IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 18, Issue 3 Ser. 16 (March. 2019), PP 41-45
www.iosrjournals.org

Clinico-Biological Profile of Neonates with Birth Asphyxia in RIMS Hospital

Dr. T. Kambiakdik,[1] Dr. Ch. Shyamsunder,[2] Dr. Sunanda Wahengbam,[3] Dr. L. Shaini[4]

[1,3] *Senior Resident,* [2]*Associate Professor, Department of Pediatrics*
[4]*Associate Professor, Department of Biochemistry*
Regional Institute of Medical Sciences, Imphal
Corresponding author : Dr. Ch. Shyamsunder

Abstract
Introduction: *Birth asphyxia is one of the major causes of perinatal mortality and morbidity with the propensity to have life-long implications, in both developing and developed countries. In many of the cases, the brain may be the only organ affected following birth asphyxia, but multiorgan dysfunction and impaired electrolyte derangements also occur as a result of systemic hypoxia and ischaemia.* ***Aim****: To determine the incidence of birth asphyxia and study the clinical features and the biochemical abnormalities associated with it* ***Materials and methods*** *: In this prospective cross sectional study conducted between October 2103 to September 2015, term newborns with an Apgar score <3 at the 1st min. or <7 at 5 mins. admitted in the Neonatology unit during the study period were included for the study and those with Apgar score > 3 at the 1st min. of >7 at 5 min., born with congenital anomalies, cardio- respiratory compromise, sepsis, gestational age < 37 weeks of gestation and parents/guardians declining consent were excluded.* ***Results****: Seventy neonates fulfilling the inclusion criteria were eligible for the study. 21(30%) neonates were in Stage I HIE; 32(45.7%) and 17 (24.3%) were in Stage II and III respectively. Seizures were the commonest presentation (70%), followed by respiratory distress (50%) and shock (40%). Subtle seizures (75.5%) were the predominant type. Hypoglycemia (31.7%) hypocalcemia (30.2%) and hyponatremia (25.4%) were the major electrolyte derangements. Cardiac dysfunction, as manifested by raised CK-MB (76.2%), Troponin I (69.8%) and Troponin T (30.2%) was a prominent feature.* ***Conclusion:*** *HIE is the most important serious manifestation of birth asphyxia, but various other organs are also affected along with significant electrolyte imbalances in relation to the severity of asphyxia.*

Key words: *Birth asphyxia, neonate, seizure*

Date of Submission: 20-03-2019 Date of acceptance: 06-04-2019

I. Introduction

Birth asphyxia is a serious clinical problem with the incidence varying from 1 to 1.5% of all live births in developed countries and along with birth injuries together contribute almost 29% of all neonatal deaths.[1] Approximately 4 million babies suffer from birth asphyxia according to the World Health Organisation (WHO) and out of these 1.2 million die and almost equal number develop severe sequelae such as developmental delay, cerebral palsy and epilepsy.[2]

Birth asphyxia is characterized by an impairment of exchange of the respiratory gases (oxygen and carbon dioxide) resulting in hypoxemia and hypercapnia, accompanied by metabolic acidosis[3] and can be caused by events that have their roots in either the antepartum, the intrapartum or the postpartum periods or combination thereof.[4]

When the neonates suffer from asphyxia, almost all the organs may be affected resulting in a series of clinical[5] and biochemical alterations[6] which can affect the outcome, but the most profound effects are observed in the central nervous system. The clinical manifestations of this injury has been termed as Hypoxic Ischaemic Encephalopathy (HIE). HIE is of concern in asphyxiated neonates as it can lead to serious long-term neuro-developmental sequelae among the survivors.[1,2] The involvement of other organs like lungs, cardiac, kidneys or the gastrointestinal tract can cause multi-organ failure in addition to varied metabolic or biochemical abnormalities observed like hyponatremia, hyperkalemia, hypocalcemia, hypomagnesemia, hypoglycaemia, cardiac enzymes and azotemia, which may also indicate a significant relationship with the severity of birth asphyxia.[7,8,9]

In our region too, cases of birth asphyxia are still being encountered. As precise data are lacking, this study was contemplated to determine the incidence, study the clinical features and associated biochemical derangements.

II. Aims And Objectives

- To determine the incidence of birth asphyxia.
- To study the clinical features and the biochemical abnormalities associated with it

III. Materials And Methods

This prospective cross sectional study was conducted in the Departments of Pediatrics, Regional Institute of Medical Sciences (RIMS) Hospital, Imphal, from October 2103 to September 2015 after obtaining clearance from the Institute's Ethics Committee. Term newborns with an Apgar score <3 at the 1st min. or <7 at 5 mins. admitted in the Neonatology unit of the Department of Pediatrics, RIMS during the study period were included for the study and those with Apgar score > 3 at the 1st min. of >7 at 5 min., born with congenital anomalies, cardio- respiratory compromise, sepsis, gestational age < 37 weeks and parents/guardians declining consent were excluded. Apgar score was the criteria used to define birth asphyxia in this study, due to unavailability of blood gas analysis in babies delivered in the hospital.

A detailed clinical examination of all the newborns enrolled for the study were carried out soon after birth. Apgar score at 1 min and 5 mins. were assessed and recorded. A complete history of the mother with regards to the age, parity, period of gestation, mode of delivery and any risk factor for sepsis were noted. Various clinical features in the newborns such as respiratory distress, encephalopathy, cranial nerve and motor abnormalities and seizures were also documented. Neonatal resuscitation was performed according to current neonatal resuscitation protocol (NRP) guidelines.

Haematological parameters – haemoglobin, total and differential leucocyte count, platelet count, immature to total neutrophil (I/T) ratio and micro erythrocyte rate (mESR) were carried out. Blood glucose, serum urea, creatinine, electrolytes – sodium(Na^+), potassium(K^+), calcium(Ca^{++}) and magnesium(Mg^{++}) were determined. Cardiac (Troponin -I and Troponin -T) and neurologic (CK-MB) markers, were measured as required. Electrolyte abnormalities were managed according to unit protocol. The detailed findings were recorded in a pre-designed proforma and data were analyzed.

IV. Results And Observation

During the study period 70 neonates fulfilled the criteria of birth asphyxia out of 725 Neonatal Intensive Care Unit (NICU) admissions giving an incidence of 9.7%. There were 39 (55.7%) male and 31 (44.3%) female babies (M:F= 1.3:1).

Table -1: Profile of mothers of asphyxiated neonates:

Variables		Frequency	Percentage
Maternal age	<20 yrs	11	15.7
	20-30 yrs	54	77.2
	>30 yrs	5	7.1
Ante-natal check up	Booked	47	67.1
	Unbooked	23	32.9
Parity	Primipara	51	72.9
	Multipara	19	27.1
Duration of labour	Prolonged	39	55.7
	Not prolonged	31	44.3
Associated maternal conditions	Anemia	23	32.9
	Hypertension	9	12.9
	Diabetes mellitus	5	7.1
	Hypothyroidism	4	5.7
	Hepatitis	4	5.7

Table -2: Profile of asphyxiated neonates:

Variables		Frequency	Percentage
Gestational age	Term	66	94.3
	Post-term	4	5.7
Birth weight (kg)	<2.5	9	12.9
	2.5 – 4	57	81.4
	>4	4	5.7
Place of delivery	RIMS	58	82.9

	Outside RIMS	12	17.1
Mode of delivery	Vaginal delivery	54	77.1
	Caesarian section	16	22.9

Majority of the newborns were term (94.3%) and delivered in the hospital (82.9%) mostly through the vaginal route (77.1%) [Table -2]. The mean birth weight was 3.20 ± 0.53 kg.

Table 3. HIE staging of asphyxiated neonates

Clinical stage	Number of patients (n=70)	Percentage(%)
Stage I	21	30.0
Stage II	32	45.7
Stage III	17	24.3

Clinical staging of hypoxic ischaemic encephalopathy (HIE), was based on Sarnat.[10] 30% (n=21)were in stage I HIE; 45.7% (n=32) in stage II and 24.3% (n=17) were in stage III[Table -3]. A total of 47 (67.1%) babies presented during the first 6 hours of life; 16 (22.9%) presented within 6 – 12 hours of life; 5(7.1%) within 12 to 24 hours and 2(2.9%) after 24 hours of birth. There were 15 mortalities (5 - HIE II and 10 - HIE III cases).

Table 4: Clinical features of neonates

Clinical details	Number of neonates (N)	Percentage (%)
Seizure	49	70.0
Respiratory distress	35	50.0
Shock	28	40.0
Hypothermia	6	8.6
Oliguria	5	7.1

Among the studied subjects, seizure was the commonest clinical presentation following birth asphyxia [Table – 4]. Of the 70 cases, 49 (70%) patients developed seizures and were observed in the HIE stages II and III cases. The next commonest presentation was respiratory distress (n=35;50%). Clinical evidence of shock was present in 28(40%) while hypothermia was detected in 8.6% of the cases.

Table 5: Types of Seizures

Clinical type	Number of neonates (n=49)	Percentage(%)
Subtle	37	75.5
Tonic	6	12.2
Clonic	5	10.2
Myoclonic	1	2.1

Subtle seizure was the most predominant type comprising of 75.5% (n=37) of all seizures. The other types of seizures were tonic, clonic and myoclonic which constituted 12.2% (n=6), 10.2% (n=5) and 2.1% (n=1) respectively [Table – 5].

Table 6: Biochemical profile of neonates with symptoms of birth asphyxia

Biochemical details	n=63	Percentage(%)
Hypocalcemia	19	30.2
Hyponatremia	16	25.4
Hypoglycemia	20	31.7
Hyperkalemia	5	7.9
CK- MB*	48	76.2
Troponin I	44	69.8
Troponin T	19	30.2
Urea	7	11.1
Creatinine	5	7.9

*CK-MB : Creatine kinase muscle/brain

The biochemical profiles of neonates with symptoms of birth asphyxia are listed (Table -6). Hypocalcemia -19 (30.2%), hypoglycaemia -20 (31.7%)), hyponatremia -16(25.4%) and hyperkalemia -5(7.9%) were the observed electrolyte imbalances. Serum urea and creatinine were elevated in 7(11.1%) and 5(7.9%) cases respectively. Creatine kinase-MB(muscle/brain) was increased in 48 (76.2%) babies; Cardiac Troponin I and Troponin-T were raised in 44(69.8%) and 19(30.2%) respectively.

V. Discussion

In this study, 70 neonates fulfilling the criteria of birth asphyxia constituted (9.7%) of the newborns admitted in the NICU and it was higher than the percentage of 1- 1.5 in all live births in developed countries

having advanced obstetric and neonatal care.[1] Yelamalli BC[11] and Amritanshu[12] also reported similar findings. WHO defined perinatal asphyxia as a failure to initiate and sustain breathing at birth.[13] 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.[14] However, 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]

The male to female ratio in this study was 1.3:1 which was similar to others' findings[11,12] Majority of the mothers were booked (67.1%); primipara mothers comprised of 72.9% and those in 20-30 years age constituted the majority [Table -1]. In primi gravida mothers prolonged labour is usually common, which might be contributing to sustained foetal hypoxia and neonatal asphyxia. Here it was found that 39 (55.7%) were born after prolonged labour. Similar findings were also reported by Shah[16] and Finer.[17]

Seizure was the commonest presentation in the affected neonates, being observed in 49 (70%) all the HIE- II and HIE- III cases. Shah GS[18] had also reported higher numbers of seizures (68%) in their study. Subtle seizure was the most frequent (75.5%) type in this study. Tonic, clonic and myoclonic seizures were present in 12.2%, 10.2% and 2.1% neonates respectively. Seizures may be attributed to cerebral edema, anoxia, trauma or other various metabolic complications. Hypoglycemia, hypocalcemia, hypomagnesemia and hyponatremia are the main metabolic abnormalities which can cause seizures.[16,17,18] Other significant clinical manifestations were - respiratory distress (50%), shock (40%) and hypothermia (8.6%) respectively which were also comparable to other's reports.[18]

In this study, HIE stages I,II and III were found in 21(30%), 32(45.7%) and 17(24.3%) neonates. Yelamali BC[11] too had observed HIE stage I - 48(56.47%), II - 11(6.74%) and III - 26(30.58%) respectively. 13(21.7%) cases of mild hypoxemic ischemic encephalopathy (HIE),27 (45%) moderate and 20 (33.3%) severe HIE were reported by Shah GS.[18]

The biochemical alterations which were significant in this study are hypoglycaemia, hypocalcemia and hyponatremia. Hypoglycemia was found in 31.7%, hypocalcemia in 30.2% and hyponatremia in 25.4% of the neonates, that was also reported similarly by others.[16,18] The possible attributions to hypoglycemia are poor intake, increased metabolic rate coupled with increased glucose utilisation and impaired glucose mobilization.[19] Hypocalcemia was another important finding in our study. Jajoo[20] also found hypocalcemia in 50% of full term infants with birth asphyxia. In perinatal asphyxia, delayed introduction of feeds, increased calcitonin production, increased endogenous phosphate load, renal insufficiency, and diminished parathyroid hormone secretion all may contribute to hypocalcemia.[21]

Hyponatremia might be the result of fluid overload as a consequence of renal compromise or due to syndrome of inappropriate secretion of anti diuretic hormone.[22] Kumar et al[23] reported hyponatremia in 10 out of 22 (45.45%) cases which was higher compared to our study, while Sood et[24] al have reported a lower incidence (17.24%). Kidney functions were affected in relatively small numbers as serum urea and creatinine were elevated in 7 and 5 cases respectively. Cardiac involvement was more marked as creatine kinase-MB was elevated in 76.2% of the cases; Troponin I and Troponin T were elevated in 69.8% and 30.2% respectively. These finding are comparable to the findings of Rajakumar PS et al[25] in which cardiac enzymes, troponin T and creatine kinase-MB were significantly elevated. Cardiac dysfunction may be due to direct effects of hypoxia and transient myocardial ischaemia.[1]

The total number of deaths observed in this study was 15(21.4%), which was comparable to Yelamali BC(20.4%)[11] but slightly less than 32% observed by Agrawal J et al.[26] Specific outcomes depend on the severity of the encephalopathy. Stage I hypoxic-ischaemic encephalopathy have a 98-100% normal neurologic outcome and less than 1% mortality.[1] In the present study maximum number of deaths occurred in HIE stage III (10 neonates) whereas 5 neonates in HIE stage II died. There were no mortalities in HIE stage I cases. Majority of death occurred in Stages II and III of HIE, showing that once severe hypoxia occurs, treatment cannot be very effective, so more attention needs to be paid to early assessment and intervention, during which intervention might be efficacious in reducing severity of brain damage.[11]

The successful treatment of birth asphyxia depends largely on the early recognition of risk factors, the clinical features as well as precise identification of various associated biochemical parameters. Effective postnatal management of the neurologic effects of asphyxia including ventilation, temperature, perfusion, and

judicious fluid management as well as maintaining the laboratory parameters inside physiologic range will prevent further complications.

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Dr. Ch. Shyamsunder. "Clinico-Biological Profile of Neonates with Birth Asphyxia in RIMS Hospital." IOSR Journal of Dental and Medical Sciences (IOSR-JDMS), vol. 18, no. 3, 2019, pp 41-45.