e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(1); 510-516

Original Research Article

Incidence of Hyperbilirubinemia in Asphyxiated Newborn in a Tertiary Care Hospital

Deepak Kumar Behera[1], Simanta Das[2], Pragnyojit Nayak[3], Sumanta Panigrahi[4]

[1]Assistant Professor, Department of Paediatrics, S.C.B. Medical College and Hospital and SVPPGIP, Cuttack

[2]Associate Professor, Department of Paediatrics, S.C.B Medical College and Hospital and SVPPGIP, Cuttack

[3]Resident, Department, S.C.B Medical College and Hospital and SVPPGIP, Cuttack

[4]Professor, Department of Paediatrics, S.C.B Medical College and Hospital and SVPPGIP, Cuttack

Received: 01-10-2025 / Revised: 15-11-2025 / Accepted: 21-12-2025

Corresponding author: Dr. Deepak Kumar Behera

Conflict of interest: Nil

Abstract

Introduction: Our study is a single center study. Multicenter studies with larger number of cases of different geographic locations would provide additional insight regarding this study. More no of case-control studies are required with large sample size to determine the severity of serum bilirubin level with birth asphyxia particularly in term neonates. Additional newborn screening to be done in cases of prolonged high serum bilirubin level like hypothyroidism, G-6-PD deficiency, hemolytic anemia, Galactosemia so that it could have been intervened early, like hypothyroidism is a treatable cause which should be treated early to prevent developmental delay and hearing loss, similarly G-6- PD screening should be done to prevent hematuria and hemoglobinuria caused by drug.

Material And Method: This prospective study was conducted in newborn unit of a tertiary care teaching hospital in Odisha, from August 2022 - July 2024 (2 years). All the neonates satisfying the inclusion criteria admitted to SCBMCH and SVPPGIP newborn unit were included in the study. Informed consent was taken from the parents/ guardians of all the enrolled patients for obtaining relevant history, clinical examination and performing necessary investigations. Antenatal history of mother will be collected in detail to study the relevant risk factors. The cases were then diagnosed clinically using previously established criteria for Hypoxia ischemic encephalopathy (HIE), General examination and systematic examination were done in details to establish clinical diagnosis.

Result: "Incidence Of Hyperbilirubinemia In Asphyxiated Newborn In A Tertiary Care Hospital" which was an institution based prospective observational study undertaken from August, 2022 to July, 2024 in SVPPGIP and SCBMCH, Cuttack that included 80 newborns with birth asphyxia and 50 non asphyxiated neonates, satisfying the inclusion criteria.

Conclusion: Birth asphyxia continues to be one of the major causes of perinatal mortality in developing nation where obstetric care and neonatal resuscitation is still in their struggling phase. Birth asphyxia is a substantial cause of unconjugated hyperbilirubinemia in newborns. Assay of total serum bilirubin and indirect serum bilirubin can be used as an easy early diagnostic marker to differentiate between babies with birth asphyxia and those without asphyxia.

DOI: 10.25258/ijcpr.18.1.84

Introduction

The WHO defines birth asphyxia as the inability to start and maintain breathing at birth. Even in cases where the damage does not result in a foetal outcome, it can also be described as a brief disruption of oxygen supply that suggests a dangerous metabolic challenge. One of the top three causes of infant fatalities worldwide is perinatal asphyxia (23%).

Infants with foetal acidosis (PH<7) are evaluated for asphyxia at five minutes. APGAR SCORE<7, multiple organ symptoms and signs, and hypoxic-ischemic encephalopathy (altered tone, decreased consciousness, seizures). The WHO classification of diseases (ICD 10) defines mild and moderate birth asphyxia as APGAR SCORE 4-7 at 1 minute, whereas severe birth asphyxia is defined as

APGAR SCORE 0-3 at 1minute.

Within minutes of fetal hypoxia there occurs bradycardia, hypotension, decreased cardiac output and severe metabolic acidosis. Almost all organs can be affected, but the brain, myocardium, kidney and bowels appear to be most sensitive to severe damage. Hypoxia impairs cerebral oxidative metabolism leading to increase in lactate, fall in pH, inefficiency of anaerobic glycolysis to generate ATP. The hypoxic brain therefore increases its glucose utilization. Vascular dilation caused by hypoxia favors increased glucose availability which leads to increased local lactic acid production, which worsens the acidosis. Local glucose stores depleted causing increased accumulation of lactic acid. During prolonged hypoxia cardiac output falls, cerebral blood flow compromise and there occurs loss of vascular auto regulation which leads to hypoxic ischemic insults. The hypoxic ischemic results in initiation of a series of protective reflexes, called diving sea reflexes, which prevent damage to more vital organs brain, heart and adrenals) at the expenses of lesser vital organs (kidney, lungs, gastrointestinal tract, liver, and spleen) in an attempt to redistribute available blood flow.

Neonatal hyperbilirubinemia is primarily caused by the accumulation of bilirubin, a byproduct of heme catabolism, in the blood and tissues of newborns. While physiological jaundice is common in the first week of life and typically resolves without intervention, pathological hyperbilirubinemia can lead to severe complications if left untreated. Asphyxia at birth, resulting from factors such as placental insufficiency, umbilical cord accidents, or prolonged labor, is a known risk factor for adverse neonatal outcomes, including hypoxic-ischemic encephalopathy and multi-organ dysfunction.

The pathophysiology of hyperbilirubinemia in asphyxiated newborns is multifactorial and may involve hemolysis, impaired hepatic conjugation of bilirubin, and decreased clearance of bilirubin due to hepatic hypoperfusion. The combination of these mechanisms can result in a rapid increase in serum bilirubin levels, predisposing the infant to bilirubin-induced neurotoxicity and potentially leading to long-term developmental delays and neurologic sequelae. In tertiary care hospitals, where complex cases are often referred, the management of neonates with hyperbilirubinemia and a history of birth asphyxia poses unique challenges. The incidence of hyperbilirubinemia in these high-risk newborns is not well-documented in the literature, and there is a need to understand the incidence, risk factors, and outcomes associated with this condition within the context of tertiary care facilities.

Study aims to investigate the incidence of hyperbilirubinemia in neonates with a history of birth asphyxia admitted to a tertiary care hospital. By elucidating the burden of hyperbilirubinemia in this specific population, the study seeks to contribute to the existing knowledge on neonatal care and inform clinical practice guidelines for the management of asphyxiated newborns with unconjugated hyperbilirubinemia.

Material and Method

Study Place: Department of Paediatrics, SCB Medical College and Hospital and SVPPGIP Cuttack, Odisha

Study Period: August 2022 to July 2024

Study Design: A Hospital based Prospective Observational study

Target Population: Asphyxiated term & preterm Neonates (Day3and Day 5 of life)

Sample Size: 80 (40 Term neonates & 40 preterm neonates), & 50 non-asphyxiated neonates

Source of Data: All relevant data was collected from Newborn wards, SNCU, NICU, ACRC(NB) of SVPPGIP & SNCU SCBMCH, Cuttack, after requesting consent from head of the department and patient's guardian.

Inclusion Criteria: Full term neonates (Gestational age 37 to 42 weeks) and Preterm neonates (Gestational age <37 weeks) with any one of the following:

- Asphyxiated newborn with APGAR <7 at 5min of birth
- Neonatal Neurological sequelae as determined by Sarnat and Sarnat of HIE
- Resuscitation with more than 1 min of PPV before spontaneous respiration

Exclusion Criteria

- Neonatal with congenital anomalies life heart disease
- Neonatal cephalhematoma
- congenital infection
- Neonates born to mothers with viral hepatitis

Ethical Clearance: The study was approved by the Institutional Ethics Committee of S.C.B. medical college and hospital, Cuttack.

Informed Consent: All newborns fulfilling inclusion criteria were enrolled in the study after obtaining informed consent of respective parents.

Methodology: This prospective study was conducted in newborn unit of a tertiary care teaching hospital in Odisha, from August 2022 - July 2024 (2 years). All the neonates satisfying the inclusion criteria admitted to SCBMCH and SVPPGIP newborn unit were included in the study. Informed consent was taken from the parents/

guardians of all the enrolled patients for obtaining relevant history, clinical examination and performing necessary investigations.

Antenatal history of mother will be collected in detail to study the relevant risk factors. The cases were then diagnosed clinically using previously established criteria for Hypoxia ischemic encephalopathy (HIE), General examination and systematic examination were done in details to establish clinical diagnosis. Data collected was sent for statistical analysis.

Results

Table1: Table Showing Mean Weight (In Kg) Of Two Group

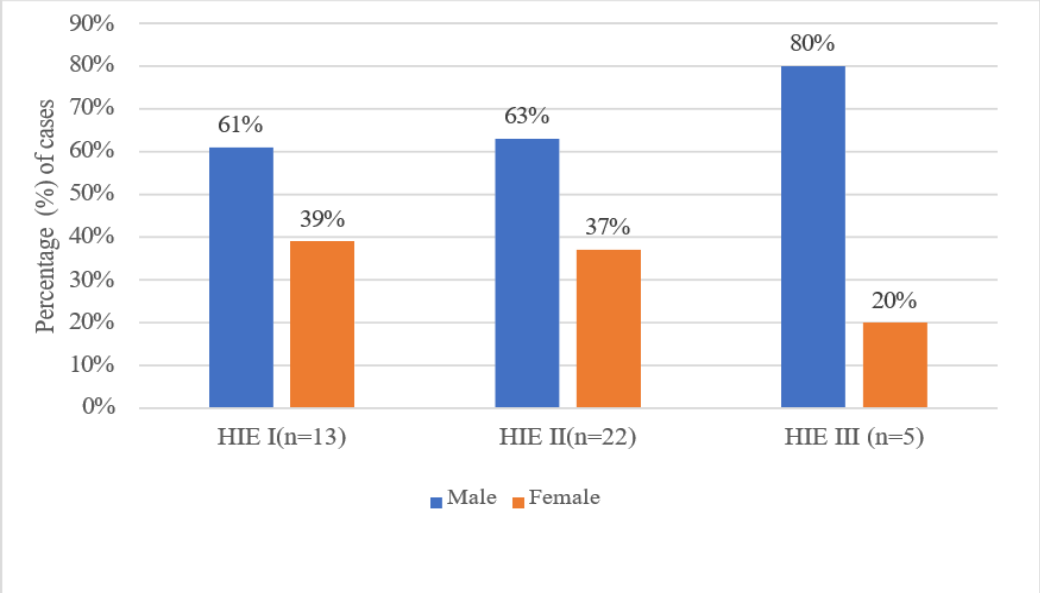
	Mean	Standard Deviation
Term	2.79	0.483
Preterm	1.93	0.600

Table 2: Mean Gestational Age of Two Group

	Mean	Standard Deviation
Term	38.825	1.106
Preterm	34.35	1.97

Table 3: Distribution of Subjects According To the Sex of Child (Term)

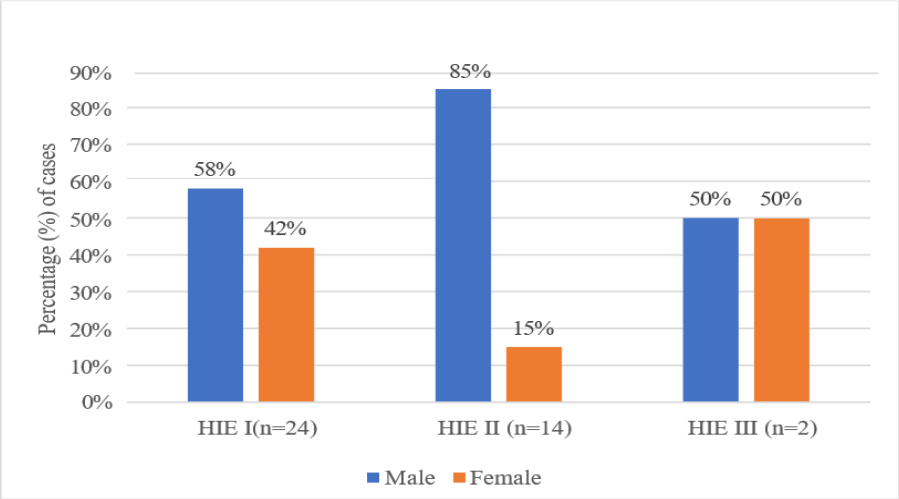
Sex	HIE I(n=13)	HIE II(n=22)	HIE III (n=5)	Total (n=40)
Male	8(61%)	14(63%)	4(80%)	26(65%)
Female	5(39%)	8(37%)	1(20%)	14(35%)



Graph 1: Showing Percentage of Cases with Sex (Term)

Table 4: Distribution of Subjects According To the Sex of Child (Preterm)

Sex	HIE I(n=24)	HIE II (n=14)	HIE III (n=2)	Total (n=40)
Male	14(58%)	12(85%)	1(50%)	27(67.5%)
Female	10(42%)	2(15%)	1(50%)	13(32.5%)



Graph 2: Showing Percentage of Cases with Sex (Preterm)

Table 5: Mean TPC of Two Groups

	Mean	Std. Deviation
Term	187600.00	88276.605
Preterm	114512.50	61549.801

Table 6: Mean CRP (Q) Of Two Groups

	Mean	Std. Deviation	CRP (Q)>6
Term	4.728250	2.9172905	15
Preterm	5.726000	1.9999203	20

Table 7: Distribution of Subjects According To Seizures

Seizures	HIE I (n=16)	HIE II (n=20)	HIE III (n=4)
Absent	12(87.5%)	0	0
Present	2(12.5%)	17(85%)	2(50%)
Prolonged	0	3(15%)	2(50%)

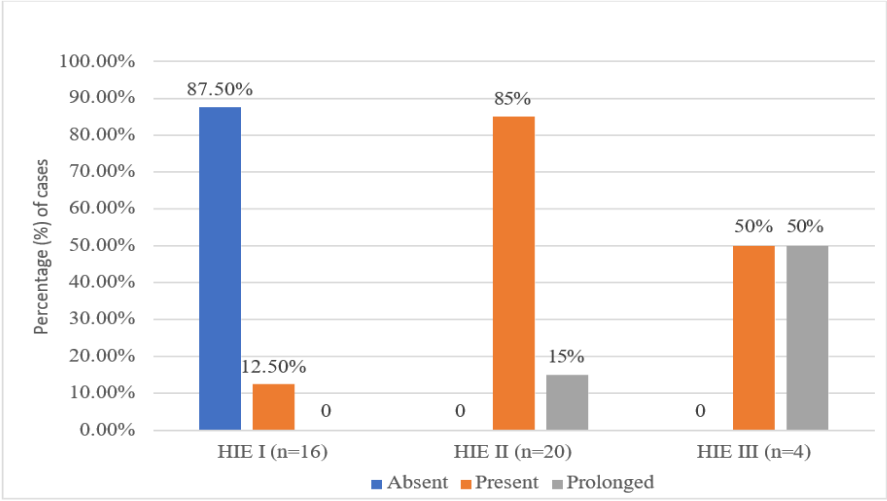


Figure 3: Graph Showing Distribution of Subjects According To Associated Symptoms

Table 8: Distribution of Subjects According To Different Type Associated Symptoms

Associated Symptoms	HIE I (n=16)	HIE II (n=20)	HIE III (n=4)
Hypothermia	2(12.5%)	5(25%)	3(75%)
Respiratory Distress	1(6.25%)	6(30%)	3(75%)
Vomiting	3(18.75%)	3(15%)	1(25%)
Jitteriness	6(37.5%)	3(15%)	0

Discussion

We evaluated the occurrence of incidence of hyperbilirubinemia following perinatal asphyxia in term and preterm neonates admitted to our newborn wards of SVPPGIP & SCBMCH Cuttack.

By applying rigid inclusion and exclusion criteria 80 newborns (40 Term and 40 Preterm) were studied in the study period. We studied the course of these newborns and classified the severity of birth asphyxia using LEVENE classification and taken serum bilirubin level on D3 & D5 along with their CBC & CRP(Q) in both groups. Obtained data have been displayed in tables with necessary statistical analysis. Table 1 displays mean birth weight of term neonates 2.79±0.48 Kg while mean birth weight in preterm was 1.93±0.60 Kg.

- Seema rai, MD Tarik Islam et al found out the mean birth weight to be 2.47±0.3 Kg, 2.78±0.2 kg respectively, which is similar to our study [1][2]
- Vibha kariya et al (2019) found out that mean birth weight to be 2.6±0.35 Kg [3]
- Chaudhary Mukesh et al (2014) in their study found out mean birth weight to be 3.02±0.36 kg. [4]

Table 2 displays mean Gestational age of study participants in term it was 38±1.106 weeks in preterm it was 34±1.97 weeks

- Seema rai et al (2022) found out mean gestational age to be 35.9±2.5 weeks.[1]

MD Tarik Islam, Chaudhary Mukesh et al found out the mean gestational age of study population is 38.99 weeks ,38.04±0.8 weeks which was similar to our study.[2][4]

Table 3 & 4 showing variation of asphyxia with sex in term and preterm neonates. perinatal asphyxia is more predominant with 61% HIE- I,63% HIE-II, 80% HIE-III IN Term & 58% HIE-I,85% HIE-II, 50% HIE-III with sex Ratio 1.85:1 in term and 2.07:1 in preterm.

Seema rai et al (2022) study shows the sex ratio of 1.63:1 with male predominance which was similar to our study.[1]

Nanda Chhavi et al (2014) found sex ratio 1.5:1 with male predominance in their study.[5] Vibha kariya et al (2019) found the sex ratio to be 1.08:1 with male predominance in their study.[3] Table 5&6 showing the degree of asphyxia with mode of delivery, which was more marked among the babies born by spontaneous vaginal delivery with 66% HIE -I, 55% HIE-II,100% HIE-III in preterm & 60% HIE- I,50% HIE-II,60% HIE-III in term which was more as compared to LSCS / forceps delivery. Asad Nauman kiyani et al (2014) observed birth asphyxia was more noted in spontaneous delivery (44.39%) more than c-section (32.14%) and instrumental delivery (23.47) which is similar to our study.[6]

Vibha Kariya et al (2019) found that birth was more common in vaginal delivery (66.7%), more than c-section (29.33%), instrument delivery (4%).[3]

- Siva saranappa S B, C.C. Nair et al (2015) in their study found that birth asphyxia was more noted in vaginal delivery (70%), more than Instrumental delivery (25%) and LSCS 5%.[17]

Table 7&8 displaying the study population according to the HIE stages in term neonates which was 32.5% HIE-I, 55% HIE-II,12.5% HIE-III whereas preterm consist of 60% HIEI, 35%HIEII, 5%HIE III.

- Seema Rai et al (2022) found that the incidence of HIE I 41%, HIE -II 44%, HIE -III 12%.[1]
- H. Namusoke et al (2018) studied and observed that the incidence of HIE-I (43%), HIE-II (34.8%), HIE-III (21.7) which was similar to our study.[8]
- Chaitali Patra et al in their study found that incidence of HIE-I (48%), HIE-II (32%), HIE-III (9%).[7]
- Chaudhary Mukesh et al (2014) found out 30% HIE-I, 47.1% HIE- II,22.8% HIE-III in their study.
- Md Tarik Islam et al (2010) et al found that out of 70 asphyxiated patients 27(38%),30(43%),13(19%) had HIE I, HIE II, HIE III respectively.[2]

Conclusion

Birth asphyxia continues to be one of the major causes of perinatal mortality in developing nation where obstetric care and neonatal resuscitation is still in their struggling phase. Birth asphyxia is a substantial cause of unconjugated hyperbilirubinemia in newborns. Assay of total serum bilirubin and indirect serum bilirubin can be used as an easy early diagnostic marker to differentiate between babies with birth asphyxia and those without asphyxia. Early rise total serum bilirubin and indirect serum bilirubin within 72 hrs. of life can function as supporting evidence of birth asphyxia especially when a newborn presents with features of HIE without a proper history of birth asphyxia or whose birth details are not well recorded. Early rise of total serum bilirubin and indirect serum bilirubin within 72 hrs. Can be used for the detection of the severity of birth asphyxia and thus can be used as a guiding tool for early intervention to prevent kernicterus and can predict mortality. Also, serve as a prognostic tool and aid in counselling of the parents.

Reference

1. Rai S, Sood M, Kaur A. Is perinatal asphyxia associated with an increase in serum bilirubin in neonates? A case-control study. J Family Med Prim Care. 2022 Jul;11(7):3840-3843. doi: 10.4103/jfmpc.jfmpc_1018_21. Epub 2022 Jul 22. PMID: 36387686; PMCID: PMC9648229.
2. Islam, M. T., Hoque, S. A., Matin, M., Islam, M. N., Hossain, M. A., Nazir, F., & Islam, S. (2010). Alteration of Hepatic Function: Helpful to Diagnose and Assess Severity of Perinatal Asphyxia. Bangladesh Journal of Child Health, 34(1).
3. Kariya, Vibha; Jain, Manish; Jategaonkar, Smita. Study of Hepatic Enzymes in Term Neonates with Perinatal Asphyxia. Journal of Clinical Neonatology 9(2): p 125-131, Apr–Jun 2020. | DOI: 10.4103/jcn.JCN_116_19.
4. Choudhary M, Sharma D, Dabi D, Lamba M, Pandita A, Shastri S. Hepatic Dysfunction in Asphyxiated Neonates: Prospective Case-Controlled Study. Clinical Medicine Insights: Paediatrics. 2015;9. doi:10.4137/CMPed.S21426.
5. Chhavi N, Zutshi K, Singh NK, Awasthi A, Goel A. Serum liver enzyme pattern in birth asphyxia associated liver injury. Paediatric Gastroenterol Hepatol Nutr. 2014 Sep;17(3):162-9. doi: 10.5223/pghn.2014.17.3.162. Epub 2014 Sep 30. PMID: 25349832; PMCID: PMC4209321.
6. Nauman Kiyani A, Khushdil A, Ehsan A. Perinatal Factors Leading to Birth Asphyxia among Term Newborns in a Tertiary Care Hospital. Iran J Paediatric. 2014 Oct;24(5):637-42. Epub 2014 Sep 12. PMID: 25793074; PMCID: PMC4359420.
7. Patra, Chaitali; Sarkar, Shatanik1, Dasgupta, Malay K.1. Study of hepatic enzyme activity as a predictor of perinatal asphyxia and its severity and outcome. Indian Journal of Health Sciences and Biomedical Research (KLEU) 9(3): p 297-302, Sep–Dec 2016. DOI: 10.4103/2349-5006.196324.
8. Namusoke, H., Nannyonga, M.M., Ssebunya, R. et al. Incidence and short-term outcomes of neonates with hypoxic ischemic encephalopathy in a Peri Urban teaching hospital, Uganda: a prospective cohort study. Maternal health, neonatal and perinatal **4**, 6 (2018).
9. Bizuneh AD, Alemnew B, Getie A, Wondmieneh A, Gedefaw G. Determinants of neonatal jaundice among neonates admitted to five referral hospitals in Amhara region, Northern Ethiopia: an unmatched case-control study. BMJ Paediatrics Open. 2020; 4(1): e000830. DOI: 10.1136/bmjpo-2020-000830. PMID: 33024837; PMCID: PMC7511639.
10. Fekete M, Horváth M, Vincellér M. Perinatal asphyxia and jaundice in newborn infants. Acta Paediatrica Academiae Scientiarum Hungaricae. 1978; 19(1):17-26. PMID: 665220.
11. Mamatha, R.; Meena, Jeetam Singh; Sulaniya, Pawan Kumar; Sehra, Ram Narain; Verma, Suniti. Comparative Study to Correlate Haematological Parameters with the Severity of Birth Asphyxia. CHRISMED Journal of Health and Research.
12. Study of Nucleated Red Blood Cell Counts in Asphyxiated Newborns and the Fetal Outcome Sylvia M Colacoa, c, Minhajuddin Ahmeda , Vinayak Y Kshirsagara , Rohit Bajpai
13. Study of haematological parameters among newborns with perinatal asphyxia Koreti S, Eske G.S, Gaur A. , Gupta A.4 1Dr. Sunita Koreti, Professor, 2Dr. Gunvant Singh Eske, Assistant Professor, 3Dr. Ajay Gaur, Professor and Head, 4Dr. Akriti Gupta, Post graduate student, all authors are affiliated with Department of Paediatrics, G.R. Medical College, Gwalior (MP), India.
14. Y, Mu L, Guo X, Li Y, Wang L, Yang W, Li S and Shen Q: Clinical significance of interleukin-6, tumor necrosis factor-α and high-sensitivity C-reactive protein in neonates with hypoxic-ischemic encephalopathy. Exp Ther Med 8: 1259-1262, 2014
15. Blood Biomarkers for Evaluation of Perinatal Encephalopathy Ernest M. Graham, Irina Burd, Allen D. Everett, Frances J. Northington
16. Ilah, Bilkisu Garba; Aminu, Muhammad Sakajiki1; Musa, Abdullahi2; Adelakun, Muyideen Bimbo; Adeniji, Akeem Oladiran; Kolawole, Taofik. Prevalence and Risk Factors for Perinatal Asphyxia as Seen at a Specialist Hospital in Gusau, Nigeria. Sub-Saharan African Journal of Medicine, Apr–Jun 2015; 2(2): 64-69.
17. Clinical profile and outcome of perinatal asphyxia in a tertiary care centre Siva Saranappa S B, Chaithanya C Nair, Madhu G N, Srinivasa S, Manjunath MN Department of Pediatrics, Kempegowda Institute of Medical Sciences and Research Hospital, Bangalore.
18. The temporal evolution of electrographic seizure burden in neonatal hypoxic ischemic encephalopathy Niamh E. Lynch, Nathan J. Stevenson, Vicki Livingstone, Brendan P. Murphy, Janet M. Rennie, Geraldine B. Boylan.
19. Organ Complications of Infants with Hypoxic Ischemic Encephalopathy Before Therapeutic Hypothermia Authors: Barbara Michniewicz, SalwanR. Al Saad, Lukasz M. Karbowski, Janusz Gadzinowski, Marta Szymankiewicz, and Dawid Szpecht.
20. Ristovska, Sanja. Respiratory Distress

Syndrome (RDS) in Newborns with Hypoxic-Ischemic Encephalopathy (HIE). PRILOZI, vol.45, no.1, 2024.
21. Neurological Aspects of Perinatal Asphyxia J. K. Brown, R. J. Purvis, J. O. Forfar, F. Cockburn 1974.
22. Mohan K, Mishra PC, Singh DK. Clinical profile of birth asphyxia in newborn. International Journal of Science & Technology.
23. Chapter 55, Cloherty and stark manual of neonatal care,
24. Report of National Perinatal Database (NNF, India)2003.
25. Northington FJ, Ferriero DM, Graham EM, Traystman RJ, Martin LJ.
26. Early neurodegeneration after hypoxia-ischemia in neonatal rat is necrosis while delayed neuronal death is apoptosis. Neurobiology of disease. 2001 Apr 1;8(2):207-19.