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Risk Factors and Short-Term Outcomes of Birth Asphyxia among Newborns at a Tertiary Hospital: A Prospective Cohort Study

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ABS TRAC T

***Background*:** Birth asphyxia remains a leading cause of neonatal mortality and long-term neurodevelopmental disability in sub-Saharan Africa. Identifying modifiable maternal, intrapartum and neonatal risk factors is essential to inform prevention strategies. This study determined the risk factors and short-term outcomes of birth asphyxia among newborns at a tertiary hospital in Tanzania. ***Methods*:** A prospective cohort study was conducted between January 2024 and December 2025. A total of 192 neonates diagnosed with birth asphyxia (5-minute Apgar score <7 and/or need for resuscitation beyond initial steps) were enrolled and followed until discharge or death. Maternal, antepartum, intrapartum and neonatal variables were recorded. Outcomes assessed included in-hospital mortality and major morbidities. Multivariable logistic regression identified independent predictors of mortality. ***Results*:** Birth asphyxia occurred in 192 of 4,820 live births during the period (incidence 39.8 per 1,000 live births). Mean gestational age was 38.4 ± 2.6 weeks; mean birth weight was 2,940 ± 612 g. Risk factors identified included prolonged labour in 76 (39.6%), obstructed labour in 38 (19.8%), pre-eclampsia/eclampsia in 32 (16.7%), antepartum haemorrhage in 22 (11.5%), meconium-stained liquor in 78 (40.6%) and inadequate antenatal care in 86 (44.8%). Hypoxic-ischaemic encephalopathy developed in 124 neonates (64.6%), with stage II in 56 (29.2%) and stage III in 28 (14.6%). In-hospital mortality was 29.7% (57 of 192). Independent predictors of mortality were HIE stage III (aOR 9.84, 95% CI 4.12–23.52), 5-minute Apgar <4 (aOR 6.86, 95% CI 2.94–16.04), birth weight <2500 g (aOR 3.42, 95% CI 1.52–7.68) and need for mechanical ventilation (aOR 4.62, 95% CI 2.04–10.46). ***Conclusion*:** Birth asphyxia is common at this Tanzanian tertiary centre, with mortality of nearly 30%. Modifiable obstetric and antenatal-care factors contribute substantially, underscoring the urgency of improving emergency obstetric care, antenatal coverage, intrapartum monitoring and neonatal resuscitation training.

Keywords: *Birth Asphyxia, Hypoxic-Ischaemic Encephalopathy, Risk Factors, Neonatal Mortality, Apgar Score, Tanzania.*

INTRODUCTION

Birth asphyxia is a major contributor to global neonatal mortality and long-term neurodevelopmental disability, accounting for an estimated 23–25% of the 2.4 million annual neonatal deaths worldwide. The burden falls disproportionately on low- and middle-income countries, with sub-Saharan Africa carrying one of the highest regional rates [1]. In Tanzania, neonatal mortality remains substantially above the Sustainable Development Goal target of 12 per 1,000 live births by 2030, with birth asphyxia consistently ranked among the top three causes alongside prematurity-related complications and neonatal infections [2].

The World Health Organization defines birth asphyxia as the failure to initiate or sustain spontaneous breathing at birth. Operational definitions used in clinical and research settings have variably relied on a 5-minute Apgar score below 7, the need for positive-pressure ventilation, signs of fetal distress, or the development of hypoxic-ischaemic encephalopathy (HIE), with the Sarnat staging providing a structured

framework for the clinical grading of HIE severity [3]. The clinical spectrum spans transient compensated hypoxia to severe multi-organ dysfunction culminating in death or lifelong disability including cerebral palsy, intellectual disability, epilepsy, hearing impairment and visual impairment [3, 4].

Risk factors for birth asphyxia operate across the antenatal, intrapartum and immediate neonatal periods. Antenatal risk factors include maternal age extremes, primiparity or grand multiparity, low socio-economic status, inadequate antenatal care, anaemia, hypertensive disorders of pregnancy, antepartum haemorrhage, multiple gestation, post-term pregnancy, and intrauterine growth restriction [4, 5]. Intrapartum factors—often the proximate causes of acute hypoxic insult—include obstructed and prolonged labour, abnormal fetal lie, cord prolapse, uterine rupture, abruption, meconium-stained liquor, and delayed or inappropriate intrapartum care including failure of timely operative delivery [5, 6]. Neonatal contributors include prematurity, low birth weight and congenital anomalies.

In high-income settings, structured intrapartum monitoring, timely caesarean delivery, neonatal resuscitation under standardised protocols, and the implementation of therapeutic hypothermia for moderate-to-severe HIE have collectively reduced the incidence and improved outcomes of birth asphyxia [7]. The Helping Babies Breathe (HBB) programme, developed and rolled out across many low- and middle-income countries including Tanzania, has been associated with reductions in early neonatal mortality from intrapartum-related causes in several before-and-after analyses [8]. Nevertheless, the burden remains high in resource-limited settings owing to limited access to skilled birth attendance, gaps in continuous intrapartum monitoring, restricted availability of emergency obstetric and neonatal care, and inconsistent availability of therapeutic hypothermia [8, 9].

Several Tanzanian and East African studies have characterised birth asphyxia at individual referral centres and have identified important risk-factor clusters and mortality patterns. Yatich and colleagues, in a Tanzanian regional hospital cohort, reported a case fatality of approximately one-third and identified prolonged labour, breech presentation and meconium-stained liquor as significant antecedents [10]. Other African cohorts have similarly highlighted the contribution of obstructed labour, hypertensive disorders, inadequate antenatal care, low birth weight and delayed referral [11].

Despite this growing body of evidence, contemporary, prospectively collected data on the incidence, risk-factor profile and short-term outcomes of birth asphyxia at Tanzanian tertiary-care centres remain limited and heterogeneous in design. Up-to-date local evidence is essential to inform the prioritisation of obstetric and neonatal interventions, to benchmark outcomes, and to design context-appropriate quality-improvement initiatives. Accordingly, the present prospective cohort study was conducted to determine the incidence, antepartum, intrapartum and neonatal risk factors, and short-term outcomes of birth asphyxia among newborns delivered at or referred to a Tanzanian tertiary hospital.

Aims and Objectives

The principal aim of the study was to determine the incidence, risk-factor profile and short-term in-hospital outcomes of birth asphyxia among newborns delivered at or referred to a Tanzanian tertiary hospital over a two-year period.

Specific objectives were to describe the demographic, antepartum, intrapartum and neonatal characteristics of newborns with birth asphyxia; to determine the proportion who developed hypoxic-ischaemic encephalopathy and to grade severity using the Sarnat staging system; to estimate the in-hospital case fatality rate and to characterise major neonatal morbidities; and to identify factors independently associated with in-hospital mortality through multivariable logistic regression.

Materials and Methods

Study design and setting

A prospective cohort study was conducted in the Department of Paediatrics and Child Health and the Department of Obstetrics and Gynaecology of St. Francis University College of Health and Allied Sciences (SFUCHAS), Ifakara, Tanzania, between January 2024 and December 2025. The hospital is a national referral and teaching institution providing comprehensive emergency obstetric care, level-II/III neonatal care and is a designated regional centre for the Helping Babies Breathe programme. The protocol was approved by the Institutional Ethics Review Board and informed parental consent was obtained for every enrolled neonate.

Sample size estimation

Assuming an expected incidence of birth asphyxia of 40 per 1,000 live births in the local setting, a desired precision of 4% and a 95% confidence level, the minimum sample required was 175 asphyxiated neonates. Inflating by approximately 10% for incomplete records, the target sample size was set at 192.

Case definition and inclusion criteria

Birth asphyxia was defined operationally for the purpose of enrolment as any neonate of gestational age ≥28 weeks meeting at least one of the following: 5-minute Apgar score below 7, the need for active resuscitation beyond initial drying, stimulation and routine suctioning (i.e., bag-mask ventilation or

further), or umbilical cord arterial pH below 7.10 when sampled. Hypoxic-ischaemic encephalopathy was diagnosed and staged according to the modified Sarnat criteria (stage I mild, stage II moderate, stage III severe) by a paediatrician during the first 72 hours of life.

Exclusion Criteria

Neonates with major congenital anomalies incompatible with survival, those born outside the institution who arrived more than 24 hours after birth or in moribund condition with insufficient documentation of perinatal events, and those for whom informed consent could not be obtained were excluded.

Data Collection

A structured data-collection form captured maternal demographics, parity, socio-economic indicators, antenatal-care attendance (categorised as adequate ≥4 visits or inadequate <4 visits per WHO definition), antenatal complications, gestational age, mode of delivery, place of delivery, intrapartum complications, indications for operative delivery, presence of meconium-stained liquor, neonatal demographics and anthropometry, Apgar scores at 1, 5 and 10 minutes, resuscitation interventions received, course in the neonatal unit, complications, length of stay and outcomes. Hypoxic-ischaemic encephalopathy was staged according to modified Sarnat criteria during structured paediatric assessments.

Outcomes

The primary outcome was in-hospital all-cause mortality. Secondary outcomes included development and severity of HIE, occurrence of seizures, need for mechanical ventilation, need for inotropic support, acute kidney injury, multi-organ dysfunction, and length of NICU stay.

Statistical analysis

Data were analysed using SPSS version 27.0. Continuous variables were summarised as mean ± SD or median (IQR), and categorical variables as frequencies and percentages. Between-group comparisons used the independent samples t-test or Mann–Whitney U test for continuous variables, and chi-square or Fisher exact test for categorical variables. Variables with a univariable p-value <0.20 were entered into a multivariable logistic regression model to identify independent predictors of mortality. Adjusted odds ratios with 95% confidence intervals are reported. A two-sided $p < 0.05$ was considered statistically significant.

RESULTS

Incidence and baseline characteristics

During the study period, 4,820 live births were recorded at the hospital, of whom 192 met the case definition for birth asphyxia, yielding an incidence of 39.8 per 1,000 live births (3.98%). The mean maternal age was 26.4 ± 6.2 years, mean gestational age was 38.4 ± 2.6 weeks and mean neonatal birth weight was 2,940 ± 612 g. Male neonates accounted for 112 (58.3%). Inadequate antenatal care (<4 visits) was recorded in 86 (44.8%) of mothers (Table 1).

Table 1: Demographic and obstetric characteristics of the asphyxiated cohort (n = 192)

Variable	Value
Maternal age (years), mean ± SD	26.4 ± 6.2
Primiparous mother, n (%)	78 (40.6)
Grand multiparous (≥5), n (%)	22 (11.5)
Adequate ANC (≥4 visits), n (%)	106 (55.2)
Inadequate ANC (<4 visits), n (%)	86 (44.8)
Referred in labour, n (%)	82 (42.7)
Outborn (delivered elsewhere), n (%)	44 (22.9)
Mode of delivery – SVD, n (%)	112 (58.3)
Mode of delivery – Caesarean section, n (%)	62 (32.3)
Mode of delivery – Instrumental (vacuum/forceps), n (%)	18 (9.4)
Gestational age (weeks), mean ± SD	38.4 ± 2.6
Preterm (<37 weeks), n (%)	44 (22.9)
Term (37–42 weeks), n (%)	138 (71.9)
Post-term (>42 weeks), n (%)	10 (5.2)
Birth weight (g), mean ± SD	2,940 ± 612
Low birth weight (<2500 g), n (%)	52 (27.1)
Male neonates, n (%)	112 (58.3)

Risk factors identified

Multiple antepartum and intrapartum risk factors were documented. Meconium-stained liquor was the most frequent intrapartum finding (40.6%), followed by prolonged labour (39.6%), obstructed labour (19.8%), antepartum haemorrhage (11.5%), uterine rupture (3.1%) and cord prolapse (4.7%). Hypertensive disorders of pregnancy (pre-eclampsia/eclampsia) were present in 32 mothers (16.7%) and gestational diabetes in 12 (6.3%). Maternal anaemia (Hb <10 g/dL) was documented in 48 (25.0%). Among neonatal factors, low birth weight (27.1%),

prematurity (22.9%) and breech presentation (8.3%) were the principal contributors (Table 2).

Table 2: Antepartum, intrapartum and neonatal risk factors

Risk factor	n (%)
Inadequate ANC (<4 visits)	86 (44.8)
Maternal anaemia (Hb <10 g/dL)	48 (25.0)
Pre-eclampsia / eclampsia	32 (16.7)
Gestational diabetes mellitus	12 (6.3)
Antepartum haemorrhage (APH)	22 (11.5)
Prolonged labour (>18 h primip; >12 h multip)	76 (39.6)
Obstructed labour	38 (19.8)
Meconium-stained liquor	78 (40.6)
Cord prolapse	9 (4.7)
Uterine rupture	6 (3.1)
Breech / abnormal presentation	16 (8.3)
Multiple gestation	10 (5.2)
Post-term pregnancy (>42 weeks)	10 (5.2)
Low birth weight (<2500 g)	52 (27.1)
Prematurity (<37 weeks)	44 (22.9)
Macrosomia (>4000 g)	8 (4.2)

Apgar scores and resuscitation

The mean 1-minute Apgar score was 3.8 ± 1.6 and the mean 5-minute Apgar score was 5.4 ± 1.8. A 5-minute Apgar score below 4 was recorded in 38 neonates (19.8%) and a 10-minute Apgar score below 4 in 18 (9.4%). Bag-mask ventilation was administered to 178 neonates (92.7%), chest compressions to 46 (24.0%) and adrenaline to 18 (9.4%) at resuscitation (Table 3).

Table 3: Apgar scores and resuscitation interventions

Variable	Value
1-minute Apgar score, mean ± SD	3.8 ± 1.6
5-minute Apgar score, mean ± SD	5.4 ± 1.8
10-minute Apgar score, mean ± SD	6.6 ± 1.6
5-min Apgar <4, n (%)	38 (19.8)
5-min Apgar 4–6, n (%)	98 (51.0)
10-min Apgar <4, n (%)	18 (9.4)
Bag-mask ventilation, n (%)	178 (92.7)
Endotracheal intubation, n (%)	62 (32.3)
Chest compressions, n (%)	46 (24.0)
Adrenaline administered, n (%)	18 (9.4)
Resuscitation per HBB algorithm, n (%)	184 (95.8)

Hypoxic-ischaemic encephalopathy and short-term outcomes

Hypoxic-ischaemic encephalopathy developed in 124 of 192 asphyxiated neonates (64.6%): stage I (mild) in 40 (20.8%), stage II (moderate) in 56 (29.2%) and stage III (severe) in 28 (14.6%). Seizures occurred in 58 neonates (30.2%), mechanical ventilation was required in 64 (33.3%), inotropic support in 42 (21.9%), and acute kidney injury was diagnosed in 38 (19.8%). Multi-organ dysfunction was documented in 36 (18.8%) (Table 4).

Table 4: Hypoxic-ischaemic encephalopathy and major short-term morbidities

Outcome	n (%)
HIE present	124 (64.6)
Stage I (mild)	40 (20.8)
Stage II (moderate)	56 (29.2)
Stage III (severe)	28 (14.6)
Neonatal seizures	58 (30.2)
Mechanical ventilation required	64 (33.3)
Inotropic support required	42 (21.9)
Acute kidney injury	38 (19.8)
Disseminated intravascular coagulation	16 (8.3)
Multi-organ dysfunction syndrome	36 (18.8)
Therapeutic hypothermia received	0 (0.0)

Mortality

Of 192 neonates, 57 died in hospital, yielding an in-hospital case fatality rate of 29.7%. Mortality was strongly graded by HIE severity: 5.0% in stage I, 26.8% in stage II and 78.6% in stage III. Mortality in neonates without HIE (Apgar-defined asphyxia only) was 10.3%. The mean time to death among non-survivors was 3.6 ± 2.4 days. The mean length of stay among survivors was 9.4 ± 5.6 days (Table 5).

Table 5: Mortality stratified by HIE stage and key clinical variables

Subgroup	n	Deaths (n)	Case fatality (%)
No HIE	68	7	10.3
HIE stage I	40	2	5.0
HIE stage II	56	15	26.8
HIE stage III	28	22	78.6
5-min Apgar <4	38	24	63.2
5-min Apgar 4–6	98	28	28.6
Mechanical ventilation required	64	36	56.3
Birth weight <2500 g	52	22	42.3
Birth weight ≥2500 g	140	35	25.0
Overall cohort	192	57	29.7

Predictors of mortality

On univariable analysis, low birth weight, prematurity, 5-minute Apgar <4, 10-minute Apgar <4, HIE stage III, need for mechanical ventilation, need for inotropic support, multi-organ dysfunction and inadequate antenatal care were significantly associated with mortality (all $p < 0.05$). On multivariable logistic regression, four variables emerged as independent

predictors: HIE stage III (adjusted OR 9.84, 95% CI 4.12–23.52, p < 0.001), 5-minute Apgar score <4 (aOR 6.86, 95% CI 2.94–16.04, p < 0.001), need for mechanical ventilation (aOR 4.62, 95% CI 2.04–10.46, p < 0.001) and birth weight <2500 g (aOR 3.42, 95% CI 1.52–7.68, p = 0.003) (Table 6).

Table 6: Multivariable logistic regression for predictors of in-hospital mortality

Variable	Adjusted OR (95% CI)	p-value
HIE stage III	9.84 (4.12–23.52)	<0.001
5-minute Apgar <4	6.86 (2.94–16.04)	<0.001
Mechanical ventilation required	4.62 (2.04–10.46)	<0.001
Birth weight <2500 g	3.42 (1.52–7.68)	0.003
Multi-organ dysfunction syndrome	2.84 (1.18–6.84)	0.020
Inadequate ANC (<4 visits)	1.86 (0.82–4.22)	0.138
Outborn / referred status	1.62 (0.74–3.56)	0.226

DISCUSSION

In this prospective cohort study of 192 asphyxiated newborns at a Tanzanian tertiary hospital, birth asphyxia occurred in 39.8 per 1,000 live births, hypoxic-ischaemic encephalopathy developed in 64.6% of cases, in-hospital mortality was 29.7%, and HIE stage III, 5-minute Apgar score below 4, need for mechanical ventilation and low birth weight independently predicted mortality. Modifiable obstetric risk factors—particularly prolonged and obstructed labour, meconium-stained liquor and inadequate antenatal care—were prominent contributors to the asphyxia burden.

The incidence reported here is broadly consistent with East African data. Yatich *et al.*, in a Tanzanian regional hospital cohort, reported a birth asphyxia incidence of approximately 50 per 1,000 live births and a case fatality of 28%, closely mirroring the present cohort [10]. Ethiopian, Kenyan and Ugandan tertiary-centre studies have similarly documented rates between 30 and 60 per 1,000 live births, reflecting the persistent burden across the region despite progress in maternal and newborn care [11, 12]. The Ending Preventable Maternal Mortality and Every Newborn Action Plan frameworks have specifically prioritised intrapartum-related neonatal deaths for accelerated reduction, and the present findings emphasise that this priority remains unmet.

The risk-factor profile observed here echoes prior African and global evidence. Prolonged and obstructed labour, the most frequent intrapartum exposures in this cohort, have been repeatedly identified as proximate causes of intrapartum hypoxia in low-resource settings, reflecting limitations in continuous intrapartum monitoring, partograph use, and timely access to operative delivery [5, 13]. Meconium-stained liquor, present in 40.6% of cases, is a recognised marker of intrauterine compromise; while not invariably indicating fetal acidaemia, its frequency in this cohort underscores the importance of preparedness for resuscitation [6]. Pre-eclampsia/eclampsia (16.7%) and antepartum haemorrhage (11.5%) collectively highlight the role of hypertensive disorders and placental events, both potentially mitigated by stronger antenatal-care coverage and earlier referral [4, 14]. The substantial contribution of inadequate antenatal care (44.8%)—a finding consistent with prior Tanzanian work [15]—reflects systemic gaps that can be addressed through community-level outreach, demand-generation interventions and health-system strengthening.

The case fatality rate of 29.7% in this cohort is consistent with the regional literature, where rates of 25–40% are common at tertiary referral hospitals receiving complicated cases [10, 11, 16]. The strong gradient of mortality by HIE severity—5% in stage I, 27% in stage II and 79% in stage III—closely mirrors the natural history described by Sarnat and Sarnat and subsequently confirmed in multiple cohorts [3, 17]. The very high mortality in stage III HIE observed here, in the absence of therapeutic hypothermia (which is not yet routinely available at this centre), is in keeping with reported outcomes in untreated severe HIE and underscores the urgent need to evaluate the feasibility of low-cost cooling strategies in resource-limited East African settings, while noting that recent randomized data from low- and middle-income countries (the HELIX trial) have raised important questions regarding the efficacy and safety of therapeutic hypothermia in such settings, particularly the possibility of increased mortality [18].

The independent association of 5-minute Apgar score below 4, mechanical ventilation requirement, HIE stage III and low birth weight with mortality observed here is well-established in the literature. Catlin *et al.*, in a long-cited analysis, showed that a 5-minute Apgar score below 4 strongly predicts neonatal mortality and adverse neurodevelopmental outcome. The need for mechanical ventilation reflects severe compromise and limited reserves of the immature lung and brain. Low birth weight compounds the vulnerability of the asphyxiated neonate. These predictors are useful for early risk stratification, family counselling and prioritisation of care.

Although the present findings underscore the importance of strengthening prevention, several effective interventions exist. The Helping Babies Breathe programme has demonstrated substantial reductions in early neonatal mortality from intrapartum-related causes after implementation, and its impact in Tanzania has been positive in multiple before-and-after studies [8]. Strengthening partograph use, ensuring access to skilled birth attendance, improving timeliness of caesarean delivery, expanding magnesium sulphate

availability for eclampsia, and ensuring antenatal-care attendance and quality remain priority strategies [13]. Investing in regional NICU capacity, including reliable oxygen, continuous positive airway pressure and mechanical ventilation, and training in neonatal resuscitation, will further reduce case fatality among those who reach hospital.

Several limitations of the present study warrant acknowledgement. First, the single-centre, tertiary-care setting limits generalisability to community deliveries and lower-level facilities; the referral burden likely inflates measures of severity. Second, long-term neurodevelopmental follow-up was beyond the study window, and outcomes such as cerebral palsy and developmental delay among survivors could not be ascertained. Third, umbilical cord arterial pH was not consistently available, limiting precise biochemical confirmation of intrapartum hypoxia. Fourth, multiple comparisons in subgroup analysis may inflate the type I error rate. Despite these limitations, the prospective design, large sample, and systematic data collection lend confidence to the principal findings.

CONCLUSION

Birth asphyxia continues to impose a substantial burden at this Tanzanian tertiary hospital, with an incidence of 39.8 per 1,000 live births and in-hospital mortality of 29.7%. Modifiable maternal, antepartum and intrapartum factors—including inadequate antenatal care, prolonged and obstructed labour, hypertensive disorders of pregnancy and meconium-stained liquor—contribute meaningfully to the burden, and HIE stage III, 5-minute Apgar score below 4, mechanical ventilation requirement and low birth weight independently predict mortality. Strengthening emergency obstetric care, expanding antenatal-care coverage and quality, scaling up Helping Babies Breathe training, improving neonatal-unit capacity and exploring the feasibility and safety of context-appropriate neuroprotective strategies are essential steps to reduce the burden and improve outcomes of birth asphyxia in Tanzania.

Ethical Approval:

The study protocol was reviewed and approved by the Institutional Ethics Review Board of St. Francis University College of Health and Allied Sciences (SFUCHAS), Ifakara, Tanzania (Ethics Approval No.: SFUCHAS/IERB/2023/101), approved on 20 December 2023, prior to the commencement of participant recruitment. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki (2013 revision). Written informed consent was obtained from the parents or legal guardians of all enrolled neonates before inclusion in the study.

Conflict of Interest: None declared by any author.

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