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Frequency of Hypocalcaemia and Hypomagnesaemia in Neonates with Birth Asphyxia

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ABSTRACT

Birth asphyxia is a major cause of mortality and morbidity in neonates in developing nations. **Objective:** To determine the frequency of hypocalcaemia as well as hypomagnesaemia amongst neonates having birth asphyxia. **Methods:** This descriptive cross-sectional study was performed at the department of Pediatrics, Ziauddin Campus, north nazimabad, Karachi from July 2021 to December 2021. A total of 207 asphyxiated term neonates with postnatal age up to 48 hours, and history of birth asphyxia were analyzed. Venous blood sample of 5 ml was drawn by a trained phlebotomist and sent to institutional laboratory for serum analysis of calcium and magnesium levels within first 24 hours of admission. Frequency of hypocalcaemia and hypomagnesaemia were recorded. Chi square or fisher's exact test were applied taking p≤0.05 as significant to judge inferential statistics. **Results:** In a total of 207 neonates, 112 (54.1%) were male. The mean post-natal age, and gestational age were 16.6±8.4 hours, and 37.7 ± 0.9 weeks, respectively. The mode of delivery was cesarean section in 121 (58.5%) cases. The frequency of hypomagnesaemia, and hypocalcaemia were documented in 36 (17.4%), and 46 (22.2%) neonates with birth asphyxia, respectively. **Conclusions:** Among term neonates with birth asphyxia, considerable frequency of hypocalcemia and hypomagnesemia was observed. These findings highlight the frequent occurrence of electrolyte imbalances in asphyxiated neonates, supporting the need for routine early biochemical screening.

INTRODUCTION

Prenatal Asphyxia (PA) is among the leading causes of neonatal mortality as almost 20% of neonatal deaths are caused by prenatal asphyxia [1, 2]. The overall frequency of PA is estimated to be 2-9 / 1,000 live full-term births [3, 4]. The incidence of PA in developing nations is 10 times higher than the developed countries [5]. Neonatal asphyxia is generally characterized by encephalopathy, low Apgar score (<7 at minute 5) and umbilical cord PH < 7.2 Prenatal asphyxia may lead to hypoxic damage in various organs of the body, while at the cellular level, these processes may initiate an inundation of a multitude of biochemical alterations leading to cell death [6]. Birth asphyxia is often linked with metabolic alterations such as hypoglycemia, hypocalcemia, hyponatremia, hyperphosphatemia and metabolic acidosis [7]. Neonatal asphyxia causes excessive production of glutamate amino acid in the brain. In the initial few days of life, excess glutamate causes permanent neuronal damage due to the activation of some receptors and influx of Calcium (Ca) in neurons [8]. Magnesium (Mg) hinders these receptors and facilitates a neuroprotective functioning blocking the effects of asphyxia on various body organs [9, 10]. During an ischemic

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insult, hypomagnesemia could be due to augmented Mg intake or continued losses resulting from ischemic injury to renal glomeruli and tubules [11]. Upon birth due to sudden stoppage of placental transfer of Ca thereby, levels begin to decrease to 8-9 mg/dl and ionized Ca to 4.4-5.4 mg/dl at 24 hours of life. Serum Ca subsequently begins to increase to levels similar to those of older children and adults by 2 weeks of life [12-14]. Although electrolyte disturbances like hypocalcemia and hypomagnesemia are recognized complications of birth asphyxia, there is limited data from Pakistan quantifying their frequency in affected neonates. Little emphasis has been documented on early metabolic derangements in birth asphyxia. This study addresses that gap by evaluating these biochemical abnormalities in asphyxiated term neonates. This study was done to determine the frequency of hypocalcaemia as well as hypomagnesaemia amongst neonates having birth asphyxia.

The exact estimation of the frequency of hypocalcaemia and hypomagnesaemia may help us arranging appropriate resources and management options to reduce the burden of these disorders among neonates with birth asphyxia.

METHODS

This descriptive cross-sectional study was conducted at the department of Pediatrics, Ziauddin campus, north nazimabad, Karachi from July 2021 to December 2021. A sample size of 207 was calculated considering confidence level of 95%, with anticipated proportion of hypomagnesemia as 16%, and margin of error as 5% using OpenEPI online sample size calculator through the formula: n = z2 * p * (1 - p) / e2 [2]. The inclusion criteria were asphyxiated term neonates (5 min APGAR score <7 as per medical record) of any gender, having gestational age ≥ 37 weeks, and postnatal age ≤ 48 hours were enrolled through emergency department [15]. The exclusion criteria were cases with congenital abnormality, renal dysfunction (serum creatinine>1.5 mg/dl), history of diuretics treatment, or maternal hypertension, diabetes mellitus, or toxaemia. Informed and written consents were obtained from parents/guardians. Approval from Institutional Ethical Committee was obtained (Reference: 3490322RAPED). Non-probability, consecutive sampling was adopted. Detailed history and clinical findings like age, gender, gestational age, birth weight, place and mode of delivery, and residence were noted. Institutional laboratory was used for the evaluation of serum Ca, and Mg levels within 1st 24-hour of admission. Frequency of hypocalcaemia, and hypomagnesaemia were recorded. Hypocalcaemia was defined as serum Ca<8.0 mg/dL [13]. Hypomagnesaemia It was defined as serum Mg <1.5 mg/dL [16, 17]. Data were analyzed using IBM-SPSS Statistics, version 26.0. Qualitative variables like like gender, presence of hypocalcaemia, presence of hypomagnesaemia, mode of delivery and place of delivery were represented as frequencies and percentages. Variables like age, gestational age, birth weight (grams) and height (cm) were represented as mean and standard deviations. Effect modifiers were controlled through stratification. Post-stratification, chi square/fisher's exact test were applied. P-value≤0.05 was considered as significant.

RESULTS

In a total of 207 neonates, 112 (54.1%) were male. Mean post-natal age was calculated to be 16.6±8.4 hours while 136 (65.7%) children were aged less than 24 hours. Mean gestational age was 37.7±0.9 weeks whereas 178 (86.0%) mothers of asphyxiated neonates had gestational age between 37 to 38 weeks. Mode of delivery was cesarean section in 121 (58.5%) cases. Place of deliver of 135 (65.2%) neonates was inborn. Area of residence of 119 (57.5%) neonates was rural. Mean body weight was calculated to be 2.52 ± 0.24 grams while 128 (61.8%) neonates had birth weight above or equal to 2.5 kg. Mean body height was noted to be 48.2±2.4 cm while 118 (57.0%) neonates had body height above or equal to 46cm. Table 1 is showing baseline characteristics of neonates with birth asphyxia.

Table 1: Characteristics of Neonates (n=207)

Characteristics	Category	Frequency (%)
Gender	Boys	112 (54.1)
	Girls	95 (45.9)
Post-Natal Age in Hours	<24	136 (65.7)
	≥24	71 (34.3)
Gestational Age in Weeks	37-38	178 (86.0)
	>38	29 (14.0)
Mode of Delivery	Cesarean Section	121 (58.5)
	Normal Vaginal Delivery	86 (41.5)
Place of Delivery	Inborn	135 (65.2)
	Out-born	72 (34.8)
Residence	Rural	119 (57.5)
	Urban	88 (42.5)
Birth Weight in Kg	<2.5	79 (38.2)
	≥2.5	128 (61.8)
Body Height in cm	<46	89 (43.0)
	≥46	118 (57.0)

Hypomagnesaemia was found in 36 (17.4%) neonates with birth asphyxia while hypocalcaemia was noted 46 (22.2%) neonates with birth asphyxia. Three (1.1%) asphyxiated neonates had both hypomagnesaemia and hypocalcaemia. Except mode of delivery (p=0.0096), all other variables were not found to have any statistically significant association with hypomagnesaemia (p>0.05), as shown in table 2.

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Table 2: Association of Hypomagnesaemia with Study Variables (n=207)

Variables	Category	Hypomagnesaemia		p-Value
		Yes Frequency (%)	No Frequency (%)	
Gender	Boys	19 (52.8)	93 (54.4)	0.8603
	Girls	17 (47.2)	78 (45.6)	
Post-Natal Age in Hours	<24	26 (72.2)	110 (64.3)	0.3644
	≥24	10 (27.8)	61 (35.7)	
Gestational Age in Weeks	37-38	28 (77.8)	150 (87.7)	0.1183
	>38	8 (22.2)	21 (12.3)	
Mode of Delivery	Cesarean Section	28 (77.8)	93 (54.3)	0.0096*
	Normal Vaginal Delivery	8 (22.2)	78 (45.7)	
Place of Delivery	Inborn	21 (58.3)	114 (66.7)	0.3400
	Out-born	15 (41.7)	57 (33.3)	
Residence	Rural	23 (63.9)	96 (56.1)	0.3927
	Urban	13 (36.1)	75 (43.9)	
Birth Weight in Kg	<2.5	21 (58.3)	58 (33.9)	0.0061*
	≥2.5	15 (41.7)	113 (66.1)	
Body Height in cm	<46	14 (38.9)	75 (43.9)	0.5840
	≥46	22 (61.1)	96 (66.1)	

*Indicates statistical significance at p-value ≤ 0.05.

Table 3 showed stratification of study variables with respect to frequency of hypocalcaemia among neonates with birth asphyxia and no significant associations were found (p>0.05).

Table 3: Association of Hypocalcaemia with Study Variables (n=207)

Characteristics	Category	Hypocalcaemia		p-Value
		Yes Frequency (%)	No Frequency (%)	
Gender	Boys	30 (65.2)	82 (50.9)	0.0864
	Girls	16 (34.8)	79 (49.1)	
Post-Natal Age in Hours	<24	35 (76.1)	101 (62.7)	0.0924
	≥24	11 (23.9)	60 (37.3)	
Gestational Age in Weeks	37-38	39 (84.8)	139 (86.3)	0.7890
	>38	7 (15.2)	22 (13.7)	
Mode of Delivery	Cesarean Section	30 (65.2)	91 (56.5)	0.2912
	Vaginal Delivery	16 (34.8)	70 (43.5)	
Place of Delivery	Inborn	28 (60.9)	107 (66.5)	0.4827
	Out-born	18 (39.1)	54 (33.5)	
Residence	Rural	31 (67.4)	88 (54.7)	0.1234
	Urban	15 (32.6)	73 (45.3)	
Birth Weight in Kg	<2.5	17 (37.0)	62 (38.5)	0.8484
	≥2.5	29 (63.0)	99 (61.5)	
Body Height in cm	<46	18 (39.1)	71 (44.1)	0.5483
	≥46	28 (60.9)	90 (55.9)	

DISCUSSION

Metabolic dysfunctions in asphyxiated newborns like hypoglycemia, hyperammonemia, hypocalcemia and hypomagnesemia are known to be linked with PA [17, 18]. This study also found that 17.4% neonates who were birth asphyxia cases suffered from hypomagnesaemia. Cotemporary research is arguing about the relationship of hypermagnesemia and asphyxia [19-21]. Some researchers have confirmed sudden loss of concentrations of Mg ions of brain in rats in course of hypoxic damage like shown by McIntosh et al [19]. Some other studies have showed that Mg infusion enhanced short-outcome in neonatal asphyxia [20, 21]. The etiology underlying alterations in total Mg levels in asphyxiated neonates remains unclear. PA can increase serum calcitonin, which suppresses the release of Ca from bone and leads to hypocalcemia. In other neonates, the phosphaturic renal response to parathyroid hormone is blunted; the high phosphate concentration results in hypocalcaemia [12, 13]. A study from Bangladesh analyzing asphyxiated term neonates revealed that hypocalcaemia was present in 43.1% cases, while hypomagnesaemia was present in 34.3% [14]. In another study from Iran, 16% of neonates with asphyxia had hypomagnesaemia [2]. In another study, the mean serum Mg levels were 1.6 ± 0.3 in Hypoxic Ischemic Encephalopathy (HIE) neonates versus 1.8±0.4 mg/dl among controls [15]. In this research, hypocalcaemia frequency was observed to be 22.2% in neonates with birth asphyxia. Improda et al., identified that there was a highly significant reduction in the extracellular Ca concentration in asphyxiated infants, and the reduction was directly proportional to the severity of asphyxia [7]. Similar to that documented by Bhoye et al., who established that the reduction in the serum concentration of both the total Ca and the ionized Ca was greater in asphyxiated neonates than in non-asphyxiated neonates [22]. PA is a comprehensive issue that leads to morbidity and mortality but recent trends have shown that the incidence of global neonatal mortality have decreased from 36/1,000 live-births to 19/1,000 in the last decade [23]. Perinatal and neonatal mortality due to PA indicate social, educational and economic principles of a society. In developing nations, such as in Pakistan where health centers are limited to cities, asphyxia prevalence is higher; however, the subject with limited local data has been extensively reviewed and researched all-inclusive.

CONCLUSIONS

Among term neonates with birth asphyxia, considerable frequency of hypocalcemia and hypomagnesemia was observed. These findings highlight the frequent occurrence of electrolyte imbalances in asphyxiated neonates, supporting the need for routine early biochemical screening. Early identification and timely correction of calcium and magnesium deficiencies may be essential components of supportive care in neonates suffering from birth asphyxia.

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Authors Contribution

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Writing, review and editing: A, MI, VK, FZ, A, LK

All authors have read and agreed to the published version of the manuscript

Conflicts of Interest

All the authors declare no conflict of interest.

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REFERENCES

[1] Kawakami MD, Sanudo A, Teixeira ML, Andreoni S, de Castro JQ, Waldvogel B *et al.* Neonatal mortality associated with perinatal asphyxia: a population-based study in a middle-income country. BioMed Central Pregnancy and Childbirth. 2021 Dec; 21: 1-0. doi: 10.1186/s12884-021-03652-5.

[2] Khalessi N, Mazouri A, Bassirnia M, Afsharkhas L. Comparison between serum magnesium levels of asphyxiated neonates and normal cases. Medical Journal of the Islamic Republic of Iran. 2017 Mar; 31: 19. doi: 10.18869/mjiri.31.19.

[3] Eghbalian F. Frequency of hypoxic-ischemic encephalopathy among hospitalized neonates in West Iran. Iranian Journal of Pediatrics. 2010 Jun; 20(2): 244-245.

[4] Bayih WA, Yitbarek GY, Aynalem YA, Abate BB, Tesfaw A, Ayalew MY *et al.* Prevalence and associated factors of birth asphyxia among live births at Debre Tabor General Hospital, North Central Ethiopia. BioMed Central Pregnancy and Childbirth. 2020 Dec; 20: 1-2. doi: 10.1186/s12884-020-03348-2.

[5] Getaneh FB, Sebsbie G, Adimasu M, Misganaw NM, Jember DA, Mihretie DB *et al.* Survival and predictors of asphyxia among neonates admitted in neonatal intensive care units of public hospitals of Addis Ababa, Ethiopia, 2021: a retrospective follow-up study. BioMed Central Pediatrics. 2022 May; 22(1): 262. doi: 10.1186/s12887-022-03238-w.

[6] Wu L, Chang E, Zhao H, Ma D. Regulated cell death in hypoxic-ischaemic encephalopathy: recent development and mechanistic overview. Cell Death Discovery. 2024 Jun; 10(1): 277. doi: 10.1038/s41420-024-02014-2.

[7] Improda N, Capalbo D, Poloniato A, Garbetta G, Dituri F, Penta L *et al.* Perinatal asphyxia and hypothermic treatment from the endocrine perspective. Frontiers in Endocrinology. 2023 Oct; 14: 1249700. doi: 10.3389/fendo.2023.1249700.

[8] Mota-Rojas D, Villanueva-García D, Solimano A, Muns R, Ibarra-Ríos D, Mota-Reyes A. Pathophysiology of perinatal asphyxia in humans and animal models. Biomedicines. 2022 Feb; 10(2): 347. doi: 10.3390/biomedicines10020347.

[9] Patel V, Akimbekov NS, Grant WB, Dean C, Fang X, Razzaque MS. Neuroprotective effects of magnesium: Implications for neuroinflammation and cognitive decline. Frontiers in Endocrinology. 2024 Sep; 15: 1406455. doi: 10.3389/fendo.2024.1406455.

[10] Maier JA, Locatelli L, Fedele G, Cazzaniga A, Mazur A. Magnesium and the brain: A focus on neuroinflammation and neurodegeneration. International Journal of Molecular Sciences. 2022 Dec; 24(1): 223. doi: 10.3390/ijms24010223.

[11] Liu Z, Wang R, He M, Kang Y. Hypomagnesemia is associated with the acute kidney injury in traumatic brain injury patients: a pilot study. Brain Sciences. 2023 Mar; 13(4): 593. doi: 10.3390/brainsci13040593.

[12] Zaman R, Mollah MA, Chowdhury MM, Yeasmin S, Chowdhury AS, Saha D. Serum magnesium and calcium status among term asphyxiated newborns with moderate to severe hypoxic-ischemic encephalopathy (HIE). Journal of Dhaka Medical College. 2017 Nov; 26(2): 148-52. doi: 10.3329/jdmc.v26i2.38833.

[13] Hossain MM, Mannan MA, Yeasmin F, Shaha CK, Rahman MH, Shahidullah M. Short-term outcome of magnesium sulfate infusion in perinatal asphyxia. Mymensingh Medical Journal. 2013 Oct; 22(4): 727-35.

[14] Njie AE, Nyandiko WM, Ahoya PA, Moutchia JS. A comparative analysis of APGAR score and the gold standard in the diagnosis of birth asphyxia at a tertiary health facility in Kenya. PLOS One. 2023 May; 18(5): e0285828. doi: 10.1371/journal.pone.0285828.

[15] Joiya SJ, Khan MA, Shabbir I. Hypomagnesaemia among children at pediatric intensive care unit, Nishtar Hospital, Multan. The Professional Medical Journal. 2020 Mar; 27(03): 461-6. doi: 10.29309/TPMJ/2020.27.03.2478.

[16] Ugowe OJ, Ugowe OO, Afeniforo OG, Adejuyigbe EA. Serum Magnesium Levels of Term Neonates with Birth Asphyxia and Relationship to Severity of Asphyxia. Nigerian Postgraduate Medical Journal. 2023 Apr; 30(2): 161-6. doi: 10.4103/npmj.npmj_1_23.

[17] Khare AK, Singh K, Paliwal A, Sharma R. How serum magnesium level is related to severity of asphyxia. International Journal of Reproduction, Contraception, Obstetrics and Gynecology. 2024 Jul; 13(7): 1714. doi: 10.18203/2320-1770.ijrcog20241764.

[18] Baştuğ O, İnan DB, Özdemir A, Çelik B, Baştuğ F, Karakükcü Ç. Tubular calcium, magnesium, and

DOI: https://doi.org/10.54393/pjhs.v6i6.3049

phosphate excretion during therapeutic hypothermia for neonatal hypoxic-ischemic encephalopathy: A prospective study. Archives de Pédiatrie. 2021 Nov; 28(8): 647-51. doi: 10.1016/j.arcped.2021.09.023.

[19] McIntosh TK, Vink R, Yamakami I, Faden AI. Magnesium protects against neurological deficit after brain injury. Brain Research. 1989 Mar; 482(2): 252-60. doi: 10.1016/0006-8993(89)91188-8.

[20] Iqbal N, Younus J, Malik M, Fatima B, Imran A, Maqbool S *et al.* The neuroprotective efficacy of postnatal magnesium sulfate in term or near-term infants with moderate-to-severe birth asphyxia. Cureus. 2021 Aug; 13(8). doi: 10.7759/cureus.16826.

[21] Shepherd E, Karim T, McIntyre S, Goldsmith S, Keir A, Badawi N *et al.* Neonatal magnesium sulphate for neuroprotection: A systematic review and meta-analysis. Developmental Medicine & Child Neurology. 2024 Sep; 66(9): 1157-72. doi: 10.1111/dmcn.15899.

[22] Bhoye SV, Bhongale ND, Thorat RM, Wade PA. Biochemical profile of term neonates with perinatal asphyxia and their correlation with severity of asphyxia. Journal of Family Medicine and Primary Care. 2024 Nov; 13(11): 5108-12. doi: 10.4103/jfmpc.jfmpc_676_24.

[23] Stofberg JP, Spittal GW, Hinkel T, Ras T. A descriptive study of suspected perinatal asphyxia at Mitchells Plain District Hospital: A case series. South African Family Practice. 2020 Oct; 62(4). doi: 10.4102/safp.v62i1.5112.