

Original Research Article

Time to recovery from asphyxia and its predictors among newborns admitted to neonatal intensive care units at public hospitals in North Shoa, Amhara region, Ethiopia

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ABSTRACT

Background: Birth asphyxia is a cause of early neonatal mortality worldwide. Similarly, the burden of birth asphyxia in Ethiopia remains high. Thus, studies on the time to recovery of asphyxiated neonates and their predictors are critical. Therefore, the current study aimed to determine the time to recovery of asphyxiated neonates and its predictors among newborns admitted to the neonatal intensive care unit of public hospitals in North Shewa, Amhara region, Ethiopia.

Methods: A retrospective cohort study was conducted on 523 asphyxiated neonates in North Shoa, using a simple random sampling method. Data was collected using Kobo and STATA, and survival times were estimated using Kaplan-Meier survival curves. Bivariate and multivariate cox proportional hazards regression analyses were performed.

Results: A total of 523 admitted asphyxiated newborns were monitored for 3682 neonatal days, ranging from a minimum of 1 day to a maximum of 28 days. The overall survival rate was 79.1%. The median recovery time was seven days (95% CI: 6.60-7.39). A depressed Moro reflex (AHR: 0.52, 95% CI: 0.38-0.87), stage I hypoxic ischemic encephalopathy (AHR: 2.5, 95% CI: 1.65-5.87), not receiving calcium gluconate (AHR: 1.40, 95% CI: 1.02-1.68), and receiving 2/3 intravenous fluids during their stay (AHR: 0.68, 95% CI: 0.53-0.99) were predictors of the time to recovery of asphyxiated newborns.

Conclusions: Compared with other research findings, the overall proportion of recovery is lower. To increase the recovery time from asphyxia, standardized care protocols, including fluid and electrolyte monitoring, could be established.

Keywords: Perinatal asphyxia, Recovery time, Neonatal intensive care unit, Survival

INTRODUCTION

Perinatal hypoxia arises due to disruption or decrease in the delivery of oxygen to the fetus around the time of birth, which then leads to oxygen deficiency and affects aerobic

metabolism, hence resulting in hypoxic damage to the tissues. Due to the high requirement for oxygen and energy, the brain is prone to hypoxic damage, causing ischemia and hypoxic-ischemic encephalopathy. Other critical organs such as the heart, kidney, liver, and the

digestive system may also be affected by ischemic damage. Therefore, perinatal hypoxia accounts for some of the major causes of neonatal morbidity and mortality.¹ As defined by the World Health Organization (WHO), birth asphyxia is “the inability of the baby to start and continue breathing immediately after birth.” Birth asphyxia causes inadequate exchange of gases, causing hypoxemia, hypercapnia, and metabolic acidosis, leading to injury of the brain and other essential organs due to hypoxic-ischemic encephalopathy.²

Birth asphyxia, an arterial pH <7.0, a base deficiency >12 mmol/l, and the need for positive pressure breathing with or without chest compressions at 10-minute intervals for continuous resuscitation can all be identified using clinical criteria, such as the need for a bag and mask during delivery and/or a five-minute APGAR score of less than seven, as well as biochemical criteria, such as hypercarpna, hypoxemia, and metabolic acidosis.³

Perinatal obstruction of blood flow through the placenta initiates adaptations in the fetal cardiovascular system that aid in maintaining the supply of oxygen to essential organs. These involve the redistribution of the cardiac output to benefit brain, heart, and adrenal glands at the expense of other organs—a phenomenon called brain sparing. These adaptations are caused by hypoxemia due to maternal, placental, and fetal factors. However, these adaptations do not work well in cases of severe or chronic hypoxemia and lead to poor myocardial contractility, reduced cardiac output, and hypotension. Ultimately, there could be circulatory failure leading to tissue ischemia and organ dysfunction.^{4,5}

Whereas most neonates affected by birth asphyxia respond well, the situation is critical for those suffering from perinatal hypoxia-ischemia, posing a threat of not only acute but also prolonged consequences. Acute complications may consist in hypoxic-ischemic encephalopathy, seizures, multi-organ dysfunction, and even mortality. However, even after surviving, the patients face serious problems like cerebral palsy and intellectual disability requiring lifelong treatment. The problem is psychologically, socially, and financially difficult for families and the whole community, especially for developing countries.⁶⁻⁸

Birth asphyxia carries considerable psychological, social, and economic burdens for families and health systems. Considering the fact that many cases of birth asphyxia can actually be prevented with appropriate obstetric and neonatal care, this condition needs to be considered one of the most important priorities in public health. Its prevention demands high quality antenatal, intrapartum, and postpartum care, which includes proper prenatal risk assessment, fetal monitoring during childbirth, skilled birth attendants, diagnosis of fetal distress, and neonatal resuscitation. Improving all of these evidence-based prevention and treatment strategies is vital in reducing the negative outcomes of perinatal hypoxia-ischemia.

METHODS

Study design

Institution-based retrospective cohort study was carried out at three public hospitals in the North Shoa, Amhara region of Ethiopia between 29 July and 24 August 2024. All asphyxiated neonates admitted to the neonatal intensive care units of selected public hospitals in North Shoa between 01 January 2020, and 31 December 2023, included in the study.

Eligibility criteria

Inclusion criteria

The study included all newborns with a confirmed diagnosis of asphyxia who were hospitalized in neonatal intensive care units of public hospitals in North China between 01 January 2020, and 31 December 2023, and who had known admission dates, discharge dates and discharge statuses.

Exclusion criteria

All asphyxiated neonates who had incomplete medical records, such as neonates with unknown admission dates, discharge dates and discharge statuses, were excluded from this study.

Sample size determination and sampling technique

Sample size determination

The sample size was determined via the log-rank test and the Freedman method via STATA software version 14.0.

$$E = \frac{(Z_{\alpha/2} + Z_{\beta})^2}{(\ln HR)^2 p(1-p)}$$

$$P(E) = \frac{1 - (\exp S1(t) + \exp S1(t) \times HR)}{2}$$

Where $Z_{\alpha/2}$ =standard normal variable at 95% confidence interval level=1.96, Z_{β} =power of 80%=0.842, hazard ratio=0.5, p =0.744, and censored=0.256 obtained from a retrospective study performed in Addis Ababa public hospitals on time to recovery and predictors of asphyxia among neonates.⁹

E =number of interested events is 67, and the probability of an event (recovery), and $P(E)$ =0.201. Therefore, the total sample size is calculated as follows.

$$n = \frac{E}{P(E)} = n = \frac{67}{0.201} = 333.33$$

Since the sampling procedure has different levels of hospitals (primary sampling unit and the selected hospitals

(secondary sampling unit) or the services given at each level of hospital are different), I took one hospital from each level and proportionally allocated for the total sample size. In such a way, an appropriate sample size design effect is necessary, $333.33 \times 1.5 = 499.99$, by adding 10% missing data, which is $499.99 + 49.99 = 549.98 \sim 550$.

Therefore, the sample size calculated with the addition of 10% missing data was 550.

Sampling technique and procedures

Since there are nine public hospitals in North China that have NICU clinics, three public hospitals, Debrebrehan Comprehensive Specialized Hospital (DBCSH), Alemketema General Hospital (AGH) and Deneba Primary Hospital (DPH), were selected.

The proportional allocation formula was then used to allocate each selected hospital on the basis of its three-year total asphyxia admission. After that, medical records of babies diagnosed with PNA were collected.

From the isolated cards in each hospital, a simple random sampling technique was applied to select the study participants. Finally, the selected medical charts (cards) were reviewed from 29 July to 24 August 2024.

Patient and public involvement

The patients were not involved in the study beyond being included in the study as the study reviewed medical charts retrospectively. The responsible chief executive directors of each hospital were involved in discussion regarding the feasibility, access and ethical considerations while handling the records of patients.

Data collection methods

The study retrieved registration books of asphyxiated baby charts admitted to three hospitals between January 2020 and December 2023. Data extraction was performed using appropriate formats. Retrospective follow-up was conducted from diagnosis to discharge, with recovery verified through discharge notes and registration.

Data collection tool/instruments

The PNA follow-up chart, the standardized health management information system (HMIS) registration book, and other peer-reviewed publications were used to create a data abstraction format (9, 13, and 23). Five components make up the data abstraction, which was created on the basis of the goals of the study.

These include checklists pertaining to sociodemographic data, obstetric and neonatal factors, clinical and laboratory factors, and management or therapy elements that were gathered from medical records.

Study variables

Dependent variable

Time to recovery from asphyxia.

Independent variables

Sociodemographic predictors include maternal age and place of residency. Obstetric predictors include anaemia, ANC follow-up, parity, prolonged labour, prolonged ROM, mode of delivery and meconium staining. Neonatal-related predictors include sex, gestational age, birth weight and age at clinical presentation.

Clinico-laboratory predictors include the Apgar score, HIE stage, clinical status at admission, infection or seizure within 24 hours, vital sign derangement, electrolyte disturbance and liver enzyme derangement.

Treatment-related predictors include bag mask ventilation, chest compression, adrenaline, aminophylline, fluid and hypothermia management.

Operational definitions

Perinatal asphyxia

This condition was considered on the basis of the clinical diagnosis made by health care providers (paediatricians, general practitioners).

Incidence density rate of survival

This rate is computed by dividing the number of events by the total follow-up time in person-days.

Stages of HIE

The stages of HIE were determined on the basis of the clinical diagnosis made by a health care provider (stage 1, stage 2 and stage 3).

Recovery time

The number of days from admission until a baby has recovered from the PNA.

Survival

In this study, surviving newborns were those who became well again (free of clinical features of asphyxia, declared recovered from PNA and/or discharged from the NICU due to completion of their management).

Censored

Censored are those newborns who had a death summary sheet, who were against medical advice (caregivers sign on

behalf of their baby to leave the treatment before recovery), or who were referred (transfer out) to other units or health facilities.

Died

Newborns who passed away and whose death report was recorded on the chart.

Even

Newborns who recover from PNA.

Data quality assurance

To ensure data quality, supervisors and data collectors underwent training on data collection from targeted sources. Data extraction forms were pre-checked to align with study needs. Daily onsite checks assessed data completeness, with prompt feedback from supervisors and the principal investigator. During data management, all completed forms were reviewed for completeness and consistency, with consistency verified through the random selection of patient charts.

Data processing and analysis

The Kobo data collection tool was used to export and clean data for analysis in STATA version 14.0. Key steps included cleaning and modifying data, verifying the Cox proportional hazard regression model assumptions using Schoenfeld residual and log-rank tests, and presenting findings through graphs and descriptive statistics. The incidence density rate was computed, and neonatal results were classified as retrieved or suppressed. Survival time was estimated via Kaplan-Meier curves, compared with the log rank test. Variables were fitted to a bivariable Cox regression model, with those showing a p value <0.25 included in the final analysis. Statistically significant predictors of survival were identified with hazard ratios, 95% CIs, and significance at $p<0.05$ in the multivariable analysis.

RESULTS

Sociodemographic and obstetric characteristics of the mothers of the study participants

A total of 550 charts of asphyxiated neonates were reviewed, and 523 (95.1%) were eligible for this study. The majority of mothers (393, 75.1%) were in the 20-34 years age group, with medians and IQRs of 26 and 23-30 years, respectively. Almost $\frac{3}{4}$ of the neonates in this age group survived. Among the 523 mothers, 245 (46.8%) and 278 (53.2%) were multiparous and prim parous, respectively. Only 11 (2.1%) mothers had no ANC follow-up. Almost half, 263 (50.3%) of the mothers gave birth via SVD, followed by C/S and assisted vaginal delivery, accounting for 140 (26.8%) and 120 (22.9%) mothers, respectively. Fetal distress (149, 28.5%), preeclampsia

(50, 9.5%), anaemia (32, 6.1%) and gestational DM (27, 5.2%) were the most common obstetric and medical complications during pregnancy (Table 1).

Neonatal characteristics of the study participants

Among the 523 study subjects, 301 (57.5%) were male. Among the neonates, 390 (74.6%) were term, 76 (14.5%) were preterm, and 57 (10.9%) were post term. Nearly three-fourths (382 (73.1%)) of the neonates had normal birth weights, 121 (23.1%) had low birth weights, and the remaining 20 (3.8%) were macrosomic. The majority of the newborns (479 (91.6%)) were younger than 24 hours. Approximately 408 (78%) of the newborns did not cry immediately after delivery (Table 2).

Clinical and laboratory characteristics of the study participants

Among the 523 neonates included in this study, 224 (42.8%) were admitted with stage II disease. Among neonates with stage 3 HIE, 44.2% (42 out of 95) were censored. More than half of the study subjects had altered consciousness (56.4%) and a depressed Moro reflex (56.8%) at admission. Among neonates admitted with PNAs, 64 (12.2%), 45 (8.6%) with hyperbilirubinemia and 41 (7.8%) with sepsis were the most frequently encountered medical complications during their hospital stay. The mean $\pm$ SD white blood cell counts among neonates who recovered from PNA were 14.64 ± 6.87 and 15.79 ± 8.09 , respectively (Table 3).

Treatment characteristics of the study participants

At birth, 407 (77.8%) newborns admitted with PNA were resuscitated. Nearly half of the 229 (53.8%) of the neonates managed with CPAP survived. The proportions of neonates who were given antibiotics, aminophylline and calcium gluconate were 459 (87.8%), 51 (9.8%) and 212 (40.5%), respectively. Among the total study subjects, 404 (77.2%) neonates were managed with two-thirds fluid (Table 4).

Time to recovery and overall proportion of neonates with asphyxia

Five hundred twenty-three admitted asphyxiated newborns were monitored for a total of 3682 neonate-days, which ranged from a minimum of 1 day to a maximum of 28 days. Among the 523 patients included in the study, 376 [71.9% (95% CI: 67.8-75.6)] recovered, whereas 147 (28.1%) were censored. Among the censored neonates, 88 (16.8%) died, 51 (9.8%) were referred and/or transferred out, and 8 (1.5%) left against medical advice. The overall incidence density rate of surviving neonates was 10 per 100 neonate-days of observation (95% CI: 9.24-11.31). Seven days (95% CI: 6.61-7.39) was the median recovery time. The estimated cumulative survival rates at 1, 3, 7, 14, 21, and 28 days were 0.97, 0.93, 0.47, 0.13, 0.04, and 0.0, respectively (Figure 1).

Time to recovery among different groups of asphyxiated neonates

Neonates identified with stage I HIE recovered faster (5 days with 95% CI: 4.65-5.35) than did neonates diagnosed with stage II and III HIE (9 days 95% CI: 8.30-9.69) and (11 days 95% CI: 8.27-13.72), respectively. Compared with neonates who had a normal reflex, those who had a depressed Moro reflex during admission experienced delayed recovery from asphyxia (9 days 95% CI: 8.12-9.87 versus. 7 days 95% CI: 6.65-7.34). Compared with neonates managed with total fluids, the median recovery time for neonates managed with 2/3 intravenous fluid was longer (8 days 95% CI: 7.38-8.61 versus 6 days 95% CI: 5.41-6.58). Compared with neonates who received calcium gluconate, those who did not receive calcium gluconate recovered faster (7 days 95% CI: 6.54-7.46) and (9 days 95% CI: 7.97-10.02), respectively (Figures 2 and 3).

Predictors of the survival status of asphyxiated neonates

Multivariate analysis revealed that newborns who were admitted with stage I hypoxicemic ischaemic encephalopathy (HIE) were 2.5 times more likely to recover faster from asphyxia than neonates admitted with stage III HIE were (AHR: 2.5, 95% CI: 1.65-5.87). Compared with their counterparts, neonates who had a depressed Moro reflex were 48% less likely to recover earlier from asphyxia. (AHR: 0.52, 95% CI: 0.38-0.87). Neonates who had not received calcium gluconate were 1.4 times more likely to recover faster from asphyxia than those who had received calcium gluconate (AHR: 1.40, 95% CI: 1.02-1.68). On the other hand, neonates who were managed with 2/3 of intravenous fluids during their hospital stay were 32% less likely to recover earlier from asphyxia than were neonates who were managed with total fluids (AHR: 0.68, 95% CI: 0.53-0.99) (Table 5).

Table 1: Sociodemographic characteristics of mothers admitted to asphyxiated neonates at North Shoa public hospitals, Ethiopia, 2020-2023 (n=523), 2024.

Covariates	Category	Total, N (%)	Status, N (%)	
			Recovered	Censored
Maternal age (years)	<20	64 (12.2)	37 (57.8)	27 (42.2)
	20-34	393 (75.1)	287 (73.0)	106 (27.0)
	>34	66 (12.7)	52 (78.8)	14 (21.2)
Place of residence	Urban	218 (41.7)	169 (77.5)	49 (22.5)
	Rural	305 (58.3)	207 (67.9)	98 (32.1)
Parity	Primi-parous	278 (53.2)	199 (71.6)	79 (28.4)
	Multi parous	245 (46.8)	177 (72.2)	68 (27.8)
Gravidity	Primi-gravida	269 (51.4)	194 (72.1)	75 (27.9)
	Multi gravida	254 (48.6)	182 (71.7)	72 (28.3)
ANC follow up	No	11 (2.1)	6 (54.5)	5 (45.5)
	Yes	512 (97.9)	370 (72.3)	142 (27.7)
Place of delivery	Inborn	303 (57.9)	221 (72.9)	82 (27.1)
	Out born	220 (42.1)	155 (70.5)	65 (29.5)
Time of delivery	Day-time	252 (48.2)	210 (83.3)	42 (16.7)
	Night-time	271 (51.8)	166 (61.3)	105 (38.7)
Mode of delivery	SVD	263 (50.3)	199 (75.7)	64 (24.3)
	C/S	140 (26.7)	94 (67.1)	46 (32.9)
	Assisted delivery	120 (23.0)	83 (69.2)	37 (30.8)
Duration of labor	Normal	461 (88.1)	347 (75.3)	114 (24.7)
	Prolonged	62 (11.9)	29 (46.8)	33 (53.2)
Duration of ROM (hours)	<18	485 (92.7)	353 (72.8)	132 (27.2)
	>18	38 (7.3)	23 (60.5)	15 (39.5)
History of abortion	Yes	55 (10.5)	40 (71.8)	15 (28.2)
	No	468 (89.5)	336 (72.7)	132 (27.3)
Obstetrics and medical condition	Anemia	32 (6.1)	24 (75.0)	8 (25.0)
	APH	12 (2.3)	8 (66.7)	4 (33.3)
	Fetal distress	149 (28.5)	97 (65.1)	52 (34.9)
	Gestational DM	27 (5.2)	17 (63.0)	10 (37.0)
	Pre/eclampsia	50 (9.5)	36 (72)	14 (28.0)
	Others	39 (7.5)	24 (61.5)	15 (38.5)
	No medical complication	214 (40.9)	170 (79.4)	44 (20.6)

Table 2: Neonatal characteristics of asphyxiated babies who were admitted to the NICU of North Shoa public hospitals, Ethiopia, 2020–2023 (n=523), 2024.

Covariates	Category	Total, N (%)	Status, N (%)	
			Recovered	Censored
Sex of the neonates	Male	301 (57.5)	224 (74.4)	77 (25.6)
	Female	222 (42.5)	152 (69.5)	70 (31.5)
Gestational age	Preterm	76 (14.5)	56 (73.7)	20 (26.3)
	Term	390 (74.6)	284 (72.8)	106 (27.2)
	Post term	57 (10.9)	36 (63.2)	21 (36.8)
Birth weight	Low birth weight	121 (23.1)	86 (71.1)	35 (28.9)
	Normal birth weight	382 (73.1)	277 (72.5)	105 (27.5)
	Macrosomic baby	20 (3.8)	13 (65.0)	7 (35.0)
Postnatal age at presentation (hours)	<24	479 (91.6)	347 (72.4)	132 (27.6)
	>24	44 (8.4)	29 (65.9)	15 (34.1)
Cried immediately after birth	No	408 (78.0)	281 (68.9)	127 (31.1)
	Yes	115 (22.0)	95 (82.6)	20 (17.4)
APGAR ¹ score at 1 st min	<7	494 (94.5)	351 (71.1)	143 (28.9)
	≥7	29 (5.5)	25 (86.2)	4 (13.8)
APGAR score at 5 th min	<7	385 (73.6)	267 (69.4)	118 (30.6)
	≥7	138 (26.4)	109 (79.0)	29 (21.0)

Table 3: Clinical and laboratory characteristics of the study participants admitted to public hospitals in North Shoa, Ethiopia (2020–2023), 2024 (n=523).

Variables	Category	Frequency, N (%)	Status, N (%)	
			Recovered	Censored
HIE stage at admission	Stage I	204 (39.0)	165 (80.9)	39 (19.1)
	Stage II	224 (42.8)	158 (70.5)	66 (29.5)
	Stage III	95 (18.2)	53 (55.8)	42 (44.2)
Level of consciousness	Normal	228 (43.6)	191 (83.8)	37 (16.2)
	Altered	295 (56.4)	185 (62.7)	110 (37.3)
Moro reflex	Normal	226 (43.2)	185 (81.9)	41 (18.1)
	Depressed	297 (56.8)	191 (64.3)	106 (35.7)
Medical complication developed during their hospital stay	Hyperbilirubinemia	45 (8.6)	34 (75.5)	11 (24.5)
	Necrotizing enterocolitis	12 (2.3)	10 (83.3)	2 (16.7)
	Seizure	64 (12.2)	28 (43.7)	36 (56.3)
	Sepsis	41 (7.8)	15 (36.6)	26 (63.4)
	Thrombocytopenia	16 (3.1)	9 (56.3)	7 (43.7)
	No	320 (61.2)	246 (76.9)	74 (23.1)
	Others	25 (4.8)	8 (32.0)	17 (68.0)
Laboratory investigations within 24 hours of age (mean±SD)	WBC (×103/l), n=498	14.95±7.23	14.64±6.87	15.79±8.09
	Platelet (mc/l), n=499	162±53	168±54	145±46
	Haematocrit (%), n=498	45.7±8.6	46.1±8.7	44.7±8.2
	Serum creatinine in mg/dl, n=368	0.65±0.33	0.62±0.34	0.72±0.32
	Sodium (mmol/l), n=357	136.9±6.14	136.7±3.03	137.5±11.03
	Potassium (mmol/l), n=356	4.86±0.93	4.84±0.90	4.92±1.01
	Chloride (mmol/l), n=357	98.55±6.26	98.78±3.63	97.88±10.75
	Ca ² (ion) (mmol/l), n=358	1.13±0.23	1.14±0.24	1.10±0.21

Table 4: Treatment characteristics of the study participants admitted to public hospitals in North Shoa, Ethiopia (2020-2023), 2024 (n=523).

Variables	Category	Frequency, N (%)	Status, N (%)	
			Recovered	Censored
Resuscitation at birth	No	116 (22.2)	94 (81.0)	22 (19.0)
	Yes	407 (77.8)	282 (69.3)	125 (30.7)

Continued.

Variables	Category	Frequency, N (%)	Status, N (%)	
			Recovered	Censored
Antibiotics given to the baby	No	64 (12.2)	41 (64.1)	23 (35.9)
	Yes	459 (87.8)	335 (73)	124 (27.0)
Is Aminophylline given to the baby?	No	472 (90.2)	339 (71.8)	133 (28.2)
	Yes	51 (9.8)	37 (72.5)	14 (27.5)
Oxygen type administered to the baby	Direct O ₂	294 (56.2)	255 (86.7)	39 (13.3)
	CPAP	229 (53.8)	121 (52.8)	108 (47.2)
Fluid management	Two third	404 (77.2)	283 (70.0)	121 (30.0)
	Total fluid	119 (22.8)	93 (88.2)	26 (21.8)
Calcium gluconate with in first day of life?	No	212 (40.5)	160 (74.5)	52 (25.5)
	Yes	311 (59.5)	216 (69.5)	95 (30.5)

Table 5: Bivariate and multivariate cox regression analysis results of asphyxiated neonates who were admitted in at NICU of public hospitals of North Shoa, Ethiopia, 2024 (n=523).

Covariates	Category	Survival status, N (%)		CHR (95% CI)	AHR (95% CI)	P value
		Recovered	Censored			
Sucking reflex	Absent	122 (66.3)	62 (33.7)	0.59 (0.46-0.76)	0.70 (0.46-1.07)	0.103
	Weak	124 (70.9)	51 (29.1)	0.67 (0.52-0.86)	0.77 (0.54-1.11)	0.176
	Strong	130 (79.3)	34 (20.7)	1	1	
Moro reflex	Normal	185 (81.9)	41 (18.1)	0.54 (0.43-0.66)	0.58 (0.038-0.87)	0.009**
	Depressed	191 (64.3)	106 (35.7)	1	1	
Temperature of the baby at admission (°C)	<36.5	287 (70.5)	120 (29.5)	2.01 (0.85-4.87)	2.13 (0.85-5.33)	0.105
	36.5-37.5	83 (76.9)	25 (23.1)	2.95 (1.22-7.15)	2.35 (0.92-6.03)	0.073
	>37.5	6 (75)	2 (25)	1	1	
Cry immediately at birth	Yes	95 (82.6)	20 (17.4)	1	1	
	No	281 (68.9)	127 (31.1)	0.54 (0.42-0.68)	1.09 (0.64-1.84)	0.745
Age of the baby at presentation (hours)	<24	347 (72.4)	132 (27.6)	0.54 (0.37-0.80)	1.06 (0.67-1.68)	0.794
	>24	29 (65.9)	15 (34.1)	1	1	
APGAR score at the 1 st min	<7	351 (71.1)	143 (28.9)	0.50 (0.33-0.76)	0.88 (0.54-1.44)	0.629
	≥7	25 (86.2)	4 (13.8)	1	1	
APGAR score at the 5 th min	<7	267 (69.4)	118 (30.6)	0.60 (0.47-0.75)	1.40 (0.87-2.26)	0.163
	≥7	109 (79.0)	29 (21.0)	1	1	
Tone of the baby at admission	Hypotonic	163 (62.5)	98 (37.5)	0.95 (0.72-1.26)	1.59 (0.92-2.46)	0.058
	Normal	139 (84.2)	26 (15.8)	1.78 (1.34-2.38)	1.28 (0.89-1.85)	0.175
	Hypertonic	74 (76.3)	23 (23.7)	1	1	
Resuscitation during hospital stay	Yes	182 (71.9)	71 (28.1)	1	1	
	No	194 (71.8)	76 (28.2)	1.57 (1.28-1.94)	1.11 (0.85-1.45)	0.580
Fluid management	Two third	283 (70.0)	121 (30.0)	0.47 (0.37-0.61)	0.72 (0.53-0.99)	0.043*
	Total fluid	93 (88.2)	26 (21.8)	1	1	
Calcium gluconate at the first day of life	Yes	160 (74.5)	52 (25.5)	1	1	
	No	216 (69.5)	95 (30.5)	1.46 (1.18-1.80)	1.31 (1.02-1.68)	0.035*
Resuscitation at birth	Yes	282 (69.3)	125 (30.7)	1	1	
	No	94 (81.0)	22 (19.0)	2.12 (1.66-2.71)	1.42 (0.86-2.34)	0.161

**P value statistically significant

Test of proportional hazard assumption and overall model fitness

In this study, goodness of fit was checked both statistically and graphically via the Cox-Snell residuals plot to assess the assumption of the Cox proportional hazard model. The Schoenfeld residual proportional hazard test was used for each covariate, and the global test was used. A p value of

>0.05 for each covariate indicates that the assumption is accepted and that the covariate is fitted to the model. The table below shows that each covariate has a p value >0.05 and that the global test p value is 0.1209, which is also >0.05. Therefore, the proportional hazard assumption was met. For the Snell residual test, the hazard function follows 45° close to the baseline hazard, which indicates that the model was better fitted (Figure 4).

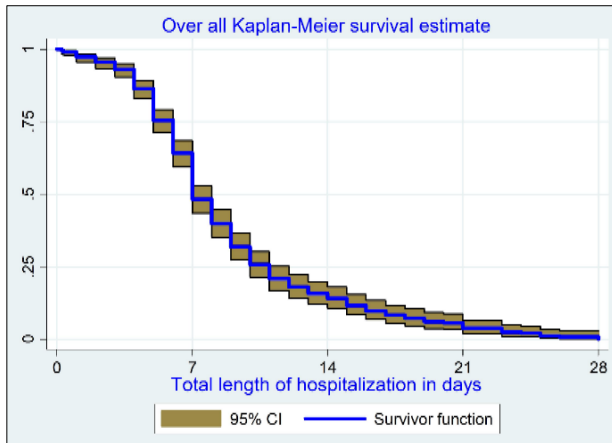


Figure 1: Overall Kaplan-Meier survival estimate of asphyxiated neonates admitted in public hospitals of North Shoa, Amhara, Ethiopia (2020-2023), 2024.

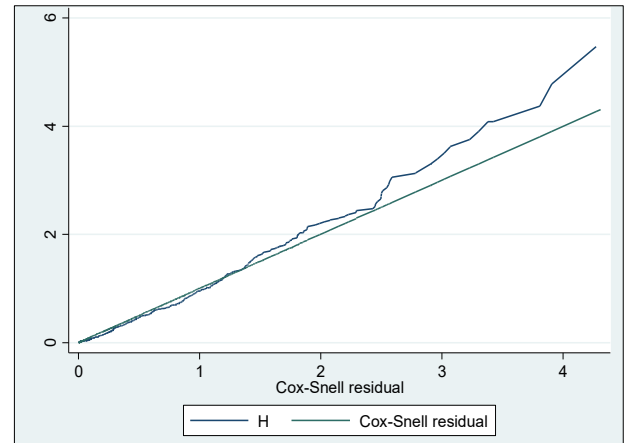


Figure 4: Cox snell residual test for proportional assumption for each covariant and overall cox proportional hazard model, 2024.

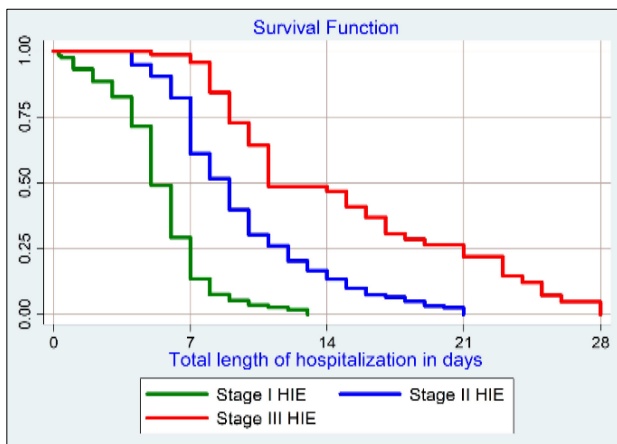


Figure 2: The KM survival curves comparing recovery time with different HIE stages of neonates admitted in public hospitals of North Shoa, Amhara, Ethiopia (2020-2023), 2024.

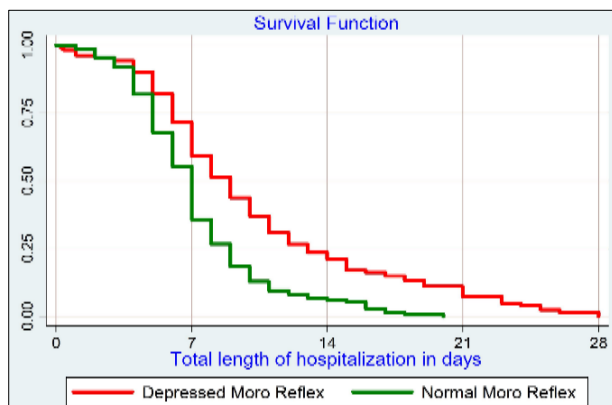


Figure 3: The KM survival curves comparing newborn Moro reflex at admission in public hospitals of North Shoa, Ethiopia, (2020-2023), 2024.

DISCUSSION

Finding the time to recovery and risk factors for birth asphyxia in newborns hospitalized to neonatal critical care units in public hospitals in North Shoa was the aim of this study. Seven days was the median healing period. The time to recover from perinatal asphyxia was found to be independently predicted by the Moro reflex, hydration management, and calcium gluconate administered to the newborn on the first day of life.

Among the 523 asphyxiated neonates included in this study, 71.9% (95% CI: 67.8-75.6) of the neonates recovered. This study is almost comparable with a cohort study conducted in Ethiopia: Addis Ababa public hospitals 74.4% and Dire-Dawa public hospitals 74.3%.^{9,10} However, the current finding is lower than that of a study performed in Debre Berhan comprehensive specialized hospitals, in which 81.8% of asphyxiated neonates recovered.¹¹

Different study settings may be the cause of the disparity in survival rates. The fact that the earlier study was carried out at a fully specialized hospital with cutting-edge medical equipment, resources, and expert medical personnel trained to handle complicated infant diseases may help to explain this. The current study, on the other hand, covered primary and general hospitals where there was a lack of resources, equipment, and qualified medical staff, which could have an impact on the standard of care given. Additionally, there may be delays in moving a newborn to a higher-level facility if their condition worsens in basic and general hospitals, which could have a detrimental effect on their chances of survival.

This study revealed that the median recovery time of asphyxiated newborns was 7 days. This finding was in line with that of a study in Dire-Dawa public hospitals lasting 7 days and comparable with that of a study in India lasting 8 days.^{10,12} In the present study, the median recovery time

was faster (shorter) than that reported by Debrebrehan Comprehensive Specialized Hospital, whose median recovery time was 9 days.¹¹

This disparity may be explained by the fact that primary hospitals frequently admit newborns soon after birth, which enables prompt intervention and early treatment for asphyxiated infants. This is because primary hospitals were included in the current study. On the other hand, referrals to specialized facilities are usually delayed, which might worsen a neonate's condition.

Additionally, neonates admitted to specialized hospitals are frequently those with more severe asphyxia or complications that cannot be managed at the primary or general hospital level. As a result, patients seen in specialized hospitals may require a longer recovery time. However, the median recovery time was longer than that reported in studies in Nigeria (3.8 days).¹³ The reason behind the inconsistency in the recovery time in infants with birth asphyxia in different studies is due to the nature of the studies' setting, the type of patients used in each study, and the organization of health services. The majority of the studies are conducted in specialized teaching hospitals or tertiary referral hospitals having highly developed neonatal facilities. Due to better availability of facilities and improved management and protocol, good results are achieved. Moreover, specialized hospitals get the difficult cases, and due to that reason, the recovery period becomes long.

The results of this study revealed that, depending on the stage of HIE, the recovery and survival rates of newborn individuals who were asphyxiated varied. Neonates admitted with stage I HIE recovered 2.5 times faster from asphyxia than did neonates admitted with stage III HIE (AHR: 2.5, 95% CI: 1.65-5.87). This result was supported by other finding.¹⁴ The scientific explanation for this finding could be that stage I HIE involves mild brain injury caused by hypoxia, which allows for quicker recovery. In addition to this stage, I HIE typically does not cause significant systemic complications, such as organ dysfunction, which is more common in stage III HIE. Additionally, the management and care of neonates are frequently inadequate or ineffective when multiple organs are damaged.¹⁵

According to this study, neonates with a depressed Moro reflex take longer to recover from asphyxia than those with normal Moro reflexes do (AHR: 0.52, 95% CI: 0.38-0.87). These findings were consistent with other findings reported in the Debrebrehan Comprehensive Specialized Hospital.¹¹ Delayed neurological recovery in neonates with a depressed Moro reflex can point to the presence of more severe neurological damage secondary to hypoxic-ischemic encephalopathy. The Moro reflex is an important indicator of neurological health; the depression of the Moro reflex indicates significant impairment in the functioning of the central nervous system and implies the

presence of a significant extent of hypoxic-ischemic brain damage. Such a problem causes the delay in neurological recovery and stabilization of neonates, who also suffer from breathing problems, heart malfunctioning, low muscle tone, and circulatory adaptation problems. Consequently, a depressed Moro reflex becomes a sign of neurological damage and general severity of the condition, resulting in longer recovery time than in other neonates with a normal Moro reflex.^{16,17}

The current study revealed that neonates who had not received calcium gluconate within the first day of life recovered faster from asphyxia than those who had received calcium gluconate (AHR: 1.40, 95% CI: 1.02-1.68). These findings contradict those of a study conducted in Addis Ababa.⁹ This finding can be scientifically explained by the fact that calcium gluconate can alter metabolic processes, potentially interfering with the baby's ability to respond to hypoxic conditions. On the other hand, calcium gluconate can affect cardiac function, possibly leading to complications that hinder recovery.

Finally, this study revealed that neonates who were managed with 2/3 of intravenous fluids during their hospital stay were less likely to recover earlier from asphyxia than neonates who were managed with total fluids were (AHR: 0.68, 95% CI: 0.53-0.99). These findings were supported by a study performed in India.¹⁸ The scientific explanation for this might be that asphyxia can lead to hypovolemia and reduce perfusion, and insufficient fluid can compromise circulation and oxygen delivery to vital organs. Fluid therapy is crucial for maintaining electrolyte balance. Adequate fluid can lead to imbalances that negatively affect cardiac and neurological function. Fluid restriction may also have an impact on metabolism; adequate fluid support helps clear waste products, and limited fluid can slow these processes, which can delay recovery from asphyxia.^{19,20}

CONCLUSION

This study revealed that neonates with birth asphyxia recovered during their stay in neonatal intensive care units, with a median recovery time of seven days. The key predictors of survival include stage I hypoxemic ischemic encephalopathy, having a depressed Moro reflex at admission, not receiving calcium gluconate within 24 hours, and neonates managed with 2/3 intravenous fluid.

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