International Journal of Life Sciences, Biotechnology and Pharma Research Vol. 13, No. 12, December 2024 Online ISSN: 2250-3137
Print ISSN: 2977-0122

DOI: 10.69605/ijlbpr_13.12.2024.24

ORIGINAL RESEARCH

Birth Asphyxia in Newborns: A Study of Electrolyte Disturbance and their Association with Acute Kidney Injury

[1]Dr. Arshdeep Jagga, [2]Dr. Dinesh Mekle, [3]Dr. Richa Rathore, [4]Dr. Jagdamba Dixit

[1]PG Resident, [2]Professor, [3]Assistant Professor, [4]Professor and Head, Department of Pediatrics, People's College of Medical Science and Research Centre, Bhopal, MP, India

Corresponding Author
Dr. Richa Rathore
Assistant Professor, Department of Pediatrics, People's College of Medical Science and Research Centre, Bhopal, MP, India

Received Date: 26 November, 2024 Accepted Date: 5 December, 2024

Abstract
Background: Birth asphyxia is a common neonatal problem contributing to mortality and morbidity. Electrolyte disturbances and acute kidney injury (AKI) are common complications. **Objective:** To study electrolyte disturbances in newborns with birth asphyxia and its association with AKI. **Methods:** Newborns with birth asphyxia (>34 weeks gestational age) were included. Asphyxiated neonates were grouped by 1-minute Apgar score: mild (6-7), moderate (4-5), and severe (≤3). They received standard NICU care. Serum electrolytes, blood urea, and creatinine were monitored at 24 hours and 72-96 hours. Urine output was tracked using collection bags or catheterization. Electrolyte disturbances and AKI were classified using serum electrolyte levels and p-RIFLE criteria. **Results:** A total 56% male and 44% female participants, with 42% having low birth weight. Sodium and potassium disturbances were common, but not statistically significant in mild and moderate asphyxia cases. Acute kidney injury (AKI) occurred in 1.15% of cases, with a significant association found in mild asphyxia cases (P<0.01). Potassium levels were significantly associated with AKI (P=0.001), but sodium levels were not. **Conclusion:** Electrolyte disturbances are common in newborns with birth asphyxia, but not significantly associated with asphyxia severity. In this study AKI is a significant complication, observed asphyxiated in newborn. Early detection and management of electrolyte disturbances and AKI are crucial in improving outcomes.

Keywords: Perinatal asphyxia, electrolyte disturbances, acute kidney injury, newborn

Introduction

Perinatal asphyxia is a common neonatal problem and contributes significantly to neonatal mortality and long term morbidity. The incidence of perinatal asphyxia is 1 – 1.5 % in most developed countries and is inversely related to gestational age and birth weight.[1,4-6] It is a common problem with the incidence varying from 0.5–2% of live births. Some report the incidence from 1 to 8 per 1000 live births.[7] Globally, four to nine million neonates are diagnosed with birth asphyxia each year. In India, the incidence of perinatal asphyxia is 5% and constitutes for 24.3%

Metabolic derangements after asphyxia alters hemodynamics of newborn and a redistribution of cardiac output occur to maintain cerebral, cardiac, and adrenal perfusion while potentially compromising renal, gastrointestinal, and skin perfusion resulting in multiple organ dysfunctions.[8-10] In term neonates with asphyxia, renal, CNS, cardiac and lung dysfunction occur in 50%, 28%, 25% and 25% respectively.[8] Neonates with perinatal asphyxia are at risk of AKI as redistribution of cardiac output occurs during hypoxia to maintain cerebral, cardiac and adrenal perfusion, thereby reducing oxygen supply to the kidneys.[8] The renal parenchymal cells also have a limited capacity for anaerobic respiration and are highly susceptible to reperfusion injury.[11]

In the past, studies on AKI in neonates with perinatal asphyxia have reported variable incidence, ranging from 11.7 to 60% based on different diagnostic criteria used.[12-13] In 2013, a standardized definition of AKI termed the neonatal modified Kidney Diseases: Improving Global Outcomes (KDIGO) was proposed to allow for consistency throughout studies and it stages AKI using absolute rise of serum creatinine (S Cr) from a previous trough or the decrease of urine output over certain time frames.[14-15] Past studies have reported that the renal failure in asphyxiated neonates is often non-oliguric, with many neonates maintaining a urine output of more than 1 ml/kg/h despite

DOI: 10.69605/ijlbpr_13.12.2024.24

significant renal dysfunction.[16] Thus, more recent studies have adopted a serum creatinine based approach in defining neonatal AKI. However, using the changes in serum creatinine for the diagnosis of AKI has its limitations, including the presence of maternal creatinine and the delayed rise of S Cr following an insult.[17-18] The present study was conducted to study electrolyte disturbances in newborn with birth asphyxia and its association with acute kidney injury.

Materials and Method

The present cross-sectional analytical study aimed to investigate electrolyte disturbances in newborns with birth asphyxia and their association with acute kidney injury. The study was conducted at the Department of Pediatrics, People's College of Medical Sciences and Research Centre, Bhopal, over a period of 18 months from November 2022 to May 2024. All newborns with birth asphyxia admitted to the Neonatal Intensive Care Unit (NICU) were included in the study. The study variables comprised demographic and clinical details, including gender, birth weight, mode of delivery, APGAR score, vital signs, physical examination, management, anthropometry, maternal details, antenatal history, and laboratory investigations. The outcome of the baby was also recorded. The study explored the relationship between electrolyte disturbances and acute kidney injury in newborns with birth asphyxia, providing valuable insights into the management and care of these vulnerable patients.

The study included all newborns with birth asphyxia who were greater than 34 weeks of gestational age. However, newborns born before 34 weeks of gestational age were excluded from the study. Additionally, newborns diagnosed antenatally with renal problems or any gross congenital malformations that could affect kidney function postnatally were also excluded. Furthermore, parents who did not provide consent for their newborn's participation in the study were excluded. To classify and assess the newborns, specific tools were used: birth asphyxia was classified based on the APGAR score, electrolyte disturbances were determined by serum electrolyte levels, and acute kidney injury (AKI) was classified according to the p-RIFLE criteria. These tools enabled the researchers to accurately identify and analyze the relationships between birth asphyxia, electrolyte disturbances, and AKI in the study population.

All asphyxiated neonates who fulfilled the inclusion criteria were enrolled in the study. Informed written consent was obtained from parents/ guardian prior to enrollment of their babies in this study. A predesigned proforma was used to collect relevant data such as gestational age, birth weight, social demographic profile, urine output and relevant perinatal history. Findings on physical examination were recorded.

On the basis of Apgar score at 1minute, asphyxiated neonates were further grouped into mild (score of 6 or 7), moderate (score of 4 or 5), and severe asphyxia (score of 3 or less). The initial management of asphyxiated neonates was as per NICU protocol.

Serum electrolytes (sodium and potassium) blood urea and serum creatinine were monitored initially within the first 24 hours of birth and then on day 3 of life (between 72-96 hours of life). Urine output was monitored by applying plastic collection bag or by catheterization method. Newborn with birth asphyxia having electrolyte disturbances were detected and association with AKI was analyzed.

Statistical Package for the Social Sciences (SPSS version 17, IBM Corp., New York, NY, USA) was used for statistical analysis. In all tests, the statistical significance was indicated by $P < 0.05$.

Results

Table 1 Reveals that out of all participants 56% were male and 44% were female , out of which 70% were term, 25% were preterm & 4.6% were post term with 42% LBW.

Table 2 reveals that low sodium level found in 7% mild and 8% moderate asphyxia subjects and high sodium level found in 2% mild asphyxia subjects at 24 hours , low sodium level found in 5% mild asphyxia subjects and high sodium level found in 4% mild asphyxia subjects at 72 hours. Mild (P=0.94) and moderate (P=0.307) asphyxia was found statistically non significant with Sodium disturbances at different time interval. Table 3 reveals that high sodium level found in 18% mild, 16% moderate and 25% severe asphyxia subjects at 24 hours , low potassium level found in 4% mild asphyxia subjects and high potassium level found in 45 mild and 8% moderate asphyxia subjects at 72 hours. Mild (P=0.87) and moderate (P=0.53) and severe (P=0.28) asphyxia was found statistically non significant with potassium disturbances at different time interval. Table 4 shows incidence of AKI was wound 1.15%. Table 5 reveals that AKI was observed in 15 mild asphyxia study participant it was found statistically significant (P<0.01). Table 6 reveals that sodium level was found high in one subject in which AKI present where as in other subjects sodium level high in 5% subjects low in 3% subjects and normal in 90% subjects at 24 hours it was found statistically non significant (P=0.722), sodium level found normal in one subjects which had AKI present after 72 hours in other subjects sodium level was found high in 3% subjects, low in 4% subjects and normal in 91% subjects after 72 hours it was found statistically non significant (P=0.157). Table 7 reveals that potassium level was found normal in one subject in which AKI was present at 24 hours and 72 hours , potassium level was found high in 18% subjects at 24 hour and 9% subjects at 72 hours in which AKI was absent, potassium level was found low in 3% subjects at 72 hours in which AKI was absent and potassium level was found normal in 81% subjects at 24 hour and 82% subjects at 72 hours in

DOI: 10.69605/ijlbpr_13.12.2024.24

which AKI was absent it was found statistically significant (P=0.001).

Table 1: Demographic details of study participants

Demographic details		Frequency	Percent
Gender	Female	38	43.7
	Male	49	56.3
Period of gestation	<37 weeks	22	25.3
	37-42 weeks	61	70.1
	>42 weeks	4	4.6
Mother's age	<18 years	0	0
	18-35 years	84	96.55
	>35 years	3	3.44
Gravida	Primigravida	26	29.9
	Multigravida	61	70.1
Birth weight	Low Birth weight <2.5	37	42.52
	Normal Birthweight>2.5	50	57.47

Table 2: Association of Sodium derangement with birth asphyxia at different time interval

Asphyxia	Sodium Level	24 Hours		72 Hours		Chi square value; p value
		Frequency	Percent	Frequency	Percent	
Mild	Hypo	5	7.04	4	5.63	0.11; 0.94
	Hyper	3	2.22	3	4.22	
	Normal	63	88.73	64	90.14	
Moderate	Hypo	1	8.33	0	0	1.04; 0.307
	Hyper	0	0	0	0	
	Normal	11	91.66	12	100.0	
Severe	Hypo	0	0	0	0	NA
	Hyper	0	0	0	0	
	Normal	4	100.0	4	100.0	

Table 3: Association of Potassium derangement with birth asphyxia at different time interval

Asphyxia	Potassium Level	24 Hours		72 Hours		Chi square value; p value
		Frequency	Percent	Frequency	Percent	
Mild	Hypo	0	0	3	4.22	4.87; 0.87
	Hyper	13	18.30	7	9.85	
	Normal	58	81.7	61	85.9	
Moderate	Hypo	0	0	0	0	0.380; 0.53
	Hyper	2	16.66	1	8.33	
	Normal	10	83.3	11	91.7	
Severe	Hypo	0	25.0	0	0.0	1.14; 0.28
	Hyper	1	25.0	0		
	Normal	3	75.0	4	100.0	

Table 4: Incidence of AKI among study subjects

AKI	Frequency	Percent
Absent	86	98.85
Present	1	1.15

Table 5: Severity of birth asphyxia in relation to AKI

Asphyxia	AKI	Frequency	Percent
Mild	Absent	70	98.6
	Present	1	1.4
Moderate	Absent	12	100.0
Severe	Absent	4	100.0
Chi square value 13.41; p value -<0.01*			

DOI: 10.69605/ijlbpr_13.12.2024.24

Table 6: Association of sodium Level derangement with AKI at different time interval Sodium

AKI	Sodium Level	24 Hours		72 Hours		Chi square value; p value
		Frequency	Percent	Frequency	Percent	
Absent	Hyper	5	5.81	3	3.48	0.64; 0.722
	Hypo	3	3.48	4	4.65	
	Normal	78	90.7	79	91.86	
Present	Hyper	1	100.0	0	0	2.00; 0.157
	Hypo	0	0	0	0	
	Normal	0	0.0	1	100.0	

Table 7: Association of potassium level derangement with AKI at different time interval

AKI	Potassium Level	24 Hours		72 Hours		Chi square value; p value
		Frequency	Percent	Frequency	Percent	
Absent	Hyper	16	18.6	8	9.30	12.77;0.001
	Hypo	0	0	3	3.48	
	Normal	70	81.4	75	87.2	
Present	Hyper	0	0	0	0	NA
	Hypo	0	0	0	0	
	Normal	1	100.0	1	100.0	

Discussion

Birth asphyxia, is the primary avoidable factor leading to brain damage in infants, causing a significant number of deaths and health issues in underprivileged countries. Perinatal asphyxia continues to be a prominent cause of mortality and acquired brain injury in babies worldwide, despite notable advancements in monitoring technologies, prenatal awareness, and neonatal disease management.[19] Timely identification of electrolyte imbalances and acute kidney injury (AKI) is crucial in newborns with birth asphyxia to enable proper management of fluids and electrolytes.

So, in present study we decided to find out the electrolyte disturbances in newborns with birth asphyxia and its association of severity of electrolyte disturbance and determined the occurrence of AKI in newborn with birth asphyxia. We made an attempt to find out the association of grade of AKI in relation to severity of birth asphyxia and electrolyte disturbances. The present study found that among a total of 87 individuals, consisting of 38 females and 49 males with majority of patients in our study were males i.e. 56.3%. Similarly, **Medani SA et al[20] found** 58.9% were males, **Prodhan MS et al[21]** found 61.67% were male, and 38.33% were female. **Thakur J et al[22]** found 68% were males in their study. According to this study, there were a higher percentage of male newborns compared to female neonates, which aligns with previous research findings.

In the present study the gestation age was seen to be less than 37 weeks in 25.3% patients, between 37 and 42 weeks in 56% subjects, and greater than 42 weeks in 4.6% subjects. In concordance to our study, **Tounsa A et al[23]** reported average gestational age was 38.29 ± 1.07 weeks. **Carroll WF et al[24]**included gestational age of 26 weeks (23-31). **Mamo SA et al[25]**, Gestational age <37weeks was reported in 13.8% cases and >42weeks in 5.8%. Our results are in accordance with **Mamo SA et al.[25]**

In present study, the majority of the mothers (96.55%) were aged between 18-35 years and only 3.44% were aged above 35 years. **Alamneh YM et al[26]** found that the age range of 19-34 years accounted for the majority i.e. 71.8% of cases and nearly half i.e.49.6% of controls. **Igboanugo S et al[27]** mentioned that maternal age below 20 years had significant and consistent impact on the risk of BA. **Khound M et al[28]**found no statistically significant correlation between maternal age, mother educational attainment, and parity and the occurrence of newborn asphyxia.

In our study majority of asphyxiated neonates i.e. 70.1% were born to Multigravida mothers. Our study is in concordance with other studies reported by **Khound M et al[28] and Mamo SA et al.[25] Khound M et al[28]**reported that multiple pregnancies have a significant impact on birth asphyxia. However, **Prodhan MDS et al[21]** reported that 70% were primipara, while only 30% of asphyxiated neonates were born to multigravida moms. Our result is in contrast to those reported by **Basu P et al[29] and Bhuiyan SJ[20] as** they mentioned that the decreased expansibility of the delivery canal in primipara moms, as well as the prolongation of labor in such situations, may be the cause.

Low birth weight <2.5 kg was observed among 42.52% subjects where as 57.47% subjects had normal weight >2.5 kg in the present study. **Tounsa A et al[23]** included neonates having birth weight ≥2 kg in their study. **Wosenu L et al et al[31]**found that low birth weight (AOR = 7.72, 95% CI: 1.88, 31.68) was strongly associated with an increased likelihood of birth asphyxia. **Tette EMA et al[32] and Suleiman BM et al[33]** found that severe prenatal hypoxia was the primary cause of mortality in all birth weight categories, save for extremely low birth weight

DOI: 10.69605/ijlbpr_13.12.2024.24

infants. However, no significant difference was evident in present study.

In our study, deranged sodium was found in 10.3% subjects at 24 hours and in 78% subjects after 72 hours. Statistically, there was no significant association detected between mild asphyxia (P=0.94) and strong asphyxia (P=0.307) with Sodium disturbances at various time intervals. Whereas **Thakur J et al**[22] and **Gupta BD et al**[34]found a significant difference in the levels of sodium among different degrees of hypoxia (p-value< 0.001). **Prodhan MDS et al**[21] found a positive correlation between serum sodium levels and the severity of hypoxia was found to be statistically significant. **Odo KE et al**[35] in their study reported sodium level was significantly lower. The reason for hyponatremia is the limited capacity of sodium reabsorption. If there is a considerable increase in the load of sodium reaching the Collecting Tubules (CT), reabsorption does not occur proportionately and the excess sodium is expelled in the urine.

In our study, deranged potassium was found in 18.4% subjects at 24 hours and in 12.6% subjects after 72 hours. Potassium derangement with birth asphyxia at different time interval results revealed that high potassium level, however the difference obtained was not significant. Similar result was reported by **Basu P et al**.[29] Our results are in accordance with **Vandana V et al,**[36] there was no significant difference in the mean values of electrolytes among neonates who were asphyxiated, both in cases and controls, as well as in different phases of HIE. Newborns experience hyperkalemia during the early neonatal period as a result of the movement of potassium from inside the cells to the area outside the cells. The extent of this change is directly related to the level of immaturity, meaning that the more preterm the baby, the higher the likelihood of hyperkalemia. Our results differ from those reported by **Odo KE et al**[35]**, Prodhan MDS et al**[21] **and Thakur J et al**[22]**,** they found a significant difference. The difference obtained could be due to difference in the sample size.

In current study incidence of AKI was 1.15%. **Tounsa A et al**[23]found in 18.02%, which was higher than those reported in our study. **D Alaro et al**[37]prevalence of AKI was 11.7 %.**Ramagopal G et al**[38] reported the incidence of acute kidney injury (AKI) in cases of birth asphyxia was determined to be 75.0%.

In present study the analysis of the birth asphyxia severity in connection to acute kidney injury showed that AKI was seen in one participant with mild asphyxia, and this finding was statistically significant (P<0.01). Our study is in accordance with **Prodhan MDS et al**[21]whofound a correlation between the occurrence and severity of prenatal hypoxia and the rising frequency of AKI. **Medani SA et al**[20]suggested that AKI was shown to be a frequent consequence in neonates who experienced suffocation. Perinatal hypoxia was identified as the primary cause of AKI in the investigations conducted by **Gallo D et al.**[39] (72%) and **Nandhagopal N et al**. (74%). [40]

Analysis of sodium levels in subjects with acute kidney injury (AKI) at different time intervals revealed that after 24 and 72 hours, there was no statistically significant difference in sodium levels (P=0.722). Our results are similar to those reported by **Refat et al**.[41] **Prodhan MDS et al**[21], who found a statistically significant correlation was seen between serum sodium levels and the grade of asphyxia. The researchers determined that in the observed cases, both hyponatremia and hypocalcemia occurred early and concurrently. Furthermore, the reduction in their serum levels was directly related to each other and to the severity of hypoxia.**Basu P et al.**[29]

Association of potassium levels derangement with AKI at different time interval results revealed that potassium level at 72 hrs in which AKI was absent and potassium level was found normal in 70 subjects at 24 hr and 75 subjects at 72 hrs in which AKI was absent it was found statistically significant (P=0.001). Similar result was reported by **Prodhan MDS et al**.[21]**Ali MA et al**[42] reported that Mean serum potassium level was 4.94 ± 0.92.**Thakur J**[22] mentioned that, it may result from abrupt renal failure caused by birth asphyxia, leading to reduced potassium excretion and hence hyperkalemia. According to **Basu P et al.**[29]**,** the average levels of potassium, was 5.05 ± 0.63 meq/l. The occurrence of high levels of hyperkalemia in the moderate and severe case group members was consistent with previous research findings.

The disparity in the outcomes can likely be attributed to variations in the timing of sample collection. We made a point to obtain blood samples during the first hour of life, minimizing the opportunity for the body's internal environment to fix electrolyte imbalances. There is a scarcity of research on electrolyte imbalance in newborns that have experienced asphyxia, particularly in relation to the severity of the asphyxia.

Smaller sample size in present study was one of the limitations observed. In present study any associated mortality and morbidly were not studied. Multiple future studies are required to confirm such results and serial analysis of electrolytes with larger sample size is warranted.

Conclusion

To conclude, we found that birth asphyxia was more common in males. Mothers age and low birth weight have a strong impact in birth asphyxia. The study results indicate that electrolyte imbalances are frequently detected in patients with birth asphyxia. Babies with birth asphyxia have a greater incidence of hyperkalemia , and there is a considerable increase in potassium levels that is directly related to the severity of the asphyxia. AKI is more prevalent in babies with birth asphyxia. We recommend that there should be

DOI: 10.69605/ijlbpr_13.12.2024.24

early identification of the infants who are prone to poor outcome

References

1. Srivastav RN "Acute renal failure in the Neonate" Periogynatol 2004, Vol 6 (4) :176-182.
2. Perlman JM, Risser R. Can asphyxiated infants at risk for neonatal seizures be rapidly identified by current high-risk markers? Pediatrics. 1996;97(4):456–62.
3. Gupta J, Bora B. A study of serum electrolytes level in newborns with birth asphyxia. International Journal Dental and Medical Sciences Research.2021;3(3):265-268.
4. Utture AA, Kanbur WK, Mondkar JM, Fernandez A R and Lokeshkar M R. Perinatal Asphyxia. Perinatology .1999; 1 (2): 87-96.
5. Victor Y H Yu Perinatology. 1999; 1(5): 251-266.
6. Peliowski A and Finer NN. Birth asphyxia in the term infant in Effective care of the Newborn. Sinclair J C and Bracken M B.1992: 249-275.
7. Perlman JM, Tack ED. Renal injury in the asphyxiated newborn infant: Relationship to neurologic outcome. J Pediatr 1988; 113: 875– 79.
8. Perlman JM, Tack ED, Martin T, Shackelford G, Amon E. Acute systemic organ injury in term infants after asphyxia. Am J Dis Child 1989; 143: 617-620.
9. Cohn HE, Sacks EJ, Heymann MA, et al. Cardiovascular responses to hypoxemia and acidemia in fetal lambs. Am J Obstet Gynecol. 1974 Nov 15;120(6):817-24.
10. Rudolph AM. The fetal circulation and its response to stress. J Dev Physiol. 1984 Feb;6(1):11-9.
11. Saikumar P, Venkatachalam MA. Role of apoptosis in hypoxic/ischemic damage in the kidney. *Semin. Nephrol.* 2003;**23**(6):511–521. doi: 10.1053/S0270-9295(03)00130-X.
12. Alaro D, et al. Prevalence and outcomes of acute kidney injury in term neonates with perinatal asphyxia. *Afr. Health Sci.* 2014;**14**(3):682–688. doi: 10.4314/ahs.v14i3.26.
13. Tanigasalam V, et al. Does therapeutic hypothermia reduce acute kidney injury among term neonates with perinatal asphyxia?—a randomized controlled trial. *J. Matern. Fetal. Neonatal. Med.* 2016;**29**(15):2545–2548.
14. Khwaja A. KDIGO clinical practice guidelines for acute kidney injury. *Nephron. Clin. Pract.* 2012;**120**(4):c179–c184.
15. Selewski DT, et al. Neonatal acute kidney injury. *Pediatrics.* 2015;**136**(2):e463–e473. doi: 10.1542/peds.2014-3819.
16. Karlowicz MG, Adelman RD. Nonoliguric and oliguric acute renal failure in asphyxiated term neonates. *Pediatr. Nephrol.* 1995;**9**(6):718–722. doi: 10.1007/BF00868721.
17. Drukker A, Guignard JP. Renal aspects of the term and preterm infant: A selective update. *Curr. Opin. Pediatr.* 2002;**14**(2):175–182. doi: 10.1097/00008480-200204000-00006.
18. Guignard JP, Drukker A. Why do newborn infants have a high plasma creatinine? *Pediatrics.* 1999;**103**(4):e49. doi: 10.1542/peds.103.4.e49.
19. Shirin N, Nahar N, Mollah AH. Risk factor, and short term outcome of birth asphyxiated babies in Dhaka medical college hospital, Bangladesh J Child Health. 2009;33(3):83-9.
20. Medani SA, Kheir AE, Mohamed MB. Acute kidney injury in asphyxiated neonates admitted to a tertiary neonatal unit in Sudan. Sudan J Paediatr. 2014;14(2):29-34.
21. Prodhan MS, Moniruzzaman A, Majumder B, Rahman MJ, Suja-Ud-Doula A, Nabi SN, et al. Serum electrolyte level and renal functional status in perinatal asphyxia. *Dinajpur Med Col J.* 2017;10:127–32.
22. Thakur J, Bhatta NK, Singh RR, Poudel P, Lamsal M, Shakya A. Prevalence of electrolyte disturbances in perinatal asphyxia: a prospective study. Ital J Pediatr. 2018 May 21;44(1):56.
23. Tounsa A, Hussain A, Hussain I, Tariq R, Saqlain M, Shaikh SA, Mumtaz H. Acute kidney injury in birth asphyxiated patients: A cross sectional study at Bahawal Victoria Hospital. Lung India. 2024 Jan 1;41(1):30-34.
24. Carroll WF, Fabres J, Nagy TR, Frazier M, Roane C, Pohlandt F, Carlo WA, Thome UH. Results of extremely-low-birth-weight infants randomized to receive extra enteral calcium supply. J Pediatr Gastroenterol Nutr. 2011 Sep;53(3):339-45.
25. Mamo SA, Teshome GS, Tesfaye T, Goshu AT. Perinatal asphyxia and associated factors among neonates admitted to a specialized public hospital in South Central Ethiopia: A retrospective cross-sectional study. PLoS One. 2022 Jan 13;17(1):e0262619
26. Alamneh YM, Negesse A, Aynalem YA, Shiferaw WS, Gedefew M, Tilahun M, Hune Y, Abebaw A, Biazin Y, Akalu TY. Risk Factors of Birth Asphyxia among Newborns at Debre Markos Comprehensive Specialized Referral Hospital, Northwest Ethiopia: Unmatched Case-Control Study. Ethiop J Health Sci. 2022 May;32(3):513-522.
27. Igboanugo, S., Chen, A., & Mielke, J. G. (2019). Maternal risk factors for birth asphyxia in low-resource communities. A systematic review of the literature. Journal of Obstetrics and Gynaecology, 40(8), 1039–1055.
28. Khound M, Sharma SJ, Baruah PK. Risk Factors for Low Birth Weight Babies in Healthy Literate Mothers Belonging To Middle Socio Economic Status: A Hospital Based Observational Risk Factors for Low Birth Weight Babies in Healthy Literate Mothers Belonging To Middle Socio Economic Status. IOSR J Pharm. 2020;10(11):12-9.
29. Basu P, Som S, Das H, Choudhuri N. Electrolyte status in birth asphyxia. Indian J Pediatr. 2010 Mar;77(3):259-62. doi: 10.1007/s12098-010-0034-0. Epub 2010 Feb 22.
30. Bhuiyan SJ. Incidence, risk factors, and immediate outcome of Asphyxia Neonatorum in hospitalized neonates: Dept. of Paed IPGMR [Dessertation]. Dhaka: BCPS; 1996.
31. Wosenu L, Worku AG, Teshome DF, Gelagay AA. Determinants of birth asphyxia among live birth newborns in University of Gondar referral hospital, northwest Ethiopia: A case-control study. PLoS One. 2018 Sep 7;13(9):e0203763.
32. Tette EMA, Nartey ET, Nuertey BD, Azusong EA, Akaateba D, Yirifere J, et al. The pattern of neonatal admissions and mortality at a regional and district

DOI: 10.69605/ijlbpr_13.12.2024.24

hospital in the Upper West Region of Ghana; a cross sectional study. PloS one. 2020;15(5):e0232406.
33. Suleiman BM, Mokuolu OA, Adesiyun OO, Adeniyi A. Pattern of perinatal mortality in babies delivered at the university of ilorin teaching hospital, ilorin, Nigeria. West African journal of medicine. 2012;31(2):102–108.
34. Gupta BD, Sharma P, Bagla J, Parakh M, Soni JP. Renal failure in asphyxiated neonates. Indian Pediatr. 2005 Sep;42(9):928-34.
35. Odo KE, Odetunde OI, Chinawa JM, Okafor HU, Aronu AE, Adimora GN. Comparison of plasma electrolytes of perinatally asphyxiated and normal term babies. J Neonatal Perinatal Med. 2019;12(4):385-389.
36. Vandana V, Amit V, Meena V, Anuradha B, Vivek B, Deepak V, Salone MR. Study of basic biochemical and haematological parameters in perinatal asphyxia and its correlation with hypoxic ischemic encephalopathy staging. J Adv Res Biol Sci. 2011;**3**(2):79–85.
37. D Alaro, A Bashir, R Musoke, L Wanaiana. Prevalence and outcomes of acute kidney injury in term neonates with perinatal asphyxia. African health sciences, 2014
38. Ramagopal G, Narayana G, Premalatha R, Belavadi GB. Incidence of acute renal failure (ARF) in birth asphyxia and its correlation with hypoxic ischemic encephalopathy (HIE) staging. Indian J Neonat Med Res. 2016;4:1–4
39. Gallo D, De Bijl-Marcus KA, Alderliesten T, Lilien M, Groenendaal F. Early acute kidney injury in preterm and term neonates: Incidence, outcome and associated clinical features. Neonatology. 2021;118(2):174-79.
40. Nandagopal N, Reddy PK, Ranganathan L, Ramakrishnan N, Annigeri R, Venkataraman R. Comparison of epidemiology and outcomes of acute kidney injury in critically ill patients with and without sepsis. Indian journal of critical care medicine: peer-reviewed, official publication of Indian Society of Critical Care Medicine. 2020 Apr;24(4):258.
41. Refat et al Early Predictor of Acute Kidney Injury in Newborns with Perinatal Asphyxia. J Child Sci 2023;13:e104–e112.
42. Ali MA, Rehman A, Ahmed E. Association of In-hospital outcome of Acute Kidney Injury (AKI) with etiology among newborns at a tertiary care unit. Pak J Med Sci.