Original Article

Clinical profile and short-term outcome of perinatally asphyxiated term neonates in a tertiary hospital in Southern Kerala

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Received – 31 March 2017 Initial Review – 27 April 2017 Published Online – 06 June 2017

ABSTRACT

Introduction: In India, in spite of improvement in perinatal-neonatal care, perinatal asphyxia accounts for 23% of the neonatal deaths. **Objective:** The objective of the study was to study the clinical profile and short-term outcome of perinatally asphyxiated term neonates. **Materials and Methods:** This prospective study conducted at a tertiary care teaching hospital in Southern Kerala from June 2011 to June 2015. 120 term asphyxiated neonates fulfilling the inclusion criteria admitted in the NICU were followed up till death or survival. **Results:** 49.2% babies were inborn and 50.8% babies were outborn. Of the total, 53 (44.2%) were delivered vaginally, 54 (45%) by cesarean section, and 13 (10.8%) by instrumental delivery. Antenatal complications were seen in 58 (48.3%) and intrapartum complications in 93 (77.5%). Hypoxic ischemic encephalopathy (HIE) was diagnosed in 78.3%, with HIE 1 in 19.3%, HIE 2 in 27.5%, and HIE 3 in 31.6%. The mortality was 31 (25.8%) and it was more in out born babies compared to inborn. Factors associated with development of severe HIE (HIE 3) were male gender (p=0.0057), need for endotracheal intubation (p=0.0114), instrumental delivery and pH <7.2 (p=0.0013). Factors associated with mortality were instrumental delivery (p=0.0032), place of birth (p=0.0012), pH ≤ 7 (p=0.0006), HIE 3 (p<0.0001), and 5 min Apgar ≤3 (p=0.0372). **Conclusion:** HIE was seen in 78.3% perinatally asphyxiated babies with HIE 3 contributing to 31.6%. The mortality rate in HIE 3 was 81.6% which was significantly associated with place of birth, instrumental delivery, pH <7, and 5 min Apgar ≤3.

Key words: *Hypoxic ischemic encephalopathy, Perinatal asphyxia, Short-term outcome*

Perinatal asphyxia is the third major cause of neonatal mortality in India [1]. It is also the fifth largest cause of underfive mortality and exerts a great pressure on the health system [2]. According to the World Health Organization (WHO), around 4 million babies develop birth asphyxia, and asphyxiated newborn may develop severe consequences such as epilepsy, cerebral palsy, developmental delay, and mental retardation. Furthermore, of 1.2 million neonatal deaths in India, 300,000-350,000 babies die due to perinatal asphyxia mostly within first 3 days of life [3]. Asphyxial injury may involve virtually every organ system of the body, but hypoxic ischemic encephalopathy (HIE) is the most studied and serious sequelae. The severity of HIE symptoms reflects the timing and duration of insult. The majority (90%) of the insults occur in the antenatal and intrapartum period. The remainder is in the immediate postnatal period due to cardio respiratory or neurological abnormalities [4]. The means of assessment include Apgar scores, blood pH, fetal heart rate abnormalities, need for resuscitation, neurological changes, and evidence of multiorgan dysfunction [5].

Umbilical cord blood gas analysis is now recommended in all high risk deliveries by both the British and American college of obstetricians and gynecologists. Low cord pH in neonates without cardiopulmonary compromise does not indicate an increased risk of adverse outcome. Babies, with pH <7 at birth and non-vigorous, have high risk of adverse outcome. In a study by Yeh et al., the ideal cord arterial blood pH was 7.26-7.30. The risk of adverse neurological outcome starts to rise at a pH <7.10, with the risk being highest at a pH <7 [6]. A systematic review in 2010 concluded that low arterial pH in umbilical cord strongly correlated with adverse outcomes such as HIE, periventricular leukomalacia (PVL), intracranial hemorrhage, cerebral palsy, and death [7]. An umbilical cord pH <7.2 immediately after birth is used as a prognostic factor for unfavorable short-term outcome in newborn [8]. In an asphyxiated newborn, an artery cord sample may underestimate the acidosis in fetus or newborn since lactic acid produced by hypoxia at tissue level will not be cleared to central circulation. As the baby is resuscitated, circulation improves and tissue lactic acid reaches the central circulation. The postnatal base deficit obtained from an asphyxiated newborn within 1st h after delivery is found to be worse than cord levels, and hence, this blood gas parameter is one of the most accurate predictors of neurological outcome [9].

In spite of improvements in obstetric and neonatal care, the incidence of birth asphyxia in India is high. The neonatal mortality has slightly decreased but morbidity in the form of neurological damage is same or increased due to survival of asphyxiated

babies [10,11]. There are only a handful of published studies in perinatal asphyxia from developing countries; especially, its relation with blood pH. To date no such study is available from Kerala; hence, this study was conducted to study the clinical profile and immediate outcome of term asphyxiated babies.

MATERIALS AND METHODS

This prospective observational study was conducted in a tertiary care teaching hospital of Southern Kerala between June 2011 and June 2015. Term babies admitted in the NICU with perinatal asphyxia were included in the study. Asphyxia was defined by the presence of any four of the following criteria - 5 min Apgar≤6/10, meconium stained amniotic fluid (MSAF), pH within 1 h <7.2, changes in fetal heart rate, and evidence of neurological or multiorgan dysfunction. Babies referred from other hospitals were included in the study, only if they were referred within the 1st h after birth. Preterm babies, babies with syndromic/congenital malformations, congenital neuromuscular disorders, congenital cardiac and pulmonary disorders, intrauterine infections, sepsis and babies referred from other hospitals after 1 h of birth were excluded from the study.

All the neonates were resuscitated according to the Neonatal Resuscitation Program guidelines and were admitted in the NICU. Venous blood gas for pH was taken within 1 h. The pro forma containing the details of antenatal and intrapartum events were recorded. Clinical and neurological examination was done twice a day and hourly monitoring was done for ventilated babies. Neurological classification of disability was graded by Sarnat and Sarnat staging. All the neonates were managed as per the hospital protocol including maintenance of oxygenation by oxygen hood/continuous positive airway pressure/ventilation, maintenance of euglycemia with intra venous fluids, calcium supplementation, maintenance of blood pressure and ionotropic support if needed and seizure control with antiepileptics. All of them were followed up in NICU till death or discharge.

Statistical methods used were descriptive statistics means, percentages and tests of significance such as Chi-square test and Fischer's exact test. Pearson correlation coefficient was calculated to find out the relation between Apgar score at 1 and 5 min versus pH.

RESULTS

A total number of babies enrolled in the study were 120. There were 61 (50.8%) outborn babies and 59 (49.2%) inborn babies. Demographic profile of the study population is presented in Table 1. Total 93 (77.5%) babies had intrapartum complications including MSAF in 42 (35%) and nonreassuring fetal status in 48 (40%) cases as shown in Table 1. 96% of the mothers with prolonged and difficult labor were delivered vaginally and 75% required instrumentation. 85.2% of babies delivered vaginally following prolonged and difficult labor developed HIE 3. Cord around neck was present in 12 (10%). 92% of babies in this group were delivered vaginally and 91% of them developed HIE 3.

Table 1: Demographic and clinical profile of the study population (n=120)

Determinant	Characteristic	N (%)
Gender	Male	58 (48.3)
	Female	62 (51.7)
Gestational age	SGA	28 (23.3)
	AGA	87 (72.5)
	LGA	5 (4.16)
Mode of delivery	Vaginal	53 (44.2)
	LSCS	54 (45)
	Instrumental	13 (10.8)
Antenatal risk factors	Any antenatal risk factor	58 (48.3)
	Gestational hypertension	23 (19.2)
	Diabetics	14 (11.7)
	IUGR	18 (15)
Intrapartum risk factors	Any intrapartum risk factor	93 (77.5)
	MSAF	42 (35)
	NRS	48 (40)
	Prolonged labor	28 (23.3)
	Cord around neck	12 (10)
Resuscitation	Bag and mask	18 (15)
	Endotracheal intubation	102 (85)
pH	≤7	13 (10.83)
	>7 and <7.2	81 (67.5)
	>7.2 to ≥7.2	26 (21.6)
HIE stage	No HIE	26 (21.6)
	HIE stage 1	23 (19.16)
	HIE stage 2	33 (27.5)
	HIE stage 3	38 (31.6)
Place of birth	Inborn	59 (49.17)
	Out born	61 (50.83)

NRS: Non reassuring fetal status, LSCS: Lower section cesarean section, SGA: Small for gestational age, LGA: Large for gestational age, AGA: Appropriate for gestational age, IUGR: Intrauterine growth restriction, HIE: Hypoxic ischemic encephalopathy

About 94 (78.3%) babies developed HIE and majority developed HIE 3 in 38 (31.6%) cases followed by HIE 2 in 33 (27.5%) and HIE 1 in 23 (19.3%) babies. All babies with HIE 3 were mechanically ventilated and all had clinical shock requiring ionotropes. The mortality in our study was 31 (25.8%). All babies with HIE 1 and 2 survived while only 7 (18.42%) out of the 38 babies with HIE 3 survived. The mortality was higher in out born 22 (70.1%) than in inborn 9 (29.9%) babies which stresses the importance of early referral of high-risk pregnancies *in utero* and their timely intervention.

Of the 13 babies with severe acidemia, 8 had history of MSAF and 7 had non-reassuring fetal heart rate pattern. 9 out of the 13 babies with severe acidemia died but only 9 out of the 31 babies who died, had severe acidemia. Pearson correlation coefficient for 1 min Apgar and pH was 0.151 (p=0.101), whereas for 5 min Apgar, it was 0.228 (p=0.012), which was highly significant. Table 2 shows the factors responsible for development of HIE while Table 3 shows the factors affecting outcome studied.

Table 2: Factors responsible for development of HIE (n=120)

Determinants	Category	HIE 1 and 2			HIE 3		
		No	Yes	p value	No	Yes	p value
Gravida	Primi	15	60	0.648	50	25	0.687
	Others	11	34		32	13	
Birth weight	SGA	6	22	1 0.581	16	12	0.153 0.619
	AGA	20	67		63	24	
	LGA	0	5		3	2	
Gender	Male	15	43	0.375	47	11	0.0057
	Female	11	51		35	27	
Mode of delivery	Vaginal	13	40	1 0.436	35	18	0.1804 0.0473
	LSCS	12	42		41	13	
	Instrumental	1	12		6	7	
Antenatal risk factors	Present	15	43	0.375	39	19	0.8460
	Absent	11	51		43	19	
Intrapartum risk factors	Present	19	74	0.597	62	31	0.638
	Absent	7	20		20	7	
MSAF	Present	8	34	0.651	26	16	0.306
	Absent	18	60		56	22	
NRS	Present	10	38	1.00	34	14	0.691
	Absent	16	56		48	24	
Endotracheal intubation	No	9	9	0.0037	17	1	0.0114
	Yes	17	85		65	37	
pH	≤7	0	13	0.685	4	9	0.0038
	>7	26	81		78	29	
pH	≤7.2	11	73	0.0012	50	34	0.0013
	>7.2	15	21		32	4	
1 min Apgar	≤3	21	84	0.3124	72	33	1.000
	>3	5	10		10	5	
5 min Apgar	≤3	3	8	0.7022	5	6	0.1002
	>3	23	86		77	32	

HIE: Hypoxic ischemic encephalopathy

Factors significantly associated with development of HIE were the need for endotracheal intubation (p=0.0037) and pH <7.2 (p=0.0012). Factors associated with development of severe HIE (HIE 3) were male gender (p=0.0057), need for endotracheal intubation (p=0.0114), instrumental delivery, severe acidemia (pH <7) (p=0.0038), and moderate acidemia (pH <7.2) (p=0.0013). Factors significantly associated with mortality were instrumental delivery (p=0.0032), place of birth (p=0.0012), pH ≤7 (p=0.0006), HIE 3 (p<0.0001), and 5 min Apgar ≤3 (p=0.0372). The relationship between pH within 1 h of birth and mortality is shown in Fig. 1. This box plot shows that the pH was very much lower in neonates who expired than in those who survived. The median (IQR) of pH in neonates who died was 7.04 (7.1, 7) as against 7.2 (7.24, 7.1) in neonates who survived. Fig. 2 shows the relationship between pH within 1 h of birth and occurrence of stage 3 HIE. The median (IQR) of pH in neonates who developed HIE 3 was 7.07 (7.14, 7.02) as against 7.2 (7.24, 7.1) in neonates who did not develop HIE 3.

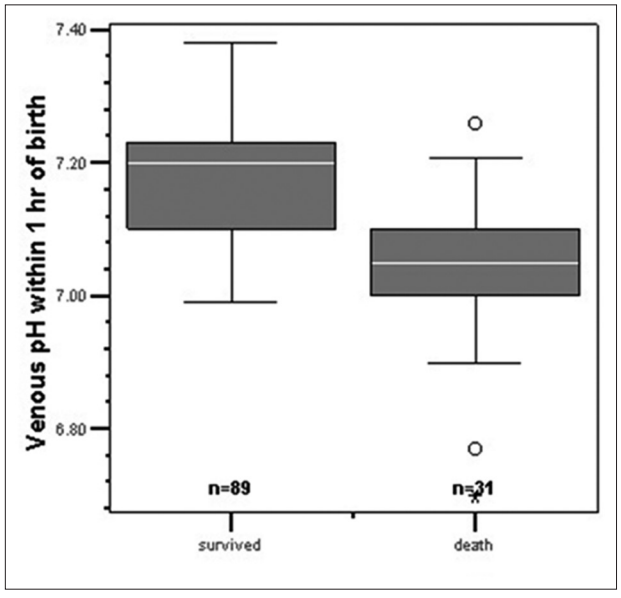

Figure 1: pH within 1st h in survived and expired babies

Table 3: Factors affecting the outcome (120)

Determinant	Characteristic	Survival	Death	Significance
Gender	Male	48	10	0.059
	Female	41	21	
Gestation	SGA	20	8	0.085
	AGA	65	22	
	LGA	3	2	0.6026
Mode of delivery	Vaginal	37	16	0.1141 0.0323
	LSCS	45	9	
	Instrumental	7	6	
MSAF	No	59	19	0.664
	Yes	30	12	
pH	≤7	4	9	0.0006
	>7	85	22	
Place of birth	Inborn	50	9	0.012
	Out born	39	22	
HIE	Stage 1	26	0	<0.0001
	Stage 2	23	0	
	Stage 3	7	31	
1 min Apgar	≤3	77	28	0.757
	>3	12	3	
5 min Apgar	≤3	5	6	0.0372
	>3	84	25	

LSCS: Lower section cesarean section, SGA: Small for gestational age, LGA: Large for gestational age, AGA: Appropriate for gestational age

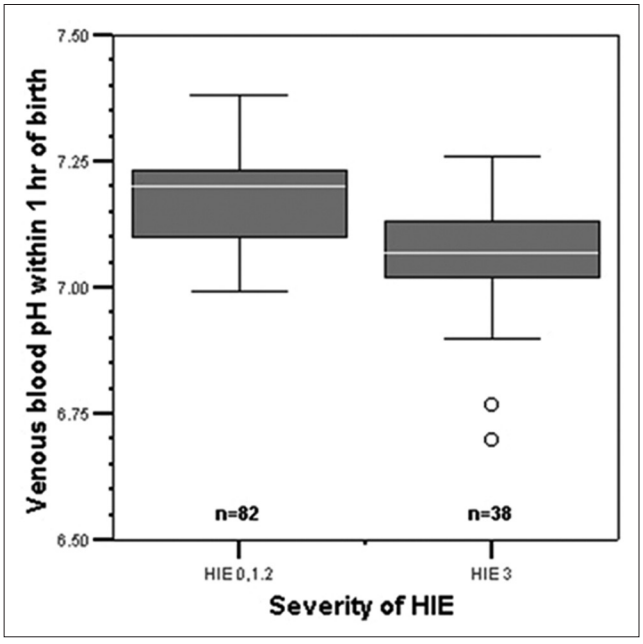

Figure 2: Venous blood pH within 1 h of birth and hypoxic ischemic encephalopathy 3

DISCUSSION

This study mainly determined the clinical profile and immediate outcome of asphyxiated term neonates. The incidence of birth asphyxia ranges from 5.4/1000 live births in Swedish study [12] to 22/1000 live hospital births in Indian studies [13]. According to WHO, incidence of birth asphyxia is around 3%, i.e., out of 130 million newborns each year globally, around four million develop birth asphyxia, and from asphyxiated babies, around 1.2 million die [14]. This variation in different studies was due to different operational definitions for birth asphyxia adopted by different researchers; Apgar score at 1 or 5 min, duration of resuscitation, breathing effort at 1 min, etc. WHO defined perinatal asphyxia as a failure to initiate and sustain breathing at birth. The National Neonatology Forum National Neonatal Perinatal Database defines moderate asphyxia as slow gasping respiration or Apgar 4-6 at 1 min and severe asphyxia as no breathing or Apgar 0-3 at 1 min of age [1]. The 5 min Apgar score is still the most practical and reliable parameter for assessing the effectiveness of resuscitation and vitality of the newborn [15]. In developing countries, rates of birth asphyxia are higher and case fatality may be 40% or higher [16].

In our study, the male to female ratio was 0.93:1. This is in contrast to other studies where males outnumbered the females [17,18]. In our study, the majority (72.5%) were appropriate for gestational age (AGA). This is similar to Kempegowda study, where 80% were AGA [17]. Post maturity had been noted to be an important risk factor of birth asphyxia by earlier workers like Azam et al. [19] which was not seen in our study. In our study, birth asphyxia was more common in babies delivered by primigravida which is in accordance to the previous studies by Dongol et al. (58.8%) [20], Bagalkot study (68.1%) [21], and Rosalind et al. (58%) [22]. In our study, 93 (77.5%) had intrapartum risk factors and MSAF (35%) and non reassuring fetal status (NRS) 48 (40%) were the major contributors. This is comparable to the study by Sarnappa et al., where MSAF was the contributing factor in 40% of cases [17] and a study in Iceland where 50% had MSAF [23]. According to Shrestha et al., 51% had intrapartum risk factors. Of these, MSAF (in 65% cases), nonvertex presentation and fetal heart rate abnormalities accounted for 4-fold risk of asphyxia [21].

In our study, all the instrumental deliveries with birth asphyxia developed HIE. 85% of the babies' required endotracheal intubation while in a study by Sonia et al., only 34% required endotracheal intubation [22]. In our study, 78.3% developed HIE, with HIE 1 in 19.3%, HIE 2 in 27.5%, and HIE 3 in 31.6%. We had a mortality of 31 (25.8%). According to Shrestha et al., 25 babies out of 85 (29%) developed HIE and of these, 6% expired. 62% babies were in stage 2 and 38% in Stage 1. This low rate of HIE and mortality with no cases of HIE 3 could be due to the inclusion criteria of taking all babies with 1 min Apgar ≤6 as asphyxiated [21]. Another study showed 38% HIE 1, 48% HIE 2, and 14% HIE 3 [22] taking the same inclusion criteria. Prolonged difficult labor was seen in 28 (23.3%) in our study. In the study by Sonia et al., there were 44% cases of prolonged labor [22]. 51% had prolonged labor in a Nigerian study [24]. According to this study, 29.7% had severe asphyxia which is comparable to our study. The mortality was 27.3% and of this 52% had severe, 43% moderate, and 5% mild HIE [24].

According to Kumar et al., the outcome in terms of mortality in HIE 1, 2, and 3 was 2 (3.3%), 35 (41.17%), and 23 (85.18%), respectively [25]. In a study by Memon et al., HIE was seen in 85% babies, with 30% in HIE 1, 35% in HIE 2 and 20% in HIE 3. 15 % of babies died in this study. All babies with HIE 3 expired or were discharged with disability. Inclusion criteria in this study were 5 min Apgar <7/delayed cry/need for resuscitation for >10 min [26]. The majority of deaths in HIE 3 show that once severe asphyxia occurs, treatment is not effective, and more attention should be focused for early assessment and intervention which is efficacious in reducing severity of brain damage. In a study by Kumar et al., 17.7% had HIE 1, 53.8% had HIE 2, and 28.5% had HIE 3 [27].

In our study, 87.5% had Apgar ≤3 at 1 min and 12.5% had Apgar 4-6 at 1 min. Furthermore, 10.83% had severe acidosis (pH ≤7) and 67.5% had moderate acidosis (pH >7 and <7.2). According to Kumar et al., 54 (40%) had Apgar score ≤3 and 87 (60%) had Apgar 4-6 at 1 min. Of the 145 cases, 54 (37.24%) had cord blood acidosis. There was strong association between cord blood acidosis (pH <7) and severity of HIE [27]. The newborns who had low Apgar scores were significantly more acidotic during labor and delivery and during the 1st h of life than babies born with higher Apgar scores [28,29].

At birth and during the 1st h of life, the neonates of high-risk mothers were more acidotic and recovered more slowly than neonates of normal mothers [30]. pH <7.1 has a 48% sensitivity, 85% specificity, 60% positive predictive value, and 78% negative predictive value in short-term outcome of encephalopathy [31]. Interestingly, the presence of MSAF and/or NRS (clinical/CTG) is of high sensitivity but low specificity. The classic features of fetal distress such as MSAF and NRS have been shown not to be sufficiently reliable as an indicator for accurate assessment of fetus. For confirmatory interpretation, it should be combined with blood gas analysis [27]. The babies with pH <7 are more likely to suffer complications in short term, while long-term outcome is correlated to neonatal encephalopathy rather than pH [30]. In our study, 69.2% of babies with severe acidemia died and 29% of babies who died had severe acidemia. Severe adverse sequelae in the newborn period are rare after birth with umbilical cord pH >7. Even at pH <7, most of the neonates still recover fully without remarkable illness. In this respect, cord pH or base excess alone are poor predictors of outcome [32].

According to King et al., combination of low pH at birth and abnormal clinical patterns becomes strongly predictive of adverse sequelae [33]. Pertman and Risser showed that a combination of cord pH <7, a requirement for intubation and 5 min Apgar ≤5 had an 80% positive predictive value for development of seizures [34]. Furthermore, Portman et al. found that a score combining cardiotocographic abnormalities, umbilical arterial base excess, low 5 min Apgar was much more strongly associated with morbidity and mortality than any individual factors. This score showed positive predictive value of 73% and negative predictive value of 99% for predicting impairment of 3 or more organ systems [35]. Goldaber et al. showed that neonatal death was much more likely at pH <7 and the cutoff at which seizures become more likely was pH <7.1. They recommended a realistic value for defining pathological acidemia as pH <7 [36]. William and Singh also found that threshold pH <7 was the best independent predictor of neonatal seizures when compared to other indices [37]. Casey et al. found that neonates in whom acidosis (pH <7.2) persisted 2 h beyond delivery had poor outcome than those whom acidosis resolved [38]. A meta-analysis carried out within the predefined groups showed that low arterial cord pH was significantly associated with neonatal mortality (odds ratio 16.9, 95% CI 9.7 to 29.5, I2=0%), HIE (13.8, 6.6 to 28.9, I2=0%), IVH or PVL (2.9, 2.1 to 4.1, I2=0%), and cerebral palsy (2.3, 1.3 to 4.2 1(2)=0%) [7]. Persistence of metabolic acidosis on the first arterial blood sample obtained in the 1st h of age was significantly associated with HIE (p<0.005) [39].

A scoring system could be developed and validated for predicting the multiorgan dysfunction and mortality following perinatal asphyxia in our country. Identification of neonates at risk of encephalopathy is especially important now that early intervention is being considered. Hence, it is imperative that appropriate diagnostic and management modalities for fetal hypoxia be available and accessible. This is the only way by which the burden of perinatal mortality and morbidity can be reduced with a view to achieving the millennium development goal of improved perinatal outcome.

CONCLUSION

The burden of perinatal asphyxia is certainly enormous and worse in the developing countries like India. In this study, 78.3% babies developed HIE with a mortality rate of 25.8%. HIE 3 constituted the majority with 81.5% mortality. The mortality was higher in out born (70.1%) compared to inborn newborns (29.9%). There was significant correlation between instrumental delivery, 5 min Apgar ≤3 and severe acidosis (pH <7) and adverse outcomes.

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Funding: None; Conflict of Interest: None Stated.

How to cite this article: Joseph S, Bindusha S, Radhika S, Krishnan R, Kumar S. Clinical profile and short-term outcome of perinatally asphyxiated term neonates in a tertiary hospital in Southern Kerala. Indian J Child Health. 2017; 4(3):399-404.