NEONATAL ASPHYXIA- DAMAGE TO NERVOUS SYSTEM- A STUDY OF 100 CASES

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ABSTRACT

BACKGROUND

As many outborn babies (mostly of unbooked mothers) are admitted in our Medical College NICU, perinatal asphyxia seems to be a major factor leading to neonatal mortality and morbidity. Other than causing damage to different systems of body e.g. cardiovascular and respiratory, damage to nervous system seems to be a significant percentage in our study.

MATERIALS AND METHODS

100 newborn babies were included in our study of varying birth weight, gestational age etc., but all were with history of neglected antenatal care, unattended delivery (mostly home). They presented with different features of e.g. seizures, coma, paresis, brisk jerks etc. Cranial ultrasonography was done in very critical cases. Neonatal sepsis was ruled out by 'Sepsis Screen.'

RESULTS

In a significant percentage of newborn babies with birth asphyxia (not crying after birth), blinking of eyes, apnoeic episodes, all manifestations of seizures, depressed alertness, diminished sucking and other reflexes were found. In cranial ultrasonography, a significant number of cases of periventricular vasodilatation was detected, especially in infants of early gestational age.

CONCLUSION

Elimination of different risk factors was identified in expectant mothers by proper antenatal care and monitoring, timely referral to higher centre, proper intervention will go a long way in preventing nervous system injury in newborn infants.

KEY WORDS

Perinatal Asphyxia; Damage to Nervous System.

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BACKGROUND

Perinatal asphyxia is the state of decreased oxygen delivery to the foetus or neonate resulting in inadequate tissue perfusion. It is the second most common cause of neonatal death after infection. Approximately, 23% of the four million global deaths are attributable to asphyxia.¹ Postasphyxial HIE occurs in about 1 to 2 per 1000 live births.² Significant proportion of these infants die or survive with severe long-term morbidity. In India around 250,000 to 300,000 neonates die each year due to asphyxia, mostly in the first 3 days of life. WHO defined birth asphyxia as "failure to initiate and sustain breathing at birth." And with an Apgar score of < 7 at one minute of life, resulting in impending or actual cessation of apparent life. 90% of asphyxia events occur in the antepartum or postpartum periods as a result of placental insufficiency.³

After an episode of asphyxia and ischaemia, anaerobic metabolism causes an increased amounts of lactate, inorganic phosphates, glutamate as well as free radicals. They are toxic and lead to damage of multiple organs of body like-

- CNS- Hypoxic Ischaemic Encephalopathy (HIE), infarction, intracranial haemorrhage, seizures, cortical atrophy.
- CVS- Myocardial ischaemia, poor contractility, hypotension.
- Pulmonary- Pulmonary hypertension, pulmonary haemorrhage, respiratory distress syndrome.
- Renal- Acute tubular or cortical necrosis.
- Adrenal- Haemorrhage.
- Gastrointestinal- Perforation, ulceration, necrosis.
- Metabolic- SIADH, hypocalcaemia, hyponatraemia, hypoglycaemia, myoglobinuria.
- Integument- Subcutaneous fat necrosis
- Haematology- DIC.

This multiple organ dysfunction is the reason for such mortality and morbidity associated with birth asphyxia.³ Death may occur in neonatal period itself or there may be lifelong handicaps in the form of mental retardation, cerebral palsy, seizure disorder etc. Risk of cerebral palsy is about 5 to 10% compared to 0.2% in general population. Most cerebral palsy is not related to perinatal asphyxia.⁴ Newer modalities of treatment are being developed to minimize the morbidity and mortality. The most promising among them is therapeutic hypothermia. It has been established that both head and body cooling are safe, effective and decrease the risk of brain injury in infants exposed to perinatal asphyxia.⁵

Neonatal Hypoxic-Ischaemic Encephalopathy (HIE) is the term used most frequently to designate the clinical and neuropathologic findings thought to occur in full-term infants following intrapartum or neonatal asphyxia. Hypoxia leads to lactate dependent cellular damage, which is the likely cause

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of brain damage.⁶ The incidence of perinatal hypoxia is about 1.0 to 1.5% of live births in most centres and is inversely related to gestational age and birth weight. It occurs in 9% infants of less than 36 weeks gestation and in 0.5% infants of more than 36 weeks gestation.⁷

CNS Involvement

By and large according to presence of primary signs of acute neonatal encephalopathy, the CNS was the most frequently involved (72%) as documented by Ana Martin et al.⁷ The primary sign of CNS injury following asphyxia included seizures, abnormal respiratory pattern to include apnoea and respiratory arrest, an apparent state of hyperalertness, jitteriness, posturing and movement disorders, impaired suck, swallow, gag and feeding, abnormal oculomotor and pupillary responses, pervasive hypotonia and a bulging of anterior fontanelle. Four neuropathological processes have been associated with perinatal asphyxia; primary

subarachnoid haemorrhage, Hypoxic-Ischaemic Encephalopathy (HIE), periventricular leukomalacia and intraventricular haemorrhage.

Hypoxic-Ischaemic Encephalopathy

If the full-term brain has been compromised during delivery, it is likely that the infant will show clinically evident disturbance in neurological behaviour, a state referred to as hypoxic-ischaemic encephalopathy. Infants showing encephalopathic behaviour depending upon the severity and duration of asphyxia event, which includes alteration in alertness, muscle tone and respiration. These clinical features were first graded by Sarnat and Sarnat.⁸

(Modified from Sarnat H; Sarnat M, Neonatal Encephalopathy: Arch Neurol 1976;33 (10)696-705).⁸

STAGE	STAGE 1 (Mild)	STAGE 2 (Moderate)	STAGE 3 (Severe)
Level of consciousness	Hyperalert, irritable	Lethargic or obtunded	Stuporous, comatose
Neuromuscular control	Uninhibited, overactive	Diminished spontaneous movement	Diminished or absent spontaneous movement
Muscle tone	Normal	Mild hypotonia	Flaccid
Posture	Mild distal flexion	Strong distal flexion	Intermittent decerebration
Stretch reflexes	Overactive	Overactive, disinhibited	Decreased
Segmental myoclonus	Present or absent	Present	Absent
Complex reflexes	Normal	Suppressed	Absent
Suck	Weak	Weak or absent	Absent
Moro	Strong, low threshold	Weak, incomplete, high	Absent
Oculo-vestibular	Normal	Overactive	Weak or absent
Tonic Neck	Slight	Strong	Absent
Autonomic function	Generalised sympathetic	Generalised parasympathetic	Both system decreased
Pupil	Mydriasis	Miosis	Mid position/ unequal
Respiration	Spontaneous	Spontaneous; occasional apnoea	Periodic; apnoea
Heart rate	Tachycardia	Bradycardia	Variable
Bronchial & salivary secretion	Sparse	Profuse	Variable
Gastrointestinal motility	Normal/ decreased	Increased, diarrhoea	Variable
Seizures	None	Common focal or multifocal (6-24 hrs age)	Uncommon (excluding decerebration)
EEG findings	Normal	Early: generalised low voltage, slowing Later: periodic pattern; seizures	Early: periodic pattern Later: totally isopotential
Outcome	About 100% normal	80% normal	About 50% die

Neuropathologic patterns of hypoxic-ischaemic cerebral injury/ patterns predominantly found in term infants

- Selective neuronal necrosis.
- Status marmoratus.
- Parasagittal cerebral injury.
- Focal and multifocal ischaemic brain injury.

Patterns predominantly found in premature infants

Periventricular leukomalacia.

MATERIALS AND METHODS

The present study is a descriptive study, carried out in the NICU of MGM Medical College and LSK Hospital, Kishanganj, Bihar.

Inclusion Criteria

Neonates admitted in NICU with-

- History of foetal distress as reported by obstetrician.
- State of neurological depression at birth including late cry after 2 - 3 minutes, limpness, apnoea > 1 minute.
- Need for resuscitative efforts at time of birth.
- Neurological depression afterward during stay in the unit.

Exclusion Criteria

- Neonates with major systemic malformation.
- Those weighing less than 2000 gms at birth.
- Gestational age < 34 weeks from mother's estimation.

A total of 100 neonates were included in the study. All these babies were observed during their stay in NICU and signs and symptoms of various systems involved were noted. Apart from routine investigations like complete blood count, serum electrolytes, renal and liver function tests, these babies were subjected to specialised investigation pertaining to the organ system affected. For example, in neonates with CNS symptoms LP, EEG, USG and CT scan of brain were done. Likewise, those presenting with tachycardia, murmurs, etc. were advised chest X-ray, ECG, Echocardiography etc.

RESULTS

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Birth Weight	No. of Cases	Percentage
2000 - 2499	38	38%
2500 - 2999	46	46%
3000 - 3499	16	16%
Total	100	100%

Table 1. Distribution of Cases according to Birth Weight

The mean birth weight was 2.60 kgs with a Std. deviation of 0.32; 38% of asphyxiated babies were low birth weight, i.e. < 2500 gms. While 62% were of birth weight > 2500 gms.

Place of Delivery	No. of Cases	Percentage
Inborn	19	19%
Outborn	81	81%
Total	100	100%

Table 2. Distribution of Cases according to Place of Delivery

Only 19% of the babies in this study were born in the Obs. Dept. of MGM Medical College, i.e. inborn and the rest were outborn.

Mode of Delivery	No. of Cases	Percentage
Normal Delivery	96	96%
LSCS	04	04%
Total	100	100%

Table 3. Mode of Delivery

In this study, 96% babies delivered at term by normal vaginal delivery. Only 4% were born by Lower Segment Caesarean Section (LSCS).

Organ System Affected	No. of Cases	Percentage
CNS	100	100%
CVS	05	05%
Respiratory	15	15%
GIT	05	05%
Liver	03	03%
Renal	02	02%
Haematology	06	06%

Table 4. Distribution of Organ System Involvement

In this all the babies had involvement of CNS. This was followed by respiratory affection in 15% cases. Involvement of other organ system ranged from 6 - 2%.

Degree of Encephalopathy	No. of Cases	Percentage
HIE I	21	21%
HIE II	56	56%
HIE III	23	23%
Total	100	100%

Table 5. Distribution of Cases according to Degree of Encephalopathy

In this study, majority (56%) babies developed grade 2 hypoxic-ischaemic encephalopathy.

	Normal	NDD	Expired	Attrition	Total
HIE I	20 (95%)	00	00	01 (5%)	21
HIE II	32(57%)	08(14%)	09(16%)	07(12%)	56
HIE III	04(4%)	03(13%)	19(82%)	00	23
Total	53	11	28	08	

Table 6. Relation of Grade of Encephalopathy with Outcome

Among the babies in this study, almost all in HIE I and majority of those in HIE II recovered completely. However, the outcome in babies with HIE III was not good with 82% of them not surviving infancy.

CNS Manifestation	No. of Cases	Percentage
HIE	100	100%
Clinical seizures	74	74%
Hydrocephalus	01	01%
Haemorrhage	01	01%

Table 7. Spectrum of CNS Involvement

Hypoxic-ischaemic encephalopathy was seen in all asphyxiated babies and clinical seizures were observed in 74% cases.

Grade Encephalopathy	Mean Birth Weight (kg)	Standard Deviation
HIE I	2.66	0.38
HIE II	2.63	0.32
HIE III	2.47	0.23

Table 8. Comparison of Birth Weight in different Degree of Encephalopathy

The difference in the mean birth weight between babies with HIE II and HIE III was found to be significant with p-value of 0.017, i.e. < 0.05. Also, the mean birth weight of all the babies in this study was 2.6 kgs and the difference between this and mean birth wt. of babies with HIE III was found to be significant. A p-value of 0.031, i.e. < 0.05.

DISCUSSION

The present study was carried out in the NICU of Dept. of Paediatrics; MGM Medical College and Hospital, Kishanganj, Bihar. A total of 100 asphyxiated babies satisfying the preformed inclusion criteria who were admitted to NICU during the period from November 2014 - December 2015 were studied.

Sample Size

In the present study, the sample size was 100 patients with perinatal asphyxia. This is comparable to the sample size of S. Riphagen⁹ et al (n= 130); V Pierrat¹⁰ et al (n= 90) and G Oswyn¹¹ et al (n= 114). It is smaller as compared to Prakash S Shah¹² et al (n= 375) and Badwani et al (n= 276), but larger than Ong LC¹³ et al (n= 45); K Karam¹⁴ (n= 63) and R Smith¹⁵ et al (n= 45).

Sex Distribution

In this study, male babies constituted 74% of study population against 26% females. This male preponderance may be a marker of still prevailing gender bias in our society. The study conducted by G Oswyn¹¹ et al had 59% males, while V Pierrat¹⁰ et al observed a female preponderance with 53% females. No association was found between sex of babies and incidence of severity of asphyxia.

Mode of Delivery

The majority of babies (90%) in the present study were delivered by the vaginal route and only 4% cases had been delivered by Caesarean section. A study by G Oswyn¹¹ et al included 73% babies delivered vaginally. And Smith et al¹⁵ included 62% babies born by vaginal route. In all those works, majority of asphyxiated babies were delivered vaginally.

Inclusion and Exclusion Criteria

The inclusion and exclusion criteria in this study were similar to other studies conducted by PS Shah¹² et al, K Karam¹⁴ et al, Ong LC¹³ et al, G Oswyn et al and Ellis et al. However, all these works included babies with low (< 5) APGAR score at 5 minutes and umbilical acidosis with pH of < 7.20.

Birth Weight

Babies in this study had birth weight ranging between 2100 gms to 3300 gms. Low birth weight babies weighing < 2500 constituted 38% of the study population. None of them had macrosomia with birth weight > 4000 gms. The mean birth weight was 2.60 kg with standard deviation of 0.32. The mean weight of babies with HIE III was 2.47 kgs, while that of HIE I and HIE II were 2.66 and 2.63 kgs respectively. The difference in mean birth weight between HIE II and HIE III was found to be significant with p-value of 0.017, i.e. < 0.05. This observation implies that babies with low birth weight are more prone to develop HIE III. G Oswyn¹¹ et al also associated increased risk of birth asphyxia with low birth weight.

CNS Involvement

In the present study CNS involvement with HIE was seen in all cases of asphyxia, HIE I comprised 21%; HIE II 56% and HIE III 23% cases. Clinical seizures were observed in 74% cases. Ana Martin⁷ et al in their study found CNS involvement in 72% cases.

CONCLUSION

Our study indicates that among asphyxiated babies, of which 81% delivered outside hospital. Those with birth weight on lower side are more likely to develop HIE III. CNS was affected in all cases. HIE I was diagnosed in 21% cases, HIE II in 56% and HIE III in 23% of study population. To conclude it must be emphasised that a lot more has to be done in order to identify the risk factors, prevention and treatment of birth asphyxia, so that the precious lives and lost intelligence of millions of babies can be saved, and smiles preserved on face of their families.

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