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A STUDY ON INCIDENCE, RISK FACTORS AND SHORT-TERM OUTCOMES OF BIRTH ASPHYXIA IN NEONATES

Paediatrics

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ABSTRACT

Introduction: Birth asphyxia is a leading cause of neonatal morbidity and mortality, particularly in developing countries. It can result in significant neurological and systemic complications, contributing to long-term disabilities in survivors. This study aims to explore the incidence, short-term outcomes, and risk factors associated with birth asphyxia in neonates admitted to a tertiary care center. **Objective:** To estimate the incidence of birth asphyxia and assess its associated risk factors and short-term outcomes in neonates. **Methods:** A prospective observational cohort study was conducted on term neonates admitted to the Neonatal Intensive Care Unit (NICU) at Ballari Medical College and Research Centre, Karnataka. The study included 110 neonates divided into two groups: 55 with asphyxia (cases) and 55 without (controls). Data were collected on maternal, natal, and neonatal factors, and outcomes were assessed up to six months of age. Statistical analysis was performed using SPSS version 22. **Results:** The incidence of birth asphyxia was 8.6%. Significant risk factors identified included prolonged labor, meconium-stained amniotic fluid, and maternal conditions such as pregnancy-induced hypertension. Neonates with birth asphyxia had significantly lower Apgar scores, higher oxygen support requirements, and increased incidence of seizures. At six months, 14.5% of asphyxiated neonates showed developmental delays, and the mortality rate was significantly higher in the asphyxiated group (12.7%). **Conclusion:** Birth asphyxia remains a significant neonatal health issue with severe short-term outcomes. Early identification and management of risk factors are crucial to improving neonatal outcomes and reducing the burden of long-term disabilities associated with asphyxia.

KEYWORDS

Birth asphyxia, neonatal outcomes, risk factors, hypoxic-ischemic encephalopathy, developmental delay, neonatal mortality.

INTRODUCTION

Birth asphyxia is a leading cause of neonatal morbidity and mortality. Perinatal asphyxia occurs when there is an interruption in blood flow or gas exchange between the fetus and the mother during the perinatal period (1). The World Health Organization (WHO) defines birth asphyxia as the inability of a newborn to initiate and sustain breathing immediately after birth (2). According to WHO estimates, approximately 3% of the 120 million babies born annually in developing countries experience birth asphyxia and require resuscitation efforts. Tragically, about 900,000 of these newborns die due to complications related to asphyxia. In addition to its high mortality rate, perinatal asphyxia can lead to severe neurological conditions, such as cerebral palsy, intellectual disabilities, and epilepsy, which can have long-lasting effects on the affected individuals and their families (3).

Perinatal asphyxia is a significant contributor to neonatal and under-five mortality rates, particularly in developing countries. Globally, it accounts for 9.4% of all deaths in children under five. Alongside prematurity and systemic infections, perinatal asphyxia is among the three most common causes of neonatal deaths (4). According to the National Neonatal Perinatal Database (NNPD) from 2002-2003, perinatal asphyxia is the leading cause of neonatal death in intramural live births, responsible for 28.8% of these deaths. Furthermore, asphyxia is the most prevalent cause of stillbirths, accounting for 45.1% of all cases. The incidence of perinatal asphyxia is observed in 8.4% of all live births (5).

Perinatal asphyxia is a multi-organ disorder that impacts nearly every system in the body, including the brain, heart, lungs, kidneys, and intestines. In term neonates suffering from asphyxia, dysfunction is observed in several critical systems: 50% experience renal dysfunction, 28% have central nervous system (CNS) issues, and both cardiac and lung dysfunctions occur in 25% of cases (6). This wide-ranging impact highlights the severity of perinatal asphyxia and underscores the urgent need for effective prevention and management strategies to reduce its toll on newborns.

Many complications resulting from perinatal asphyxia can be life-threatening, and the extent of organ system involvement largely determines the prognosis for an affected neonate. Therefore, providing appropriate medical support is crucial to facilitate recovery. Hypoxic-ischemic encephalopathy (HIE) is a form of central nervous system (CNS) dysfunction associated with asphyxia, which not only poses a high risk of mortality but also significantly increases the likelihood of severe long-term neuromotor complications among survivors.

For neonates who survive severe HIE, potential outcomes include mental retardation, epilepsy, and cerebral palsy. The risk of long-term complications is closely linked to the severity of HIE. Up to 80% of neonates with stage-3 HIE may die, while the remaining 20% often experience neurological sequelae. In cases of moderate birth asphyxia, the mortality rate can be as high as 5%. Among survivors of moderate asphyxia, 30-50% may develop long-term complications, and 10-20% may suffer from minor neurological impairments. Conversely, neonates with mild HIE generally do not experience any long-term neurological sequelae (7). These statistics highlight the need for further research on birth asphyxia to understand its impact better and to develop effective prevention and treatment strategies. Such measures are essential to reduce the burden of asphyxia and prevent the severe outcomes associated with this condition.

According to the third consensus statement by the American College of Obstetricians and Gynecologists, intra-partum asphyxia is diagnosed when the following four essential criteria are met (8): Metabolic acidosis: Defined as a pH of less than 7 and a base deficit of 12 or more in an umbilical artery blood sample. Moderate or severe encephalopathy: Indicating significant brain dysfunction. Cerebral palsy: Specifically, the spastic quadriplegia or dyskinetic type.

EXCLUSION OF OTHER CAUSES: Ruling out alternative explanations for the observed symptoms. In addition to these primary criteria, five additional criteria may support the diagnosis: a) The presence of a sentinel event (e.g., uterine rupture, cord prolapse). b) Abrupt changes in fetal heart rate patterns. c) An APGAR score of 3 or less persisting beyond 5 minutes after birth. d) Evidence of multi-system failure within the first 72 hours of life. e) Early imaging findings suggestive of brain injury.

The incidence of perinatal asphyxia in developed countries ranges from 1% to 1.5% of live births and is inversely related to both gestational age and birth weight. It occurs in approximately 0.5% of live births at or beyond 36 weeks of gestation and is responsible for 20% of perinatal deaths (rising to 50% when stillbirths are included). Higher rates of perinatal asphyxia are observed in newborns of mothers with diabetes or preeclampsia, those with intrauterine growth restriction, breech presentations, and those born post-term (9). In 2015, out of 5.9 million deaths in children under five, 2.7 million occurred during the neonatal period. Events related to intrapartum complications accounted for 0.691 million of these deaths,

representing 10.7% of all neonatal deaths (10).

Pathophysiologically, asphyxia is characterized by a combination of hypoxia (lack of oxygen) and hypoperfusion (reduced blood flow), which disrupts tissue gas exchange and leads to tissue lactic acidosis. In term newborns, asphyxia can occur during the antepartum or intrapartum periods due to impaired gas exchange across the placenta, resulting in inadequate oxygen delivery and failure to remove carbon dioxide (CO2) and hydrogen ions (H+) from the fetus. Asphyxia can also develop in the postpartum period, typically due to pulmonary, cardiovascular, or neurological abnormalities.

The causes of hypoxia-ischemia in neonates are diverse and can be categorized into several groups: maternal factors such as hypertension (both acute and chronic), hypotension, infections (including chorioamnionitis), hypoxia due to pulmonary or cardiac disorders, diabetes, maternal vascular disease, in utero exposure to cocaine, and neurologic disorders; placental factors including abnormal placentation, abruption, infarction, and fibrosis; uterine rupture; umbilical cord accidents like prolapse, entanglement, true knots, and compression; abnormalities of the umbilical vessels; fetal factors such as anemia, infection, cardiomyopathy, hydrops, and severe cardiac or circulatory insufficiency; and neonatal factors like cyanotic congenital heart disease, persistent pulmonary hypertension of the newborn (PPHN), cardiomyopathy, and other forms of neonatal cardiogenic or septic shock. In response to a hypoxic-ischemic event, the body initiates reflex mechanisms to redistribute cardiac output, increasing blood flow to vital organs such as the brain, heart, and adrenal glands (known as the diving reflex). However, in cases of severe but brief asphyxia, this protective blood flow redistribution may not occur, leading to specific patterns of injury to the subcortical and brainstem nuclei. With prolonged asphyxia, there can be a loss of autoregulation of blood pressure and CO2 vasoreactivity, potentially causing further disruptions in cerebral perfusion, especially if accompanied by cardiovascular compromise such as hypotension or reduced cardiac output.

A decrease in cerebral blood flow during an asphyxic event leads to a shift from aerobic to anaerobic metabolism in the brain. This metabolic shift results in inefficient energy production and an increased consumption of glucose, causing a rapid depletion of essential energy stores such as glycogen, phosphocreatine, and adenosine triphosphate (ATP). The lack of sufficient ATP disrupts cellular functions, ultimately leading to cellular energy failure and potential cell death. Prolonged periods of asphyxia can cause widespread damage to both cortical and subcortical brain structures, affecting areas responsible for critical neurological functions (9). Birth asphyxia is not only a leading cause of neonatal mortality but is also linked to significant long-term neurodevelopmental impairments, such as cognitive deficits, cerebral palsy, and epilepsy. These neurological sequelae profoundly impact the quality of life for survivors and impose a heavy burden on families and healthcare systems. Therefore, this study is crucial in highlighting the outcomes, incidence, and risk factors associated with birth asphyxia. Understanding these factors is essential for assessing the overall burden of the condition and developing and implementing effective preventive and management strategies to improve neonatal outcomes and reduce the risk of long-term disabilities.

MATERIAL AND METHODS

This study observational, prospective cohort study focuses on term newborns with asphyxia admitted to the Neonatal Intensive Care Unit (NICU) at the Department of Pediatrics, Ballari Medical College and Research Centre, Ballari, Karnataka, during the designated study period. An ethical approval has been obtained from the Neonatal Intensive Care Unit (NICU) at the Department of Pediatrics, Ballari Medical College and Research Centre, Ballari, Karnataka.

STUDY POPULATION

The study consists of a total sample size of 110 neonates, divided into two groups: 55 in the exposed group (asphyxiated cases) and 55 in the control group (non-asphyxiated).

INCLUSION CRITERIA : for the study are neonates admitted to the NICU at Ballari Medical College and Research Centre, Ballari, who meet any of the following conditions: a history of fetal distress as reported by the obstetrician, signs of birth asphyxia such as delayed cry, limpness, generalized cyanosis, apnea lasting more than one minute, or an APGAR score of ≤7 at 5 minutes, a need for resuscitative measures at birth, or evidence of neurological depression during their NICU stay.

EXCLUSION CRITERIA: include preterm neonates born before 37 weeks of gestation, those weighing less than 2000 grams, neonates with congenital malformations, and babies born outside the facility.

DATA ANALYSIS

Data were entered into Microsoft Excel and analyzed using SPSS version 22 and Epi-info version 7.2.1 (CDC Atlanta). Categorical data were presented as frequencies and proportions, and the Chi-square test was used for qualitative data analysis. Continuous data were expressed as mean and standard deviation, with normality tested using the Kolmogorov–Smirnov and Shapiro–Wilk tests. The independent t-test was applied to assess the mean difference between two quantitative variables, and graphical representations were created using MS Excel and MS Word. A p-value of less than 0.05 was considered statistically significant.

RESULTS

In one year, there were 7,205 total live births, with 620 neonates experiencing asphyxia, resulting in a birth asphyxia incidence of 8.6%. In terms of gender distribution, 38.2% of the cases and 45.5% of the controls were female, while 61.8% of the cases and 54.5% of the controls were male, with a Pearson Chi-Square p-value of 0.439. Gestational age distribution showed that 85.5% of the cases and 94.5% of the controls were between 37 to 40 weeks, with 14.5% of cases and 5.5% of controls beyond 40 weeks; the p-value was 0.112, indicating no significant difference. The maternal age was also similar between groups, with mean ages of 23.60 ± 3.375 years for Cases and 24.09 ± 4.070 years for Controls (p = 0.493).

Table 1: Antenatal factors distribution comparison between two groups.

		Group				P value
		Cases		Controls		
		Count	%	Count	%	
Antenatal	Nil	34	61.8%	41	74.5%	0.250
	Anemia	1	1.8%	2	3.6%	
	Cardiac disease	0	0.0%	1	1.8%	
	HBsAg	2	3.6%	0	0.0%	
	Hypothyroidism, Twin gestation	0	0.0%	1	1.8%	
	Hypothyroid	2	3.6%	0	0.0%	
	Oligohydramnios	3	5.5%	2	3.6%	
	PE	1	1.8%	0	0.0%	
	PIH	10	18.2%	5	9.1%	
	PIH, VPCs	0	0.0%	1	1.8%	
	PIH, Hypothyroid	0	0.0%	2	3.6%	
	Polyhydramnios	1	1.8%	0	0.0%	
	Short stature	1	1.8%	0	0.0%	

The distribution of antenatal conditions showed no significant difference between the groups (p = 0.250). Among the Cases, 61.8% had no antenatal conditions, compared to 74.5% in the Controls. Conditions like anemia, hypothyroidism, and other antenatal complications were similarly distributed across both groups.

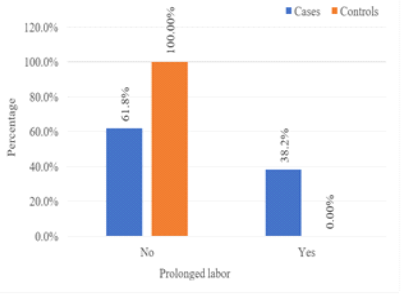

Figure 1: Prolonged labor distribution comparison between two groups Prolonged labor was significantly more frequent in the Cases group compared to the Controls (p < 0.001). Among the Cases, 38.2% experienced prolonged labor, while none of the Controls did. This demonstrates a strong association between prolonged labor and the Cases group.

Table 2: Natal History distribution comparison between two groups.

		Group				P value
		Cases		Controls		
		Count	%	Count	%	
Natal History	Breech	3	5.5%	0	0.0%	<0.001*
	Cord around neck	2	3.6%	0	0.0%	
	CPD	1	1.8%	0	0.0%	
	Deep transverse arrest	3	5.5%	0	0.0%	
	Difficult extraction	1	1.8%	0	0.0%	
	Fetal distress	11	20.0%	0	0.0%	
	Not significant	34	61.8%	55	100.0%	

A significant difference in natal history was observed between the groups ($p < 0.001$). Among the Cases, 38.2% experienced complications such as breech presentation, cord around the neck, or fetal distress, while none of the Controls had these issues. This indicates a strong association between natal complications and the Cases group.

Table 3: Distribution APGAR score, Oxygen support and MSAF comparison between two groups

		Group				P value
		Cases		Controls		
		Count	%	Count	%	
APGAR1	0-3	17	30.9%	0	0.0%	<0.001*
	4-6	36	65.5%	32	58.2%	
	7-10	2	3.6%	23	41.8%	
APGAR5	0-3	4	7.3%	0	0.0%	<0.001*
	4-6	23	41.8%	0	0.0%	
	7-10	28	50.9%	55	100.0%	
		Cases		Controls		
		Count	%	Count	%	
Oxygen Support	Nil	0	0.0%	55	100.0%	<0.001*
	BMV	35	63.6%	0	0.0%	
	MV	12	21.8%	0	0.0%	
	TS-O2	8	14.5%	0	0.0%	
		Count	%	Count	%	
MSAF	No	32	58.2 %	50	90.9 %	<0.001*
	Yes	23	41.8 %	5	9.1%	

Oxygen support requirement was significantly higher in the Cases group compared to the Controls ($p < 0.001$). None of the Controls required oxygen support, whereas 63.6% of Cases required BMV, 21.8% required MV, and 14.5% required TS-O2. This indicates a significant association between oxygen support requirement and the Cases group. The presence of meconium-stained amniotic fluid (MSAF) was significantly higher in the Cases group compared to the Controls ($p < 0.001$). In the Cases, 41.8% had MSAF, while only 9.1% of the Controls did. This suggests a significant association between MSAF and the Cases group.

Table 4: The distribution of HIE stage

		Group Cases	
		Count	%
HIE-Stage	1	24	43.6%
	2	20	36.4%
	3	11	20.0%

The distribution of Hypoxic-Ischemic Encephalopathy (HIE) stages among the Cases was as follows: 43.6% were in Stage 1, 36.4% in Stage 2, and 20.0% in Stage 3. This shows the severity distribution of HIE within the Cases group.

Table 5: Seizures and Outcome distribution comparison between two groups

		Group				P value
		Cases		Controls		
		Count	%	Count	%	
Seizures	No	30	54.5%	55	100.0%	<0.001*
	Yes	25	45.5%	0	0.0%	

Outcome	Death	6	10.9%	1	1.8%	0.051
	Discharged	49	89.1%	54	98.2%	

Seizures were significantly more frequent in the Cases group than in the Controls ($p < 0.001$), with 45.5% of Cases experiencing seizures compared to none in the Controls, indicating a strong association between seizures and the Cases group. The outcome distribution, however, showed no significant difference between the groups ($p = 0.051$). In the Cases, 10.9% resulted in death and 89.1% were discharged, while in the Controls, 1.8% resulted in death and 98.2% were discharged. This suggests a trend toward higher mortality in the Cases group, though it is not statistically significant.

Table 6: Follow-up at 6 months distribution comparison between two groups.

		Group				Pvalue
		Cases		Controls		
		Count	%	Count	%	
Follow-up at 6 months	Death	7	12.7%	1	1.8%	0.001*
	Developmental delay	8	14.5%	0	0.0%	
	Nil	40	72.7%	54	98.2%	

Follow-up outcomes at 6 months showed significant differences between the groups ($p = 0.001$), with 12.7% of Cases resulting in death, 14.5% experiencing developmental delays, and 72.7% having no issues, compared to 1.8% death and 98.2% with no issues in the Controls. This indicates a significant disparity in outcomes between the groups.

DISCUSSION

A prospective observational cohort study was done to estimate the incidence and to determine the short-term outcomes and risk factors of asphyxiated neonates admitted to NICU of a tertiary care hospital and medical college, Ballari Medical College and Research Centre and followed up till 6 months of age. Perinatal asphyxia remains a major cause of morbidity and mortality in neonates.

In a study by Sunny et al., male neonates were found to have a higher risk of birth asphyxia compared to females. However, in this study, the gender distribution between the exposed and control groups was similar, rendering the difference statistically insignificant, which aligns with findings from Yadav N et al., where the gender ratio was 1:1, and Khuntdar et al., where the control group consisted of 60% males and 40% females, and the case group had 51% males and 49% females. Despite this, several studies, including those by Badawi et al. and Futrakul et al., have reported a male predominance in birth asphyxia cases. Regarding gestational age, 85.5% of cases in this study were between 37-40 weeks, compared to 94.55% of controls, while 14.5% of cases were beyond 40 weeks, with only 5.5% of controls falling into this category. This finding contrasts with Mir NA et al., who identified postmaturity as a significant risk factor for a higher incidence of asphyxia. Conversely, Mac Donald et al. found prematurity to be the most significant predictor of asphyxia, particularly in neonates born before 27 weeks of gestation, a finding supported by Lee et al., who reported a 2.28-fold higher risk of asphyxia in preterm births, and Aslam et al. However, this study focused exclusively on term neonates, so the impact of prematurity as a risk factor for birth asphyxia could not be assessed [10-16].

In this study, the mean age of mothers in the case group was 23.60 years, while in the control group, it was 24.09 years, indicating a similar maternal age distribution between the two groups, comparable to a study by Yadav N et al., where the mean maternal age was 24.28 years. There was no significant difference in parity distribution between cases and controls, with 63.6% of cases born to multigravida mothers and 36.4% to primigravida, aligning with the Chiabi et al. study, which reported 67.8% multigravida and 32.2% primigravida. Conversely, Yadav et al. found that 51% of cases were born to primigravida mothers, similar to the Aslam et al. study, where 56.9% of cases were primigravida.Maternal diseases were similarly distributed between the case and control groups, but conditions like pregnancy-induced hypertension (PIH) were found in 18.2% of cases versus 9% in controls, and oligohydramnios (5.5%) and hypothyroidism (3.6%) were slightly more prevalent in the cases group. These findings are consistent with studies by Yadav N et al. and Mohan et al., which identified PIH and oligohydramnios as common maternal risk factors, and with the Aslam et al. study, where oligohydramnios was present in 7.3% of cases, and the Babu et al. study, where PIH was a statistically significant risk factor (18.1% in cases vs. 4% in controls) [11, 16-18].

In this study, prolonged labor was significantly more common in the case group (38.2%) than in the control group (0%), which aligns with findings from Babu et al. (37.4% vs. 4%), Chiabi et al., Mohan et al.,

and Meshesha et al., reflecting the higher risk of fetal distress and subsequent asphyxia in cases of prolonged labor. A significant difference was also noted in natal history between the groups, with 38.2% of cases having natal risk factors such as malpresentation, nuchal cord, deep transverse arrest, and fetal distress, while none of these factors were present in the control group. This finding is consistent with Yadav et al., where common fetal risk factors included intrauterine growth restriction (IUGR), fetal distress, meconium aspiration syndrome (MAS), and malpresentations, and Mohan et al., who reported fetal distress in 58.33% of neonates. The presence of meconium-stained amniotic fluid (MSAF) was significantly higher in the case group (41.8%) compared to the control group (9.1%), suggesting a strong association between MSAF and birth asphyxia, similar to the Yadav N et al. study, where MAS was present in 12.5% of cases. Although the mode of delivery did not significantly differ between groups, instrumental vaginal delivery using forceps or vacuum was observed in 1.8% of the case group but not in the control group. This contrasts with findings from studies by Sunny et al., Asefa et al., and Tesfaye et al., where assisted vaginal delivery significantly increased the odds (1.87 times) of developing birth asphyxia compared to spontaneous vaginal delivery [11, 17-22].

In this study, Apgar scores at 1 and 5 minutes were significantly lower in the case group, with 30.9% scoring 0-3 at 1 minute and 7.3% at 5 minutes, compared to 0% in the control group for both time points. All controls had Apgar scores of ≥7 at 5 minutes, unlike in the Shah G et al. study, where 80% of cases scored 4-6 and 20% scored ≤3. Oxygen and ventilatory support were required by 63.6% of cases for bag and mask ventilation, 21.8% for mechanical ventilation, and 14.5% for oxygen support, while no controls needed any support. Comorbidities such as meconium aspiration syndrome (MAS) were significantly more common in cases (34.5% vs. 5.5% in controls), and MAS was identified as a major risk factor for birth asphyxia in studies by Yadav N et al., Mehta V et al., and Nadeem et al. [11, 23-25]

In this study, the distribution of hypoxic-ischemic encephalopathy (HIE) stages among the cases was 43.6% in HIE-I, 36.4% in HIE-II, and 20% in HIE-III, whereas Rathi Y et al. found 12.5% in HIE-I, 58.3% in HIE-II, and 29.1% in HIE-III. Seizures were significantly more common in cases (45.5%) compared to controls (0%); none of the HIE-I cases had seizures, while all HIE-II and 45.5% of HIE-III cases did. Natal factors, Apgar scores, and the need for ventilatory support showed significant associations with HIE stages. Mortality was higher in the exposed group (10.9%) compared to controls (1.8%), consistent with other studies. The outcome at discharge was significantly associated with HIE stage, with 100% of HIE-I cases discharged, while 5% of HIE-II and 45.5% of HIE-III cases died. At 6 months follow-up, 12.7% of the exposed group had died, 14.5% had developmental delays, and 72.7% were normal, compared to 98.2% normalcy in the control group. These findings align with Finer et al., where higher mortality and developmental delays were observed in asphyxiated neonates compared to controls [26].

The 6-month outcomes were significantly associated with the stage of HIE, with HIE-3 cases showing the highest mortality (45.5%) and developmental delay (54.5%), compared to 5% mortality and 10% developmental delay in HIE-2 cases, while HIE-1 cases had no developmental delays. Mortality was also higher in neonates born to primigravida mothers (85.7%) compared to multigravida (14.3%), and in cases with maternal risk factors like oligohydramnios, pre-eclampsia, PIH, and HBsAg positive status. Apgar scores at 5 minutes were significantly associated with outcomes, as 85.7% of those who died had scores of ≤7, highlighting its prognostic value. Neonates requiring mechanical ventilation had significantly higher mortality (85.7%), indicating a poor prognosis for those with severe asphyxia, as supported by studies like Giannakis et al. Additionally, 85.7% of those who died had meconium-stained amniotic fluid (MSAF), suggesting its role as a risk factor for mortality and a marker of severe asphyxia and intrauterine distress. Cases with comorbidities such as MAS, MODS, pneumothorax, and LONS had significantly worse outcomes, with 57.2% of deaths associated with MAS [27].

This study found a significant association between seizures and poor outcomes in neonates, with 57.1% of those who died experiencing seizures compared to 20.4% among those discharged, mirroring results from Finer et al., Mulligan et al., and MacDonald et al. Additionally, mortality was significantly linked to the stage of HIE; 83.3% of deaths occurred in HIE-3 cases and 16.7% in HIE-2, with none in HIE-1, consistent with findings from Panthee et al. and Yelamali et al., demonstrating that HIE-3 is associated with high mortality rates [14, 26-30].

CONCLUSION

In conclusion, this study highlights that birth asphyxia remains a significant concern, with its outcomes strongly associated with various factors, including the stage of hypoxic-ischemic encephalopathy (HIE), presence of seizures, and need for mechanical ventilation. Neonates in the HIE-3 stage showed the highest mortality and developmental delays, underlining the severity of this condition. The occurrence of seizures and low Apgar scores at 5 minutes were also critical indicators of poor prognosis. Moreover, maternal factors such as primigravida status and conditions like oligohydramnios and pregnancy-induced hypertension (PIH) were associated with higher mortality. These findings emphasize the need for early identification and management of at-risk neonates to improve outcomes and reduce the burden of birth asphyxia.

REFERENCES:

1. Volpe JJ. Neurology of the Newborn. Philadelphia: Saunders, 2001
2. World Health Organization. Maternal and Newborn Health/Safe Motherhood Unit, Division of Reproductive Health. Basic Newborn Resuscitation: A Practical Guide. Geneva: World Health Organization; 1999:10-18.
3. Padayachee N, Ballot DE. Outcomes of neonates with perinatal asphyxia at a tertiary academic hospital in Johannesburg, South Africa. S Afr J Child Health. 2013;7(3):89-94
4. Liu L, Johnson HL, Cousens S, Perin J, Scott S, Lawn JE, Rudan I, Campbell H, Cibulskis R, Li M, Mathers C, Black RE; Child Health Epidemiology Reference Group of WHO and UNICEF. Global, regional, and national causes of child mortality: an updated systematic analysis for 2010 with time trends since 2000. Lancet. 2012 Jun 9;379(9832):2151-61
5. Report of the National Neonatal Perinatal Database (National Neonatology Forum, India)2003
6. Perlman JM, Tack ED, Martin T, Shackelford G, Amon E. Acute systemic organ injury in term infants after asphyxia. Am J Dis Child. 1989 May;143(5):617-20.
7. Robertson C, Finer N. Term infants with hypoxic-ischemic encephalopathy: outcome at 3.5 years. Dev Med Child Neurol. 1985 Aug;27(4):473-84.
8. Task Force American College of Obstetricians and Gynecologists and The American Academy of Pediatrics. Neonatal encephalopathy and CP. Defining the pathogenesis and pathophysiology. Washington DC: ACOG, 2003.
9. Arora S, Evan YS. Manual of neonatal care. In John P Cloherty, Eric C, Eichenwald and Ann R Stark (Ed.) 7th Edition, Lippincott Williams & Wilkins, 2012, pp. 536- 555.
10. Sunny, A.K., Paudel, P., Tiwari, J. et al. A multicenter study of incidence, risk factors and outcomes of babies with birth asphyxia in Nepal. BMC Pediatr 21, 394 (2021).
11. Yadav N, Damke S. Study of risk factors in children with birth asphyxia. Int J Contemp Pediatr 2017;4:518-26
12. Khuntdar BK, Roy AK, Das RK, Pattanayak U, Madhwani KP. A clinicoepidemiological profile of neonates with birth asphyxia in SNCU and NICU of a tertiary care hospital with special reference to renal parameters and serum calcium levels in birth asphyxia. Int J Reprod Contracept Obstet Gynecol 2023;12:2212-7
13. Badawi N, Kurinczuk JJ, Keogh JM, Alessandri LM, O'Sullivan F, Burton PR, et al Antepartum risk factors for newborn encephalopathy: The Western Australian case-control study BMJ. 1998;317:1549–53
14. MacDonald HM, Mulligan JC, Allen AC, Taylor PM. Neonatal asphyxia. I. Relationship of obstetric and neonatal complications to neonatal mortality in 38,405 consecutive deliveries. J Pediatr. 1980 May;96(5):898-902.
15. Mir NA, Faquih AM, Legnain M. Perinatal risk factors in birth asphyxia: relationship of obstetric and neonatal complications to neonatal mortality in 16,365 consecutive live births. Asia Oceania J Obstet Gynaecol. 1989 Dec;15(4):351-7.
16. Aslam HM, Saleem S, Afzal R, Iqbal U, Saleem SM, Shaikh MW, et al. Risk factors of birth asphyxia. Ital J Pediatr. 2014;40(1):94
17. Chiabi A, Nguefack S, Mah E, Nodem S, Mbuagbaw L, Mbonda E, et al. Risk factors for birth asphyxia in an Urban Health Facility in Cameroon. Iran J Child Neurol. 2013;7(3):46-54.
18. Mohan K, Mishra PC, Singh DK. Clinical profile of birth asphyxia in newborn. Int J Sci Tech. 2013;3(1):10-9
19. Babu BVA, Devi SS, Kumar BK. "Birth asphyxia- Incidence and immediate outcome in relation to risk factors and complications". Int J Res Health Sci [Internet]. 2014 Oct 31;2(4):1064-71.
20. Meshesha AD, Azage M, Worku E, Bogale GG. Determinants of Birth Asphyxia Among Newborns in Referral Hospitals of Amhara National Regional State, Ethiopia. Pediatric Health Med Ther. 2020 Jan 7;11:1-12
21. Asefa, Negese & Legesse, Tadesse & Tejineh, Solomon. (2023). Glob J Reprod Med Magnitude and Determinants of Birth Asphyxia: Unmatched Case Control Study Assela Referral Teaching Hospital, Arsi Zone, Ethiopia.10.19080/GJORM.2019.07.555705
22. Tesfaye Tunta, Tadele Dana, Abiyot Wolie, Temesgen Lera,Determinants of birth asphyxia among neonates admitted to neonatal intensive care units in hospitals of the Wolaita zone, Southern Ethiopia: A case-control study,Heliyon,Volume 10, Issue 1,2024,e23856,ISSN 2405-8440
23. Shah P, Riphagen S, Beyene J, Perlman M. Multiorgan dysfunction in infants with post-asphyxial hypoxic-ischaemic encephalopathy. Arch Dis Child Fetal Neonatal Ed. 2004 Mar;89(2):F152-5.
24. Mehta V, Adarsh E, Spoorthi,Archana, Hassan M. Meconium-stained amniotic fluid as a risk factor for perinatal asphyxia. Int J Contemp Pediatr 2019;6:325-8.
25. Nadeem G, Rehman A, Bashir H (October 13, 2021) Risk Factors Associated With Birth Asphyxia in Term Newborns at a Tertiary Care Hospital of Multan, Pakistan. Cureus 13(10): e18759.
26. Finer NN, Robertson CM, Richards RT, Pinnell LE, Peters KL. Hypoxic-ischemic encephalopathy in term neonates: perinatal factors and outcome. J Pediatr. 1981 Jan;98(1):112-7.
27. Giannakis S, Ruhfus M, Markus M, Stein A, Hoehn T, Felderhoff-Mueser U, SabirH. Mechanical Ventilation, Partial Pressure of Carbon Dioxide, Increased Fraction of Inspired Oxygen and the Increased Risk for Adverse Short-Term Outcomes in Cooled Asphyxiated Newborns. Children (Basel). 2021 May 21;8(6):430.
28. Mulligan JC, Painter MJ, O'Donoghue PA, MacDonald HM, Allan AC, Taylor PM. Neonatal asphyxia. II. Neonatal mortality and long-term sequelae. J Pediatr. 1980 May;96(5):903-7.

29. Panthee K, Sharma K, Kalakheti B, Thapa K. Clinical profile and outcome of asphyxiated newborn in a Medical College Teaching Hospital. Journal of Lumbini Medical College. 2016;4(1):1-3.
30. Yelamali, B & Pol, Ramesh & Talawar, K & Naik, Sanjay & Badakali, Ashok. (2020). Outcome of newborn with birth asphyxia in tertiary care hospital -a retrospective study. 3. 78-83.