared with that in the nonhypoglycemia group. The incidence of hypoglycemia was significantly high in neonates born to mothers with hypothyroidism (9.2% vs 1.7%) and chorioamnionitis.

More number of neonates born to women with GDM had hypoglycemia (46.2%) compared with the number of neonates born to women without GDM (30.6%) ($P < .05$). The mean gestational age for the detection of diabetes mellitus was significantly lower in women who delivered neonates with hypoglycemia than those who delivered neonates without hypoglycemia.

Intrapartum parameters such as prolonged labor and fetal distress were found to be significantly associated with hypoglycemia in the neonates ($P < .05$). The incidence of hypoglycemia was higher in neonates born through cesarean delivery compared with those born through vaginal delivery (53.8% vs 46.2%) ($P < .05$).

A comparison of the feeding-related parameters among the hypoglycemia and the nonhypoglycemia groups showed that a delay of > 1 hour of the first feed (71.8% vs 29.2%); feeding of milk other than breast milk (69.4% vs 27.6%); neonates not being breastfed directly (38.8% vs 9.8%); poor latching to the breast (30.0% vs 2.2%), frequency of feeds (ie, < 8 times a day [26.6% vs 2.2%]); and lack of early support from a health care worker in initiating breastfeeding within the first 2 hours of birth (78.5% vs 59.8%) were significantly

associated with hypoglycemia ($P < .05$). Other parameters such as maternal sedation and inadequate breast milk as perceived by the mother were also significantly common in the hypoglycemia group.

Upon performing multivariate logistic regression analysis, it was observed that LBW, SGA, maternal hypothyroidism, chorioamnionitis, a delay of > 1 hour of the first feed, lack of direct breastfeeding as the primary feeding method, inadequate breast milk as perceived by the mother, feeding frequency < 8 times/day, > 4 hours of interval between night feeds, lack of support from a health worker for the early initiation of breastfeeding, and maternal sedation were the independent predictors of neonatal hypoglycemia as shown in Table 2.

Table 2. Predictors of Hypoglycemia as Determined Using the Logistic Regression Analysis

Predictor/Variable	β-Coefficient	*P* Value	Adjusted OR	95% CI
LBW	3.124	.003	22.727	2.911–177.467
SGA	3.232	< .001	25.328	4.542–141.230
Maternal Hypothyroidism	2.750	.019	15.647	1.567–156.243
Clinical Chorioamnionitis	2.101	.040	8.177	1.096–60.982
Interval of > 4 h Between Night Feeds	2.124	.003	8.364	2.013–34.757
Lack of Support From Health Care Worker for the Initiation of Breastfeeding Within 2 h of Birth	3.246	0	25.699	5.725–115.357
Inadequate Breast Milk	2.128	.001	8.395	2.362–29.836
> 1 h Delay in the First Feed	2.291	< .001	9.887	3.170–30.837
Lack of Direct Breastfeeding	2.080	.004	8.002	1.956–32.746
Feeding Frequency < 8 Times/d	3.360	.001	28.783	3.996–207.323

LBW, low birth weight; SGA, small for gestational age.

In majority (73.8%) of the neonates with hypoglycemia, the BGL got normalized with regular feeding itself, whereas 26.1% of the neonates with hypoglycemia required glucose infusion. The mean duration of glucose infusion was 58.6 ± 49.4 hours.

Neonatal outcomes at 3 and 6 months of life

Of the 65 neonates with hypoglycemia, 10 were lost to follow-up at 3 months. Of the 55 neonates who were followed up, 51 neonates were developmentally normal, whereas 4 neonates showed a delay in the developmental domains. These 4 neonates continued to have a developmental delay at 6 months of age as well. At 6 months, 5 more neonates were lost to follow-up. Of the 50 neonates who were followed up, 46 were neurologically sound at 6 months of age. Vision was the most frequently affected domain in symptomatic neonates—poor eye-to-eye contact at 3 months and lagging while following objects at 6 months were observed. The speech domain was not affected. The results of the brain MRI, BERA, VEP, and EEG screening were as follows:

Brain MRI: At 3 months of age, 3 of the 10 neonates and at 6 months of age, 1 of the 7 neonates had an abnormal MRI report. Signal alteration in the parieto-occipital area and age-inappropriate atrophic changes in the parieto-occipital area, internal capsule, and basal ganglia were noted.

BERA: An increased interpeak latency and threshold were seen in 3 of the 31 neonates followed up at 3 months and 1 of the 16 neonates followed up at 6 months of age.

VEP: At 3 months, 9 of the 23 neonates had an abnormal VEP, and at 6 months, 7 of the 22 neonates had an abnormal VEP.

EEG: Of the 25 neonates with hypoglycemia, 3 had epileptiform discharges at 3 months and 1 of these 3 neonates had persistent epileptiform discharges even at 6 months of age.

Discussion

A variation can be noted in the incidence of hypoglycemia reported in earlier studies because of the differences in the inclusion criteria, methods of testing, and definition of hypoglycemia. In this study, we found the incidence of hypoglycemia in the at-risk group to be 13.5%.

This is in line with the incidence reported in previous studies.[5,14] However, Harris et al,[12] in their study, found 51% of at-risk neonates to be hypoglycemic, which widely differs from that of our study.

In neonates, the BGL decreases drastically in the first 1 to 2 hours of life and then increases and stabilizes gradually. The presence of associated risk factors can cause a delay in the early stabilization of BGL, leading to frequent episodes of hypoglycemia. In this study, the majority of the episodes occurred within the first 24 hours of life because of the associated risk factors. Similar results were observed in previous studies too.[5,15-17] We found a higher incidence of hypoglycemia in SGA neonates (55.4%) compared with that reported in other studies.[15,17,18] The increase in the proportion of SGA in India and standardized BGL monitoring could be the reasons for this high incidence. In this study, the incidence of hypoglycemia was higher in preterm neonates than in term neonates, which is similar to the data reported in other studies.[19,20] Also, factors such as faulty and delayed feeding and sepsis significantly affected the incidence of hypoglycemia. Nonmodifiable risk factors such as maternal hypothyroidism and GDM also played a significant role in causing hypoglycemia. These findings are similar to that reported in previous studies.[21-23]

Exclusive breastfeeding is necessary to meet the metabolic needs of neonates, and any delay in the early establishment of breastfeeding (eg, supplementing with water, glucose water, or formula instead of breast milk) predisposes neonates to hypoglycemia. Not only delayed initiation of breastfeeding, but also other variables that have not been discussed in previous studies such as not using breastfeeding as the primary feeding method, poor latching to the breast, decreased frequency of feeding, and mothers' perception of having inadequate breast milk were observed to be the predictors of neonatal hypoglycemia in this study. Besides, we found that the lack of support from health care workers for the initiation of early breastfeeding could also be a contributing factor for the increased incidence of neonatal hypoglycemia.

Jitteriness was the predominant sign in symptomatic cases, which is similar to that reported in other studies.[24-26] Using the standard protocol for treating hypoglycemia, we found that regular feeding was sufficient to correct hypoglycemic episodes in the majority of the neonates. This shows the significant role of early establishment of breastfeeding in supporting metabolic adaptation in a neonate and reducing the incidence of hypoglycemia.

Furthermore, persistent or recurrent hypoglycemia in neonates can result in brain injury, cognitive impairment, vision disturbance, and other long-term consequences.[27-29] In this study, we found that most of the hypoglycemic episodes did not result in significant neurodevelopmental problems. The majority (> 90%) of the neonates with hypoglycemia were developmentally normal. But, the neonates who had recurrent hypoglycemia showed difficulty in making proper eye contact and following an object, when assessed at 3 months of age. This indicates an impairment in the vision domain that resulted due to the occipital lobe injury. In the neonates with severe and recurrent hypoglycemic episodes, the MRI reports showed the involvement of the parieto-occipital region, as seen in other studies.[30,31]

This study had a few limitations. Although this was a prospective study, there was loss to follow-up. Also, MRI and other tests for developmental assessments could not be done in many of the neonates because of resource constraints.

Conclusion

In this study, we found that prematurity, SGA, LGA, LBW, and GDM increased the risk of hypoglycemia in neonates, especially in the early hours of life when the metabolic adaptation occurs. Thus, active screening of these at-risk neonates, even before the appearance of symptoms of hypoglycemia, is justifiable. In the at-risk neonates, special attention should be paid to initiate feeding within the first hour of birth, either through direct breastfeeding or expressed breast milk with the active involvement of a health care worker. Also, care must be taken to ensure that the interval between 2 consecutive feeds does not exceed beyond 4 hours, especially at night. Further, neonates who develop hypoglycemia need to be monitored for developmental delays, seizures, and hearing and vision problems during follow-ups.

References

1. Cornblath M. Neonatal hypoglycemia 30 years later: does it injure the brain? Historical summary and present challenges. *Acta Paediatr Jpn*. 1997;39(Suppl 1):S7–S11.
2. Burris HH. Hypoglycemia and hyperglycemia. In: Cloherty JP, et al, eds. *Manual of Neonatal Care*. 7th ed. PA, USA: Lippincott Williams & Wilkins; 2008:312–326.
3. Cornblath M, et al. Controversies regarding definition of neonatal hypoglycemia: suggested operational thresholds. *Pediatrics*. 2000;105(5):1141–1145.
4. Rozance PJ, Hay WW. Hypoglycemia in newborn infants: features associated with adverse outcomes. *Biol Neonate.* 2006;90(2):74–86.
5. Maayan-Metzger A, Lubin D, Kuint J. Hypoglycemia rates in the first days of life among term infants born to diabetic mothers. *Neonatology.* 2009;96(2):80–85.
6. Zhou W, et al. Hypoglycemia incidence and risk factors assessment in hospitalized neonates. *J Matern Fetal Neonatal Med.* 2015;28(4):422–425.
7. Lodhi MA, Shah NA, Shabir G. Risk factors associated with neonatal hypoglycemia. *Prof Med J.* 2006;16:687–690.
8. Holtrop PC. The frequency of hypoglycemia in full-term large and small for gestational age newborns. *Am J Perinatol.* 1993;10(2):150–154.
9. Ashworth A, Waterlow JC. Infant mortality in developing countries. *Arch Dis Child.* 1982;57(11):882–884.
10. National Neonatology Forum NNPD Network. *National Neonatal-Perinatal Database: Report 2002-2003*. New Delhi, India: All India Institute of Medical Sciences; 2005.
11. Dashti N, Einollahi N, Abbasi S. Neonatal hypoglycemia: prevalence and clinical manifestations in Tehran Children's Hospital. *Pakistan J Med Sci.* 2007;23(3):340–343.
12. Harris DL, Weston PJ, Harding JE. Incidence of neonatal hypoglycemia in babies identified as at risk. *J Pediatr.* 2012;161(5):787–791.
13. World Health Organization. *Hypoglycaemia of the Newborn. Review of the Literature.* Geneva, Switzerland: World Health Organization; 1997:1–61.
14. Ministry of Health & Family Welfare, Government of India. Rashtriya Bal Swasthya Karyakram (RBSK). https://nhm.gov.in/images/pdf/programmes/RBSK/Resource_Documents/RBSK_Job_Aids.pdf. Accessed January 6, 2022.
15. Singh YP, et al. Hypoglycemia in newborn in Manipur. *J Med Soc.* 2014;28(2):108–111.
16. Hawdon JM, Ward Platt MP, Aynsley-Green A. Patterns of metabolic adaptation for preterm and term infants in the first neonatal week. *Arch Dis Child.* 1992;67(4 Spec No):357–365.
17. Sasidharan CK, Gokul E, Sabitha S. Incidence and risk factors for neonatal hypoglycaemia in Kerala, India. *Ceylon Med J.* 2004;49(4):110–113.
18. Bhat MA, et al. Hypoglycemia in small for gestational age babies. *Indian J Pediatr.* 2000;67(6):423–427.
19. Burdan DR, Botiu V, Teodorescu D. Neonatal hypoglycemia-the incidence of the risk factors in Salvator Vuia obstetrics-gynecology hospital, Arad. *Timisoara Med J.* 2009;59:77–80.
20. De AK, et al. Study of blood glucose level in normal and low birth weight newborns and impact of early breast feeding in a tertiary care centre. *Ann Nigerian Med.* 2011;5(2):53.
21. Pal D, et al. Neonatal hypoglycaemia in Nepal 1. Prevalence and risk factors. *Arch Dis Child Fetal Neonatal Ed.* 2000;82(1):F46–F51.
22. Raivio KO, Österlund K. Hypoglycemia and hyperinsulinemia associated with erythroblastosis fetalis. *Pediatrics.* 1969;43(2):217–225.
23. Betti M, et al. Neonatal outcome in newborns from mothers with endocrinopathies. *Gynecol Endocrinol.* 2011;27(4):248–250.
24. Misra PK, Sharma B. Hypoglycemia in newborns-a prospective study. *Indian Pediatr.* 1977;14(2):129–132.
25. Bhand SA, et al. Neonatal hypoglycemia; presenting pattern and risk factors of neonatal hypoglycemia. *Prof Med J.* 2014;21(04):745–749.
26. Dhananjaya CD, Kiran B. Clinical profile of hypoglycemia in newborn neonates in a rural hospital setting. *Int J Biol Med Res.* 2011;2(4):1110–1114.
27. Brand PLP, et al. Neurodevelopmental outcome of hypoglycaemia in healthy, large for gestational age, term newborns. *Arch Dis Child.* 2005;90(1):78–81.
28. McKinlay CJD, et al. Association of neonatal glycemia with neurodevelopmental outcomes at 4.5 years. *JAMA Pediatr.* 2017;171(10):972–983.
29. Udani V, et al. Neonatal hypoglycemic brain injury - a common cause of infantile-onset remote symptomatic epilepsy. *Indian Pediatr.* 2009;46(2):127–132.
30. Barkovich AJ, et al. Imaging patterns of neonatal hypoglycemia. *Am J Neuroradiol.* 1998;19(3):523–528.
31. Beverley DW, et al. Relationship of cranial ultrasonography, visual and auditory evoked responses with neurodevelopmental outcome. *Dev Med Child Neurol.* 1990;32(3):210–222.

Authors' Affiliations

Dr Praneet Kumar, Resident; **Dr Shakal Narayan Singh**, Professor; **Dr Shalini Tripathi**, Additional Professor; **Dr Mala Kumar**, Professor, Department of Pediatrics, King George's Medical University, Lucknow 226003, Uttar Pradesh, India