

MRI Brain Changes in Neonatal Hypoglycemia in a Rural Tertiary Care Centre

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Abstract

Introduction: Approximately half of a newborn baby's energy needs are fulfilled by glucose during birth, and glucose is an essential fuel for brain metabolism. The purpose of this study is to evaluate the neuroimaging characteristics of hypoglycaemia in neonates using MRI.

Methods: A prospective observational study was conducted among 30 neonates who were enrolled in the study after taking informed written consent from parents or attendant accompanying the neonate in the Department of Paediatrics of SHKM GMC, Nalhar, Nuh. All babies with hypoglycemia underwent MRI brain when they were stabilized at discharge. All babies' MRI changes were recorded.

Results: In our study Neonatal Hypoglycaemia was found more commonly in 34-37 weeks of gestational age(50%), specially in appropriate for gestational age babies(90%) compared to term babies. Majority of the cases were Male(63.3%) with majority of normal vaginal mode of delivery with Birth weight between 1.5-2.5 kg in most cases(63.3%).Maximum patients had Lower Blood Glucose level between 20-40 mg/dl (80%), with sepsis with meningitis(30%) as most common cause of hypoglycaemia. In our study Lowest blood glucose level observed was 11 mg/dl which was associated with abnormal MRI Brain change (Focal hemorrhagic lesions over parietal and occipital regions).Most common abnormal MRI Brain change were observed in parietooccipital region with

Sepsis with meningitis as most common cause of hypoglycemia in these patients. Sepsis was present in most cases with abnormal MRI findings (10 cases) among which 9 cases were positive for Meningitis, 2 of them had proven sepsis (Acinetobacter species). 1 patient was found to have prolonged hypoglycemia who received GIR above 12 and steroids, associated with abnormal MRI finding Periventricular Leukomalacia.

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Conclusion: This study had shown that, MRI results suggested aberrant hyperintensity in regions of brain. The lesions ranged from hyperintensity, signal intensity and hemorrhages across the parietal and occipital regions.

Keywords: Hypoglycaemia, occipital region, haemorrhage, periventricular leucomalacia, Sepsis

Introduction

Approximately half of a newborn baby's energy needs are fulfilled by glucose during birth, and glucose is an essential fuel for brain metabolism. For over a century, blood sugar levels have been monitored in newborns, and neonatal Hypoglycaemia was initially documented eighty years ago.¹ Nonetheless, there is a dearth of convincing data and disagreement regarding the definition of newborn Hypoglycaemia.² Neonatal blood glucose concentrations often follow a distribution that is influenced by birth weight, postnatal age, dietary intake, and additional variables. Some babies with low blood glucose levels experience clinical indications, but these are not very specific.

Hypoglycaemia is one of the most common metabolic problems seen in both the newborn nursery and neonatal intensive care unit (NICU). Many babies with low blood sugar have no clinical symptoms, while others have symptoms that can cause permanent brain damage. Data have shown that if the neonatal hypoglycaemia is not timely and properly treated, the infants may develop permanent brain injury, namely, neonatal hypoglycaemic encephalopathy the neonatal brain develops rapidly. Persistent or recurrent hypoglycaemia may lead to long-term visual disturbance, hearing impairment, cognitive abnormalities, secondary epilepsy, and other disorders in the central nervous system.⁴

The most common abnormal findings were located on the parietal and occipital lobes of the brains.⁶

Neonatal Hypoglycaemia can also be manifested as haemorrhage or middle cerebral artery infarction and parietooccipital cortex injury, although the basal ganglia and thalamus can also be involved.⁷

Brain injury related to neonatal hypoglycaemia seldom involves deep grey matter nuclei.

Neonatal hypoglycemia can show more abnormal imaging findings than expected. The brain injury patterns seen on imaging vary, and the severity of

hypoglycemia does not always match the extent of the injury.⁶

Hypoglycaemia in neonates usually affects occipital region of the brain and occipital region of brain can be assessed by VEP (visually evoked potential) & MRI.

The severity of neonatal hypoglycemia does not significantly correlate with specific MRI injury patterns. Furthermore, MRI findings in symptomatic neonatal hypoglycemia are heterogeneous, indicating diverse underlying mechanisms of injury. Abnormal imaging findings in hypoglycaemia of newborns may be more common than the insult suspected. Neonatal hypoglycaemia is prone to occur and requires vigilant management because of the risk factors for hypoglycaemia, which include preterm, small for GA or intrauterine growth retardation, maternal diabetes mellitus, or large for GA.⁸

Need for study:

As discussed, neonatal hypoglycaemia is more common than is believed and may lead to more serious neurological sequelae. The purpose of this study is to evaluate the neuroimaging characteristics of hypoglycaemia in neonates using MRI.

Aim and Objectives

AIM: To assess Neuroimaging Patterns in Neonatal Hypoglycaemia with the help of MRI .

OBJECTIVE: To evaluate all the MRI images of Brain in Neonates with Hypoglycaemia

Material and Methods

A prospective observational study was conducted among 30 neonates who were enrolled in the study after taking informed written consent from parents or attendant accompanying the neonate in the Department of Paediatrics of tertiary care center of rural North India. This study was conducted for a period of 1 year. Clearance from institutional ethics committee was obtained and an informed consent form was obtained from all the parents. The inclusion and exclusion criteria were as follows:

Inclusion Criteria:

- All neonates with hypoglycaemia admitted in the Paediatric Department of tertiary care center of rural north India.

Exclusion Criteria:

- Neonates with kernicterus
- Neonates with severe perinatal asphyxia
- Neonates with brain malformations

Methodology

All neonates who were at risk of hypoglycaemia (Preterm, Neonatal Sepsis, Small for Gestational Age(SGA), Infant of Diabetic Mother (IDM), Perinatal asphyxia with HIE-1 and HIE-2) were monitored for blood glucose level by glucometer (Accusure) and one reading of glucometer was confirmed by laboratory value daily.

Criteria for Diagnosis of Hypoglycemia:

A blood glucose levels of less than 45mg/dl was defined as hypoglycaemia.

MRI brain image used for this study was plain MRI T2 weighted, FLAIR Sequences and DWI band contrast enhanced with T1 weighted imaging using Philips multiva 1.5T-16 channel MRI with FMRI. All the Expenses of MRI Brain was covered under "Janani Sishu Suraksha Karyakram".⁴³

Statistical analysis

All the data was checked, edited and coded for computer entries. Two groups were made, one with no MRI brain changes & other with MRI brain changes. Paired t-test was applied with the data. After compilation of collected data, Data was entered in Excel and compiled and analyses by using Statistical Program for Social Sciences (SPSS) Version 20. Appropriate statistical tests were used during analysing the data. A p value of less than 0.05 was considered as statistically significant.

Power analysis calculation-

Sample size

The sample size was calculated by using the following formula,

$$n = Z^2pq / d^2$$

n= sample size

Z = 1.96= Standard normal deviate

p = Expected proportion in population Incidence of hypoglycaemia in neonates according to Roeper et al=15%

d= Absolute error or precision = 15%

$$n = 1.96 \times 1.96 \times 0.15 \times 0.85 / 0.15 \times 0.15 = 0.489804 / 0.0225 = 23.94$$

Although the minimum required sample size was calculated to be 24 participants, the decision was made to include 30 participants.

Ethical Consideration

All neonates were enrolled in study after obtaining informed consent from parents. No extra blood sampling was done. Investigations and treatment was done as per standard protocol.

Results

A total of 30 neonates were included in this study. The key findings include: Most consistently involved area was found to be parieto occipital regions.

Table 1: MRI Findings

| MRI FINDING | FREQUENCY | PERCENT |
|-------------|-----------|---------|
| NORMAL | 18 | 60% |
| ABNORMAL | 12 | 40% |
| TOTAL | 30 | 100 |

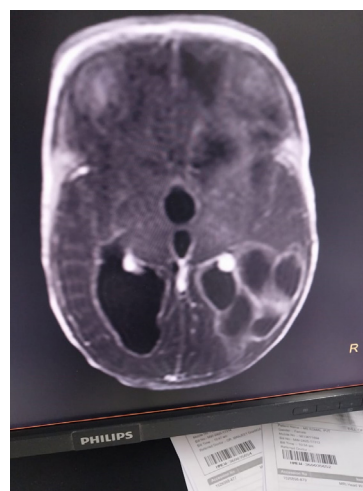


Figure 1: MRI Images showing axial section showing multiple cystic lesions in the brain, likely within the parietal and occipital lobes

Table 2: Abnormal MRI findings

| MRI findings | Frequency | Percent |
|---|-----------|---------|
| Abnormal hyperintensity in parietal and occipital lobes. | 1 | 8.3 |
| Abnormal Hyperintensity in the globi pallidi and mixed hypointensity and hyperintensity in occipital cortex and white matter. | 1 | 8.3 |
| Abnormal intensities over basal ganglia with posterior limb of internal capsule involvement | 1 | 8.3 |
| Abnormal signal intensity with cortical infarction and hemorrhage over parieto occipital region | 1 | 8.3 |
| Bilateral areas of increased signal in posterior limb of internal capsule, cerebral cortex, hippocampus and basal ganglia | 1 | 8.3 |
| Focal hemorrhage in white matter involving bilateral occipital lobes | 1 | 8.3 |
| Focal hemorrhagic lesions over parietal and occipital Regions | 1 | 8.3 |
| Hyperintensity in parietal and occipital lobes and progressive parenchymal loss of predominant occipital lobe. | 1 | 8.3 |
| large areas of encephalomalacia in parietooccipital regions bilaterally. | 1 | 8.3 |
| Periventricular leukomalacia | 1 | 8.3 |
| Periventricular leukomalacia ?hypoxic insult | 1 | 8.3 |
| White matter injury with focal hemorrhage in occipital and parietal lobes. | 1 | 8.3 |
| Total | 12 | 100 |

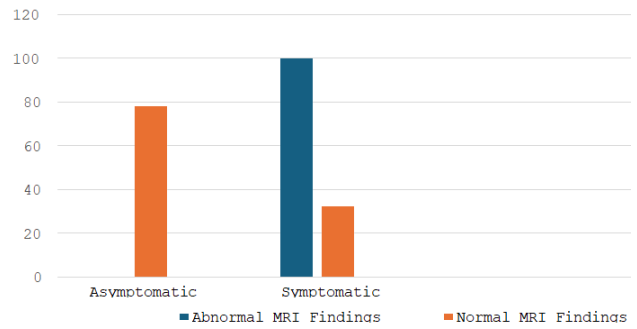


Figure 2. Distribution of the study group according to symptoms

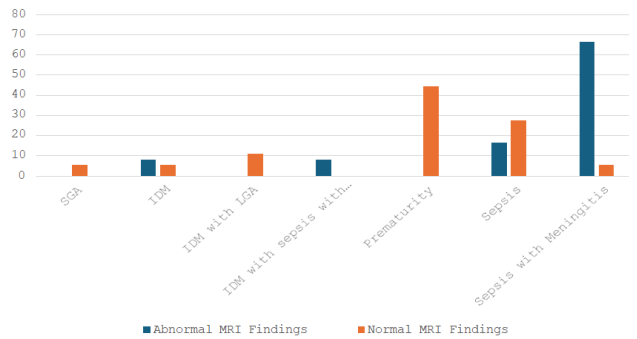


Figure 3. Distribution of the study group according to Cause of Hypoglycaemia

Discussion

During birth, glucose meets around half of a newborn baby’s energy needs, and it is a vital fuel for brain metabolism. Newborn blood sugar levels have been tracked for more than a century, and neonatal hypoglycaemia was first reported eighty years ago.¹ However, there is a lack of compelling evidence and controversy surrounding the definition of hypoglycaemia in neonates.² Neonatal blood glucose concentrations frequently exhibit a distribution that is impacted by a number of factors, including food intake, postnatal age, and birth weight. Clinical indicators are experienced by some babies with low blood glucose levels, however they are not very precise.

17(56.67%) presented at <24 hour of life, 7(23.3%) presented at 24-48hours of life, 5(16.6%) presented at 48 -72 hours of life and 1(3.33%) presented at 72 hours of life.

There were 19(63.33%) male and 11(36.67%) female. There was male predominance out of which 7(58.3) males had abnormal MRI findings. In a study

by Gu et al, 72.22% of the cases with abnormal MRI findings and 53.33% with normal MRI findings were males.⁶ In a study by Wong et al, about 54% of the cases with no hypoglycaemia and 67% with hypoglycaemia were males.³⁹ In a study by Nivins et al, 50.0% of the cases with no hypoglycaemia and 47.0% with any evidence of hypoglycaemia were males.³⁸

In this study, 14(46.67%) cases were delivered via LSCS and 16(53.33%) cases were delivered via Normal vaginal delivery.

7(23.33%) cases belonged to <34 weeks of gestational age, 15(50%) cases belonged to 34-37 weeks of gestational age group and 8(26.67%) belonged to >37 weeks of gestational age group. In a study by Gu et al, the mean gestational age was 38.44 weeks in cases with abnormal MRI findings and 38.6 weeks in cases with normal MRI findings.⁶ In a study by Wong et al, the mean gestational age was 39.26 weeks in cases with no hypoglycaemia and

39.12 weeks in cases with hypoglycaemia.³⁹ In a study by Nivins et al, the mean gestational age was 38.4 weeks in cases without hypoglycaemia and 38.3 weeks in cases with hypoglycaemia.³⁸

Of study population 2(6.67%) cases belonged to <1.5kg Birth weight category, 19(63.33%) cases belonged to 1.5-2.5 kg Birth weight category and 9(30%) belonged to >2.5 kg birth weight category. In a study by Gu et al, the birth weight was 3001 gms in cases with abnormal MRI and 3341.33 gms in cases with normal MRI.⁶ In a study by Wong et al, the birth weight was 33.89 grams in cases with no hypoglycaemia and 3194 gms in cases with hypoglycaemia.³⁹ In a study by Nivins et al, the mean birth weight in cases without hypoglycaemia was 3230 gms and 3206 gms in cases with hypoglycaemia.³⁸

Of study population 5(16.67%) cases had lower blood glucose levels <20mg/dl. Out of these 2 patients belonged to gestational <34 weeks, and 2 cases had sepsis as cause of hypoglycemia, in 1 of those 5 cases sepsis with meningitis was cause of hypoglycaemia .

24(80%) cases has lower blood glucose levels between 20-40 mg/dl . 9(37.5%) cases of these were found to have abnormal MRI findings. Lowest blood glucose level observed was 11 mg/dl which was associated with abnormal MRI Brain change (Focal

hemorrhagic lesions over parietal and occipital regions).

1(3.33%) had blood glucose levels >40mg/dl. In a study by Gu et al, the lowest serum glucose level was 27mg/dl in cases with abnormal MRI and 30mg/dl in cases with normal MRI.⁶ In a study by Nivins et al, mean blood glucose was 3.8 in cases with no hypoglycaemia and 3.3 in cases with hypoglycaemia.³⁸

As per study Age at discharge/death was <7 days in 2(6.67%) cases, 7-10 days in 27(90%) cases and > 10 days in 1(3.33%) cases.

Sepsis was present in 10(83.3%) cases of abnormal MRI findings and 3(16.7%) of the cases of normal MRI findings. This difference was statistically significant.

All of the cases with abnormal MRI findings had symptomatic hypoglycemia- Of which most common symptom was seizures (6), 3 had Symptomatic - Apnea and 2 had symptomatic Cyanosis and 1 had lethargy. About 77.8% were asymptomatic.

In 8 (66.7%) patients, sepsis or meningitis was the primary cause of Hypoglycemia, while 8(44.4%) of those with normal MRI results also had prematurity. Between the two groups, there was a statistically significant difference.

10(55.6%) cases with normal MRI findings received feeding only, while 10(83.3%) of the ones with unfavourable MRI findings received GIR 10-12 . There was a statistically significant difference. 1 patient was found to have prolonged hypoglycemia who received GIR above 12 and steroids, associated with abnormal MRI finding Periventricular Leukomalacia.

10(83.3%) of the cases with abnormal MRI findings received GIR for 48-72 hrs, 1(8.3%) case received GIR >72 hrs and 8.3% of the cases with abnormal MRI findings received GIR for 24- 48 hrs. GIR was not given in 55.6% of cases with Normal MRI findings . 38.9 % of cases with normal MRI findings and 5.6% of cases with normal MRI findings were given GIR for 24-48 hrs and <24 hrs respectively.

As per study Age at discharge/death was <7 days in 2(6.67%) cases, 7-10 days in 27(90%) cases and > 10 days in 1(3.33%) cases.

In 9(75.0%) of cases with aberrant MRI results and 1(5.6%) of those with normal MRI findings, the CSF was indicative of meningitis. There was a statistically significant difference.

In cases with low blood glucose, the MRI results suggested aberrant hyperintensity in many brain regions. Most consistently involved area was found to be parieto occipital regions .

18(60%) cases had normal MRI findings. Out of abnormal MRI findings, 50% of the abnormal findings were found to be involving Parietooccipital regions . 2(16.6%) cases had severe changes involving Basal Ganglia and Internal Capsule . Hippocampus and Globi Pallidi were involved in 8.3% cases each . 2 cases (16.67%) were found to have leukomalacia changes. In a study by Gu et al, most of the infants had some changes in white matter, 6.06% had severe WM injury had focal haemorrhage. All injuries were present in occipital and parietal lobe. About 33.3% of the cases had moderate abnormality in high signal and 14 cases had severe changes in the posterior WM. About 21.21% of the cases had abnormal signal intensity associated with cortical infarction and haemorrhage.⁶ In a study by Wong et al, the clinical evidence of no injury was present in 47% of the cases, watershed injury was present in 9%, Basal ganglia in 14% of the cases.³⁹

Conclusions

This study was undertaken to study the MRI changes in cases with neonatal hypoglycaemia. This study had shown that, MRI results suggested aberrant hyperintensity in regions of brain. The lesions ranged from hyperintensity, signal intensity and hemorrhages across the parietal and occipital regions.

Research gap:

A major research gap is the lack of clear understanding regarding the specific brain regions most vulnerable to hypoglycemia-induced damage, particularly when considering the variability in injury patterns across different individuals, and how the severity of hypoglycemia correlates with the extent and location of MRI abnormalities, especially in milder cases where clinical symptoms may be absent; further research is needed to better define the

sensitivity and specificity of MRI in detecting subtle brain injuries in neonates with hypoglycemia.

Key points about this research gap:

Inconsistency in findings:

While studies often point to the occipital lobe as a primary site of injury in neonatal hypoglycemia, there is variability in reported patterns of brain damage across different studies, with some showing involvement of other regions like the parietal lobe or deep brain structures.

Correlation with clinical severity:

A significant gap exists in determining the precise relationship between the severity of hypoglycemia and the extent of MRI abnormalities, making it challenging to predict long-term neurological outcomes based on imaging findings alone.

Subtle changes and early detection:

Identifying subtle MRI changes in early stages of hypoglycemia, especially in asymptomatic infants, is crucial for early intervention but requires further research on sensitive imaging techniques and standardized interpretation criteria.

Limitations

This study was not without limitations. The sample size was small to generalize the findings.

A prospective study design with adequate sample size and standardized MRI protocols can bring out more facts about the hypoglycaemic MRI changes in neonates.

Ethical Clearance: Ethical clearance was taken before study.

IEC number- SHKM GMC IEC, Date-22/8/2022, Reference no-SHKM/IEC/2022/63

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Conflict of Interest: NIL

References

1. Cornblath M. Neonatal Hypoglycemia 30 years later: does it injure the brain? Historical summary and present challenges. *Acta Paediatr Jpn.* 1997;39:S7-S11.

2. Sinclair JC. Approaches to the definition of neonatal Hypoglycaemia. *Acta Paediatr Jpn.* 1997;39:S17-S20.
3. Anderson Enni JB, Narasimhan SR, Huang A, Jegatheesan P. Screening and diagnosis of neonatal hypoglycaemia in at-risk late preterm and term infants following AAP recommendations: a single centre retrospective study [published correction appears in *BMJ Paediatr Open.* 2023 Jul;7(1):]. *BMJ Paediatr Open.* 2023;7(1):e001766.
4. Tam EW, Haeusslein LA, Bonifacio SL, et al. Hypoglycaemia is associated with increased risk for brain injury and adverse neurodevelopmental outcome in neonates at risk for encephalopathy. *J Pediatr.* 2012;161:88-93.
5. Kallem VR, Pandita A, Gupta G. Hypoglycaemia: When to Treat?. *Clin Med Insights Pediatr.* 2017;11:1179556517748913.
6. Gu MH, Amanda F, Yuan TM. Brain Injury in Neonatal Hypoglycaemia: A Hospital- Based Cohort Study. *Clin Med Insights Pediatr.* 2019; 13: 1179556519867953.
7. Jun Su, Li Wang. Research advances in Neonatal hypoglycemic brain injury, *Transl Pediatr.* 2012 Oct; 1(2): 108-115.
8. Burns CM, Rutherford MA, Boardman JP, Cowan FM. Patterns of cerebral injury and neurodevelopmental outcomes after symptomatic neonatal Hypoglycaemia. *Pediatrics.* 2008;122:65-74.
9. Cornblath M. Neonatal Hypoglycaemia 30 years later: does it injure the brain? Historical summary and present challenges. *Acta Paediatr Jpn* 1997;39:S7-11.
10. Cornblath M, Odell GB, Levin EY. Symptomatic neonatal Hypoglycaemia associated with toxemia of pregnancy. *J Pediatr* 1959;55:545-62.
11. Lou LF, Zhang J. Current Status of Diagnosis and Treatment in Hypoglycemic Encephalopathy. *Yi Xue Zong Shu* 2010;16:2008-10.
12. Hampl SE, Hassink SG, Skinner AC, et al. Clinical Practice Guideline for the Evaluation and Treatment of Children and Adolescents With Obesity [published correction appears in *Pediatrics.* 2024 Jan 1;153(1):]. *Pediatrics.* 2023;151(2):e2022060640.
13. Boluyt N, van Kempen A, Offringa M. Neurodevelopment after neonatal Hypoglycaemia: a systematic review and design of an optimal future study. *Pediatrics* 2006;117:2231-43.
14. Jain A, Aggarwal R, Jeeva Sankar M, et al. Hypoglycaemia in the newborn. *Indian J Pediatr* 2010 ;77:1137-42.
15. Liu ZW, Chen HJ. Guideline for Management of Neonatal Hypoglycaemia in United States. *Shi Yong Er Ke Lin Chuang Za Zhi* 2010;25:618-20.
16. Vannucci RC, Vannucci SJ. Hypoglycemic brain injury. *Semin Neonatol.* 2001;6:147- 55.
17. Giouleka S, Gkiouleka M, Tsakiridis I, et al. Diagnosis and Management of Neonatal Hypoglycaemia: A Comprehensive Review of Guidelines. *Children (Basel).* 2023;10(7):1220.
18. Castillo-Castrejon M, Powell TL. Placental Nutrient Transport in Gestational Diabetic Pregnancies [published correction appears in *Front Endocrinol (Lausanne).* 2019 Jan 28;10:5]. *Front Endocrinol (Lausanne).* 2017;8:306.
19. Lerner J. Insulin and the stimulation of glycogen synthesis. The road from glycogen structure to glycogen synthase to cyclic AMP-dependent protein kinase to insulin mediators. *Adv Enzymol Relat Areas Mol Biol.* 1990;63:173-231.
20. Ron I, Lerner RK, Rathaus M, et al. The adipokine FABP4 is a key regulator of neonatal glucose homeostasis. *JCI Insight.* 2021;6(20):e138288. Published 2021 Oct 22.
21. Edridge CL, Dunkley AJ, Bodicoat DH, et al. Prevalence and Incidence of Hypoglycaemia in 532,542 People with Type 2 Diabetes on Oral Therapies and Insulin: A Systematic Review and Meta-Analysis of Population Based Studies. *PLoS One.* 2015;10(6):e0126427.
22. Anderson Enni JB, Narasimhan SR, Huang A, Jegatheesan P. Screening and diagnosis of neonatal hypoglycaemia in at-risk late preterm and term infants following AAP recommendations: a single centre retrospective study [published correction appears in *BMJ Paediatr Open.* 2023 Jul;7(1):]. *BMJ Paediatr Open.* 2023;7(1):e001766.
23. Rajendran R, Rayman G. Point-of-care blood glucose testing for diabetes care in hospitalized patients: an evidence-based review. *J Diabetes Sci Technol.* 2014;8(6):1081-1090.
24. Wu J, Liu Y, Yin H, Guo M. A new generation of sensors for non-invasive blood glucose monitoring. *Am J Transl Res.* 2023;15(6):3825-3837.
25. Rao PN, Shashidhar A, Ashok C. In utero fuel homeostasis: Lessons for a clinician. *Indian J Endocrinol Metab.* 2013;17(1):60-68.
26. Plows JF, Stanley JL, Baker PN, Reynolds CM, Vickers MH. The Pathophysiology of Gestational Diabetes Mellitus. *Int J Mol Sci.* 2018;19(11):3342.

27. Hay WW Jr. Placental-fetal glucose exchange and fetal glucose metabolism. *Trans Am Clin Climatol Assoc.* 2006;117:321-340.
28. Goel P, Choudhury SR. Persistent hyperinsulinemic Hypoglycemia of infancy: An overview of current concepts. *J Indian Assoc Pediatr Surg.* 2012;17(3):99-103.
29. Weinstein DA, Steuerwald U, De Souza CFM, Derks TGJ. Inborn Errors of Metabolism with Hypoglycemia: Glycogen Storage Diseases and Inherited Disorders of Gluconeogenesis. *Pediatr Clin North Am.* 2018;65(2):247-265.
30. Giouleka S, Gkiouleka M, Tsakiridis I, et al. Diagnosis and Management of Neonatal Hypoglycemia: A Comprehensive Review of Guidelines. *Children (Basel).* 2023;10(7):1220.
31. Aliefendioğlu D, Çoban A, Hatipoğlu N, et al. Management of Hypoglycemia in newborn: Turkish Neonatal and Pediatric Endocrinology and Diabetes Societies consensus report. *Turk Pediatri Ars.* 2018;53(Suppl 1):S224-S233.
32. Adamkin DH. Neonatal Hypoglycemia. *Semin Fetal Neonatal Med.* 2017 Feb;22(1):36-41.
33. Rehni AK, Dave KR. Impact of Hypoglycemia on Brain Metabolism During Diabetes. *Mol Neurobiol.* 2018;55(12):9075-9088.
34. Mergenthaler P, Lindauer U, Dienel GA, Meisel A. Sugar for the brain: the role of glucose in physiological and pathological brain function. *Trends Neurosci.* 2013;36(10):587-597.
35. Newcomer JW, Farber NB, Olney JW. NMDA receptor function, memory, and brain aging. *Dialogues Clin Neurosci.* 2000;2(3):219-232.
36. Woodward LJ, Clark CA, Bora S, Inder TE. Neonatal white matter abnormalities an important predictor of neurocognitive outcome for very preterm children. *PLoS One.* 2012;7(12):e51879.
37. Caro-Domínguez P, Lecacheux C, Hernandez-Herrera C, Llorens-Salvador R. Cranial ultrasound for beginners. *Transl Pediatr.* 2021;10(4):1117-1137.
38. Shenton ME, Hamoda HM, Schneiderman JS, et al. A review of magnetic resonance imaging and diffusion tensor imaging findings in mild traumatic brain injury. *Brain Imaging Behav.* 2012;6(2):137-192.
39. Nivins S, Kennedy E, Thompson B, et al. Associations between neonatal hypoglycaemia and brain volumes, cortical thickness and white matter microstructure in mid-childhood: An MRI study. *Neuroimage Clin.* 2022;33:102943.
40. Wong DS, Poskitt KJ, Chau V, et al. Brain injury patterns in Hypoglycemia in neonatal encephalopathy. *AJNR Am J Neuroradiol.* 2013;34(7):1456-1461.
41. Qiao LX, Wang J, Yan JH, et al. Follow-up study of neurodevelopment in 2-year-old infants who had suffered from neonatal Hypoglycemia. *BMC Pediatr.* 2019;19(1):133.
42. Burns CM, Rutherford MA, Boardman JP, Cowan FM. Patterns of cerebral injury and neurodevelopmental outcomes after symptomatic neonatal Hypoglycemia. *Pediatrics.* 2008;122 (1):65-74.
43. Janani Shishu Suraksha Karyakram(JSSK) :: National health mission <https://nhm.gov.in/index4.php?lang=1&level=0&lid=171&linkid=150>