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Original Research Article

Moderate and severe birth asphyxia in term neonates: early and late outcomes

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ABSTRACT

Background: Aim was to compare the morbidities, laboratory parameters, immediate outcomes, and growth and development in term neonates with moderate and severe birth asphyxia.
Methods: This prospective cohort study was carried out over a duration of 18 months. The neonates fulfilling the inclusion criteria (n=84) were enrolled, and were followed up till 6 months. Their growth was assessed and neuromotor assessment was done using Amiel-Tison angles at the time of discharge, 3 and 6 months of age and neurodevelopmental screening was done using Trivandrum Developmental Screening Chart (TDSC) at 3rd and 6th month and Developmental Assessment Scale for Indian Infants (DASII) as confirmatory test at 6 months.
Results: Incidence of perinatal asphyxia was 17.6 per 1000 live births in our study. Of the cohort of 84 term intramural neonates 69.1% of the babies had moderate birth asphyxia and 30.9% had severe birth asphyxia. 34% infants with moderate birth asphyxia had respiratory failure whereas all babies (100%) with severe birth asphyxia suffered respiratory failure. Mean levels of LDH and lactate collected within 6 hours, were significantly higher in severely asphyxiated neonates ($p<0.05$ and $p<0.001$ respectively). At 3 months, 25 (96.2%) babies with severe asphyxia failed TDSC, 23 (88.5%) had post discharge seizures and 24 (92.3%) had tone abnormalities. Developmental delay assessed by DASII at 6-month follow-up was statistically significantly higher in severely asphyxiated babies ($p<0.001$).
Conclusions: Incidence of comorbidities is higher in severely asphyxiated babies in comparison to moderately asphyxiated neonates. Laboratory markers like lactate and LDH at birth can be used as predictors of severity of birth asphyxia. Growth, development can be affected and picked up as early as 3 months in these babies on close follow up.

Keywords: Birth asphyxia, Neurodevelopmental outcome, Amiel-Tison angles, TDSC, DASII

INTRODUCTION

Perinatal asphyxia is defined as the inability of the newborn to initiate and sustain enough respiration after delivery and is characterized by a marked impairment of gas exchange.[1] Perinatal asphyxia is one of the predominant causes of neonatal mortality third only to sepsis and prematurity.[2] The incidence of asphyxia is 1-6 per 1000 births in developed countries and 5-10 per 1000 births in developing countries.[3] According to World Health Organization (WHO), in developing countries 3% of infants (3.6 million people) suffer from moderate to severe asphyxia, of whom 23% (840,000) die, and almost the same number suffer from the associated consequences.[4] According to the National Neonatal Perinatal Database (NNPD) 2002-2003 network report, the incidence of birth asphyxia is 1.4%.[5]

Although the majority of these disorders are transient, the long-term consequences of asphyxia may affect the central nervous system (CNS), which can ultimately lead to major neurological sequel like cerebral palsy, mental retardation, epilepsy, visual and auditory impairment or mild motor deficits in later life or subtle neurological abnormalities.[4,6] Ideally all high-risk babies need close surveillance throughout infancy. Severity of birth

asphyxia has an impact on morbidities, growth and development of neonates.[7-9] This study aimed at finding out the short term and long-term outcome with respect to severity of birth asphyxia in term neonates.

METHODS

This study was carried out at neonatal intensive care unit of Department of Pediatrics at SMIMER medical college and hospital, Surat, Gujarat, India. It was a prospective cohort study carried out over period of 18 months (May 2021 through October 2022). This study was approved by institutional ethics committee. The admitted neonates who fulfilled the inclusion criteria were enrolled. All the babies with major congenital malformations, inborn errors of metabolism, those for whom the consent was not given, those who expired or who were lost to follow up during the study period were excluded from the study. We included those term babies who had an APGAR score 6 or less at 1 minute as per the NNPD network report. Further based on the Apgar score they were classified as moderate (APGAR score of 4-6 at 1 minute) and severe birth asphyxia (APGAR score of 0-3 at 1 minute of age).[5] After taking the informed written consent from the parent or guardian, the relevant information regarding pregnancy, delivery and neonatal period were collected from the parents and neonatal records and entered in a predesigned proforma.

The neonates were treated as per routine NICU protocol for fluid management, nutrition, thermal control, seizures, electrolyte imbalance and other complications of birth asphyxia. Clinical assessment, laboratory investigations, detailed neurological examination and HIE staging using Sarnat and Sarnat staging system was done during the NICU stay.[10] On discharge babies were assessed for abnormal neurological signs (which included tone abnormalities and abnormal reflexes), neuromotor assessment by Amiel-Tison neurological assessment at term (ATNAT) and anthropometry (weight, length and head circumference).[11] All the infants were followed up on an outpatient basis and neuromotor assessment was done using Amiel-Tison angles at 3 and 6 months of age with anthropometry using standard instruments and charted on WHO child growth standard charts. Neurodevelopmental screening was done using TDSC at 3 and 6 months of age. DASII was used as confirmatory test at the end of 6 months of age (performed under a trained supervisor). Outcome was considered abnormal if the development quotient was ≤70% in either motor or mental scale.

Statistical analysis

Data was entered into MS excel database and then analysed using SPSS software (version 20.0). Categorical variables were described as frequencies and percentages. Continuous variables were described as mean and standard deviation. Proportions were compared between groups using chi-square test or Fischer Exact analysis or t-test for independent samples, whichever applicable. ANOVA test was used to compare the mean values of quantitative data for more than 3 independent groups. A $p<0.05$ was considered significant. Logistic regression was applied to find out significant variable for neurodevelopmental delay.

RESULTS

This study was done over a duration of 18 months from May 2021 to October 2022 in the NICU of a tertiary care hospital. During this period 96 neonates were eligible, as per inclusion criteria with moderate and severe birth asphyxia. 8 neonates expired and 4 were lost to follow up. They were excluded from analysis. Analysis of 84 babies is being presented here.

Total deliveries during this study period were 6021 and incidence of birth asphyxia was 17.6 per 1000 live births. We included babies with moderate or severe birth asphyxia. Of the cohort of 84 term intramural neonates with moderate and severe birth asphyxia, 69.1% of the babies had moderate birth asphyxia and 30.9% had severe birth asphyxia. Male to female ratio was 1.4:1. Significantly higher comorbidities (respiratory failure, sepsis, MAS, AKI and hypocalcaemia) were seen in the group with severe birth asphyxia than moderate birth asphyxia as shown in Table 1.

All babies with severe birth asphyxia had some grade of HIE but 5.1% of babies with moderate birth asphyxia did not develop HIE. HIE stage 3 was seen in 26.9% babies of severe birth asphyxia and this was only in 8.6% in babies with moderate birth asphyxia ($p=0.064$). Mean levels of laboratory markers LDH and lactate collected within 6 hours were significantly higher in severely asphyxiated neonates ($p<0.05$ and $p<0.001$ respectively). Mean hospital stay also was prolonged in severely asphyxiated babies as compared to moderately asphyxiated babies (14.08/8.66 days). Breastfeeding establishment was faster in babies with moderate birth asphyxia. 50 (59.52%) babies had tone abnormalities on discharge. Significantly higher number of neonates with severe birth asphyxia showed tone abnormalities on discharge as compared to moderate birth asphyxia. Of the initial 96 neonates included in the study, 8 deaths and 4 lost to follow-up were excluded from the study. Mortality was 26% in SBA versus 8.6% in MBA ($p<0.001$).

Growth failure with respect to weight for age, length for age and weight for length seen in 33.3%, 5.9% and 41.7% respectively at 3 months for entire cohort of asphyxiated babies, which increased at 6 months shown in Table 2. Incidence of microcephaly was 13.1% at 3 months and 6 months. 48.4% babies failed TDSC at 3 months and this increased to 66.6% at 6 months follow up.

Severity of asphyxia affected the growth and developmental parameters, as seen in Table 3. At 3 months follow up, growth parameters were significantly

affected in babies of severe birth asphyxia. Anthropometric parameters like weight for length, weight for age and head circumference, were significantly lower in babies with severe birth asphyxia. At 3 months, 25 (96.2%) babies with severe asphyxia failed TDSC, 23 (88.5%) had post discharge seizures and 24 (92.3%) had tone abnormalities. All were significantly higher in the severe asphyxia group, as compared to moderate birth asphyxia babies. At 6 months follow up, the length for age also got affected in severely asphyxiated babies in addition to above growth parameters. Growth delay, tone abnormalities increased in both groups at 6 months as compared to the 3 months follow up. The 53.6% babies had abnormal outcome as assessed by DASII at 6 months. Severity of birth asphyxia correlated with abnormal developmental outcome. More babies of severe birth asphyxia had developmental delay as compared to those with moderate birth asphyxia (96.2% versus 34.5% respectively) at 6 months (p<0.001).

The significant determinants of abnormal developmental outcome being mean weight at 6 months of age, severity of HIE, respiratory failure, MAS, hypocalcaemia, AKI, microcephaly at 6 months of age, weight for age and length and abnormal angles at 6 months, seen in Table 4.

Serum lactate and CPK at birth were the laboratory parameter which showed a significant correlation to abnormal developmental outcome at 6 months. Longer hospital stays and establishment of breast feeding also correlated to the abnormal developmental outcome at 6 months.

Using univariate analysis, a logistic regression was carried out on all relevant indicators for participants having abnormal developmental outcome as assessed by the DASII. According to this, infants with HIE stage 2 had a 12.22-fold higher likelihood than those with HIE stage 1 to exhibit abnormal development.

Table 1: Co-morbidities in neonates with moderate and severe birth asphyxia (n=84).

Variables	N (%)	MBA, 58 (69.04)	SBA, 26 (30.95)	P value
Neonatal morbidities, N (%)				
Respiratory failure	46 (54.76)	20 (34.4)	26 (100)	<0.001
Sepsis	30 (35.71)	14 (24.1)	16 (61.5)	0.001
MAS	25 (29.76)	13 (22.4)	12 (46.15)	0.02
Hypocalcemia	24 (28.57)	12 (20.6)	12 (46.15)	0.02
Apnea	5 (5.95)	2 (3.4)	3 (11.5)	>0.99
PPHN	5 (5.95)	4 (6.8)	1 (3.8)	>0.99
AKI	5 (5.95)	1 (1.7)	4 (15.38)	0.03
Polycythemia	03 (3.57)	1 (1.7)	2 (7.6)	0.22
Anemia	03 (11.90)	5 (8.6)	5 (19.2)	0.27
Hypoglycemia	3 (3.57)	2 (3.4)	1 (3.8)	>0.99
IVH	1 (1.19)	1 (1.7)	0 (0)	>0.99
HIE stages				
No HIE	03 (3.57)	3 (5.1)	0 (0)	0.064
HIE stage 1	22 (26.19)	18 (31.0)	4 (15.38)	
HIE stage 2	47 (55.95)	32 (55.17)	15 (57.69)	
HIE stage 3	12 (14.28)	5 (8.6)	(26.9)	
Laboratory parameters, (Mean (SD)				
LDH (U/l)	2009 (821.15)	1886.5 (828.58)	2282 (748.92)	0.040
CPK (U/l)	1371.1 (677.43)	1351.9 (706.25)	1431.9 (619.34)	0.619
Lactate(mmol/l)	5.85 (3.76)	4.796 (3.34)	8.2 (3.64)	<0.001
Outcome at discharge, (Mean (SD)				
Mean duration of hospital stay (days)	10.33 (5.57)	8.65 (4.86)	14.07 (5.28)	<0.001
Mean duration of establishment of breast feeding (days)	6.41 (4.14)	5 (3.366)	9.5 (4.042)	<0.001
Microcephaly	9 (10.71)	4 (6.89)	5 (19.23)	0.1
Abnormal tone	50 (59.52)	26 (44.83)	24 (92.31)	<0.001

MBA-moderate birth asphyxia, SBA-severe birth asphyxia, MAS-meconium aspiration syndrome, PPHN-persistent pulmonary hypertension of the newborn, AKI-acute kidney injury, IVH-intraventricular hemorrhage, HIE-hypoxic ischemic encephalopathy, LDH-lactate dehydrogenase and CPK-creatine phosphokinase.

Table 2: Growth and development on follow up.

Variables	3 months, n=84 (%)	6 months, n=84 (%)	P value
W/A (<-3SD)	28 (33.33)	43 (51.19)	0.02
L/A (<-3SD)	5 (5.95)	7 (8.33)	0.76

Continued.

Variables	3 months, n=84 (%)	6 months, n=84 (%)	P value
W/L (<-3SD)	35 (41.66)	30 (35.71)	0.52
Microcephaly	11 (13.09)	11 (13.09)	0.81
TDSC fail	41 (48.8)	56 (66.67)	0.04

W/A-weight for age, L/A-length for age, W/L-weight for length, TDSC-Trivandrum development screening chart and DASII-Developmental assessment scale for Indian infants.

Table 3: Growth and development at 3 and 6 months of age.

Follow up at 3 months				Follow up at 6 months			
Growth	**MBA n=58 (%)**	**SBA n=26 (%)**	**P value**		**MBA n=58 (%)**	**SBA n=26 (%)**	**P value**
W/A	10 (17.2)	18 (69.2)	<0.001	W/A	20 (34.5)	23 (88.5)	<0.001
L/A	2 (3.4)	3 (11.5)	0.169	L/A	1 (1.7)	6 (23.1)	<0.003
W/L	17 (29.3)	18 (69.2)	<0.001	W/L	16 (27.6)	14 (53.4)	0.02
Tone abnormalities	26 (44.8)	24 (85.5)	<0.001	Tone abnormalities	30 (51.7)	25 (96.2)	<0.001
Microcephaly	3 (5.2)	8 (30.8)	0.003	Microcephaly	3 (5.2)	8 (30.8)	0.003
Post-discharge seizures	12 (20.7)	23 (88.5)	<0.001	TDSC fail	31 (53.4)	25 (96.2)	<0.001
TDSC fail	16 (27.6)	25 (96.2)	<0.001	Abnormal DASII at 6 months	20 (34.5)	25 (96.2)	<0.001

W/A-weight for age, L/A-length for age, W/L-weight for length, TDSC-Trivandrum development screening chart and DASII-Developmental assessment scale for Indian infants.

Table 4: Logistic regression for developmental outcome determinants.

Variables	Developmental outcome		P value
	Normal, n=39 (46.42%)	**Abnormal, n=45 (53.57%)**	
Mean weight at birth (SD) (grams)	2727 (0.340)	2590 (0.334)	0.079
Mean weight at 6 months of age (SD) (grams)	6800 (0.653)	6300 (0.766)	0.002
Microcephaly at 6 months	0 (0)	11 (24.4)	0.001
Laboratory parameters mean (SD)			
LDH (U/l)	1859.3590 (675.1967)	2138.6889 (917.1561)	0.12
CPK (U/l)	1206.5103 (516.9754)	1513.8222 (768.1194)	0.037
Lactate (mmol/l)	3.9333 (3.1026)	7.5111 (3.5155)	<0.001
Mean duration of hospital stay days (SD)	7.5641 (4.6892)	12.7333 (5.1847)	<0.001
Mode of oxygenation			
Only O_2 support	25 (64.1)	13 (28.9)	0.001
Ventilator support	14 (35.6)	32 (71.1)	0.001
Mean duration of breastfeeding establishment days (SD)	4.56 (3.393)	8 (4.101)	<0.001
Comorbidities			
HIE stages			
No HIE	3 (7.7)	0 (0)	0.001
HIE stage 1	17 (43.6)	5 (11.1)	0.001
HIE stage2	16 (41)	31 (68.9)	0.001
HIE stage 3	3 (7.7)	9 (20)	0.001
Respiratory failure	14 (35.9)	32 (71.1)	0.001
MAS	8 (20.5)	28 (62.2)	<0.001
Hypocalcemia	7 (17.9)	17 (37.8)	0.045
AKI	0 (0)	5 (11.1)	0.04

LDH-lactate dehydrogenase, CPK-creatine phosphokinase, O_2-oxygen, HIE-hypoxic ischemic encephalopathy, MAS-meconium aspiration syndrome and AKI-acute kidney injury

DISCUSSION

Our study revealed that of the total neonates (n=84) with moderate and severe birth asphyxia, 26 (30.95%) neonates had severe birth asphyxia and 58 (69.04%) neonates had moderate birth asphyxia. Respiratory failure was seen in 100% babies with severe birth asphyxia as against 34.5% in moderate birth asphyxia. HIE stage III was seen 8.6% babies with moderate birth asphyxia and in 26.9% of babies with severe birth asphyxia. Duration of NICU stay and time for establishment of breastfeeding was significantly longer in babies of severe birth asphyxia. Mortality was higher in group with severe birth asphyxia. At 3 month and 6 month follow up, growth lag, and microcephaly were more common in babies with severe birth asphyxia. At 3 month follow up 96.2% of infants with severe birth asphyxia and 27.6% of infants with moderate birth asphyxia failed TDSC. The 96.2% babies of severe birth asphyxia had developmental delay as assessed by DASII at 6 months in comparison to 34.5% in babies of moderate birth asphyxia.

Incidence of birth asphyxia in our study is 17.6 per 1000 live births. According to the NNPD 2002-2003 network report, the incidence of birth asphyxia is 1.4%. Among intramural deliveries, APGAR scores <7 at 1 minute (includes moderate and severe asphyxia) were documented in 8.4%.[5] The incidence observed in Prakash et al and Boskabadi et al was 2% and 1% respectively.[12,13] This varied incidence observed may be due to the fact that this study was conducted in a tertiary care centre where many of the high-risk pregnancies are referred and managed. A higher incidence of birth asphyxia was observed in males (59.5%) as compared to females (40.5%) in our study. This was consistent with studies conducted in Bangladesh (60.8%), Karachi (61.3%) and Nigeria.[14-16] In our study, the incidence of moderate birth asphyxia was higher (69.04%) than the incidence of severe birth asphyxia (30.95%). Similar findings were present in Bayih et al where the incidence of moderate birth asphyxia was 78.8%.[17] In our study morbidities like respiratory failure and MAS cases increased with increasing severity of birth asphyxia. This finding was consistent with the findings of Shah et al where they had considered respiratory distress could be due to either MAS and/or brain edema.[18]

Reduced energy supply through hypoxic-ischemia (HI) with exhaustion of energy reserves and subsequent failure of oxidative metabolism is recognized as the primary insult. This acute phase is characterized by anaerobic metabolism, depletion of adenosine triphosphate and a subsequent decrease in transcellular transport of ions resulting in an accumulation of intracellular sodium, water and calcium. During this secondary phase, delayed neuronal death encompasses a complex cascade of events including secondary energy failure, cytotoxic edema, mitochondrial dysfunction, calcium influx, caspase release, oxygen and nitrosative stress leading to apoptosis and necroptosis.[19-27] This process triggers the innate immune response including astrocyte and microglial activation and production of inflammatory cytokines and chemokines.[28,29] This secondary injury is often associated with clinical deterioration and encephalopathy due to brain swelling and seizure activity.[25,30] The clinical state of encephalopathy after a HII is commonly referred to as HIE.

It was also observed that severity of asphyxia was directly proportional to other associated comorbidities like sepsis, hypocalcemia and AKI. Similar findings were present in a study by Acharya et al.[31] Birth asphyxia results in ischemia to the proximal tubule, thereby developing acute tubular necrosis and acute renal failure.[31] Oligo-anuria following hypoxic-ischemic injury is common, frequently associated with haematuria, and results from renal tubular damage. Serum creatinine and blood urea concentrations increase progressively, reaching the peak in the days following the injury.[32] AKI was found in 5 (5.95%) babies in our study. Laboratory parameters like LDH levels were shown to be closely related with the severity of birth asphyxia which showed $p<0.05$ in this study and this was in keeping with the findings of Antil et al where LDH in mild, moderate and severe asphyxiated newborns showed a statistically significant difference.[33] In our study, the serum lactate levels were found to be higher in babies with severe asphyxia than those having moderate birth asphyxia. Higher mean lactate and CPK levels at birth are early markers of abnormal developmental outcome at 6 months. A study conducted by Anusha et al had similar observations.[34] There was a strong correlation seen between length of stay and severity of asphyxia in our study where neonates with severe asphyxia required on an average 5.42 days longer stay as compared to moderate birth asphyxia. Similar findings were observed in Acharya et al and Thakkar et al.[31,35] Most studies have correlated the development of babies with HIE, not with severity of birth asphyxia.[31] Most studies have picked up the delay at 6 months or later.[7-9,35] In our study 96.2% babies with severe birth asphyxia and 27.6% babies with moderate asphyxia failed TDSC at 3 month follow up. At 6 months the percentage of babies with moderate birth asphyxia to fail TDSC increased to 34.5%. DASII at 6 months was abnormal in all severely asphyxiated babies who failed TDSC at 3 months and 6 months, so TDSC is a good screening test to pick up developmental delay as early as 3 months.

In our study seizures were more common in SBA (88.5%) than MBA (20.7%) at 3 months follow up post discharge. In our literature search we did not find studies correlating growth parameters at 3 months and 6 months in asphyxiated babies. It was noted by Das et al that infant with developmental delay had significantly higher incidence of growth failure.[36] In our study growth parameters like weight for length and weight for age were more affected in babies with severe birth asphyxia than in babies with moderate birth asphyxia. Onset of growth lag also was earlier in babies with severe birth asphyxia.

69.2% of babies with severe asphyxia had severe wasting (weight for length ≤-3SD), in comparison to 17.2% with moderate asphyxia. Severe underweight (weight for age ≤3SD) was also more in severely asphyxiated babies at 3 month follow up (69.2 versus 29.3% respectively for severe and moderate asphyxia respectively). Low mean weight for age at 6 months was a significant determinant of abnormal development. Naseer et al found that mean weight for age was also significantly less at 6 months of age in babies with HIE.[37] Microcephaly was seen in 30.8% at 3 months and 6 months in babies of severe birth asphyxia in our study. In a study conducted in Nigeria it was noted that severe birth asphyxia was a major cause of microcephaly seen in 70.5% of all of microcephaly.[38]

Limitation of the study was that it included only full-term babies with asphyxia. For defining asphyxia, we have used NNPD definition and have not done biochemical investigations like cord blood pH. It is a single center study with a limited sample size so result could not be generalized for the whole population. Strength of this study was the robust follow-up, early determinants for developmental and neurological outcome were identified successfully, and birth asphyxia was used as an inclusion criterion rather than only HIE as in most studies.

CONCLUSION

Incidence of comorbidities is higher in severely asphyxiated babies in comparison to moderately asphyxiated neonates. Laboratory markers like serum lactate and LDH at birth can be used as predictors of severity of birth asphyxia. Growth and development can be affected and picked up as early as 3 months in these babies on close follow up.

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