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Research Article

Clinico laboratory determinants of outcome among babies with perinatal asphyxia in Osogbo, Southwestern Nigeria

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ABSTRACT

Background: Many clinical, pathological, biochemical and metabolic changes occur as a result of perinatal asphyxia. These changes affect many organ and systems like central nervous system, cardiovascular system, pulmonary, renal, adrenal, gastrointestinal tract, skin and haemopoetic systems. The aim of the study was to identify various clinical and biochemical determinants of outcome in perinatal asphyxia so as to institute proactive the management of such babies.

Methods: All newborn infants with birth asphyxia over 5 year period (2009-2013) were retrospectively studied. The data studied included place of birth, gestational age, Apgar score, mode of resuscitation, details of complete physical examination especially as regard each of the system. Results of investigations like haematocrit, serum electrolytes and urea, blood glucose done in the first 24 hours of life and also other investigations like lumbar puncture, full blood count, cultures were noted. The outcome studied was survival and death of the babies.

Results: One thousand, six hundred and seven babies were admitted into special care baby's unit over the 5 year period, between 2009 and 2013. Nine hundred and seventy nine (60.9%) of them were males while 628 (39.1%) were females, M:F ratio was 1.6:1. Of the 1607 babies, 563 (35.0%) were asphyxiated. Of 1607 admitted during the period of study, 304 (18.9%) died while 128 (22.7%) of 563 babies with perinatal asphyxia died. Therefore, perinatal asphyxia accounted for 42.1% of the total mortality. 22 (7.8%) of the 280 babies who suffered moderate asphyxia compared with 106 (37.9%) of 283 babies who suffered severe asphyxia died. ($\chi 2$ = 72.4, p=0.000). Many of the asphyxiated babies had multisystemic adverse features. Significantly more babies who were out born, low birth weight, macrosomic and hypothermic than otherwise died. Also more babies with cyanosis, respiratory distress, apnoea, abdominal distension, feed intolerance, oliguria/anuria, bleeding disorder, abnormal muscle tone, seizures, bulging frontannel, and coma died, $p \geq 0.001$. Also, mean haematocrit, plasma potassium and urea was significantly lower while plasma sodium was significantly higher among the babies who survived ($p \geq 0.001$).

Conclusions: Our findings have highlighted the major role of asphyxia in neonatal mortality and multisystemic morbidities or complications which contributed to death. It is therefore, likely that efforts at preventing perinatal asphyxia will be more rewarding. Such efforts include free and compulsory antenatal care, training of more skilled labour attendants and women empowerment.

Keywords: Clinico-laboratory, Perinatal asphyxia, Outcome, Nigeria

INTRODUCTION

In asphyxia, there is impairment of blood-gas exchange, resulting in hypoxemia and hypercapnia. The combination of hypoxia and ischemia results in clinical and biochemical changes, which lead to multiorgan cell damage including neuronal cell death and brain damage.[1] Continuous asphyxia will also lead to multiple organ and systems dysfunction.

As a result of perinatal asphyxia, many clinical, pathological, biochemical and metabolic changes occur. These changes affect many organs and systems like central nervous system, cardiovascular system, respiratory and haemopoetic systems or organs like renal, adrenal, gastrointestinal tract and skin.[2] However, the cerebral complications are the most devastating resulting in acute brain death or chronic morbidity or neurological sequelae like cerebral palsy, mental subnormality and cranial nerve palsies. Perinatal asphyxia is a serious clinical problem worldwide and contributes greatly to neonatal mortality and morbidity.[1,3,4] Diagnosis of asphyxia is usually made on the clinical criteria. The most frequent abnormalities involved are found in kidneys, respiratory, central nervous and cardiovascular systems.[2] The spectrum of disorders to which neonate is exposed as a result of asphyxia include feed intolerance, necrotizing enterocolitis, systemic hypotension, cardiogenic shock, congestive cardiac failure, disseminated intravascular coagulopathy (DIC), meconium aspiration and a wide variety of metabolic problems including hyponatremia, hypoglycemia, hypocalemia and metabolic acidosis.[5] The objective of the study was therefore to identify the clinical and biochemical determinants of outcome in perinatal asphyxia so as to institute anticipatory management of such babies.

METHODS

Ethical clearance was obtained from the hospital. All newborn infants with birth asphyxia were retrospectively studied. Information from admission records were recorded into the research proforma designed for the study. Excluded from the study were preterm with <34 weeks of gestation and/or birth weight <1.5kg, neonates with major congenital malformations of cardiovascular, central nervous system, respiratory system or dysmorphic features. Babies with birth asphyxia were those with APGAR score of ≤ 7 at five minutes, those who did not cry immediately after delivery whether or not they had neurologic symptoms.[6,7] For babies delivered outside our health facility birth asphyxia was presumed in babies in whom there was documented history of failure or delay in crying or breathing at birth or who had gasped for a long time or had to be stimulated for a prolonged period of time or was unable to suck in the first 24 hours with or without associated pallor, cyanosis, neurologic dysfunction and multisystemic features like respiratory distress, abdominal distension, coma or seizures. Maternal and neonatal data such as maternal age, antenatal check-up, place of delivery, parity, type of delivery, presence of meconeum whether labour was induced or spontaneous, pregnancy complications, gestational age, Apgar score, mode of resuscitation and mode of transportation to the health facility extracted and entered into the proforma. Referral notes were considered. Additional information about newborn records were age of baby at admission, gestational age, birth weight, sex, clinical features, severity of birth asphyxia based on Apgar Score, temperature at admission, details of complete physical examination especially as regard each of the systems. Anthropometry measurements of each baby were recorded, and in the case of babies born outside our health facility (outborn) with no recorded birth weight, the weight at presentation was recorded as admission weight and assumed to be approximate to birth weight since most of the babies presented within 24 to 48 hours after birth. Moderate asphyxia is Apgar score of 4 and 5 while severe asphyxia was Apgar score of ≤3. Results of investigations like haematocrit, serum electrolytes and urea, blood glucose done in the first 24 hours of life and also other investigations like lumbar puncture, full blood count, cultures were noted. Also, documented were management instituted and subsequent final outcome was recorded in form of dead or alive.

The management of asphyxiated babies in our centre consisted of nursing in a thermoneutral environment, monitoring vital signs, monitoring of oxygen saturation and administration of oxygen as indicated, administration of intravenous fluids, correction of fluid and electrolyte imbalance, termination of seizures with intramuscular paraldehyde and then parenteral phenobarbitone and sometimes phenytoin sodium whenever phenobarbitone was unavailable. There were no facilities in our centre for electroencephalography (EEG) monitors, modern neuroimaging techniques, viral studies, estimates of blood and urine amino acids. There was also no facility for blood pH and blood gases hence these were not done.

Facility for therapeutic hypothermia (either selective head cooling or total body cooling) was not available. However, babies were protected against increased temperature. Other definitive treatments were usually given as dictated by the observed systemic abnormality. This includes antibiotics for bacterial infections and correction of metabolic abnormalities. Autopsy was not done in the majority of dead babies because of refusal of consent by the parents.

The data generated was entered into HP personal computer. Analysis of the data was undertaken with the statistical package for the social sciences (SPSS version 17). Means and standard deviations were determined for continuous variables like haematocrit, blood glucose, plasma electrolytes and urea were compared between asphyxiated babies who survived and those who died using analysis of variance and Students‘t’ test. Proportions and percentages were compared using chi-square (χ^2) test. P values, <0.05 was taken as statistically significant.

RESULTS

All admissions

One thousand, six hundred and seven babies admitted into special care baby unit over the 5 year period, between 2009 and 2013 were retrospectively studied. Nine hundred and seventy nine (60.9%) were males while 628 (39.1%) were females, M:F ratio was 1.6:1. Of the 1607 babies, 563 (35.0%) were asphyxiated; 348 (61.8%) were males, 215 (38.2%) females, M:F was 1.6:1.

Perinatal asphyxia

Of the 563 babies who suffered perinatal asphyxia, 339 (60.2%) and 224 (39.8%) were inborns and outborns respectively. Two hundred and eighty three (50.3%) had moderate asphyxia (Apgar score 4 and 5) and 280 (49.7) had severe asphyxia (Apgar score ≤3). Table 1 shows the comparison of gender, delivery and outcome data between moderately and severely asphyxiated babies. There was no gender variation in the degree of asphyxia. Significantly higher proportions of babies delivered SVD and inborn were asphyxiated. Also, while higher proportions of inborn were moderately asphyxiated, more of the babies delivered outborn were severely asphyxiated. Table 2 shows comparison between clinical profiles of babies who suffered from moderate and severe asphyxia. Significantly higher proportion of babies with severe asphyxia had hypothermia, respiratory distress, gastrointestinal, renal and central nervous sytems abnormalities.

Table 1: The comparison of some characteristics between moderately and severely asphyxiated babies.

Parameter	Moderate asphyxia N = 283	Severe asphyxia N= 280 (%)	Total N (%)	χ^2	p value
Gender					
Male	175 (61.8)	173 (61.8)	348 (61.8)	0.00	0.9
Female	108 (31.2)	107 (31.2)	215 (31.2)		
Mode of delivery					
SVD	136 (48.1)	181 (64.6)	317 (56.3)	15.9	0.000
C/S	145 (51.2)	97 (34.6)	242 (43.0)		
Vacuum extractor	2 (0.7)	2 (0.7)	4 (0.7)		
Place of birth					
Inborn	227 (80.2)	112 (40.0)	339 (60.2)	94.8	0.000
Outborn	56 (19.8)	168 (60.0)	224 (39.8)		
Outcome					
Alive	261 (92.2)	174 (52.1)	435 (77.3)	72.4	0.000
Dead	22 (7.8)	106 (37.9)	128 (22.7)		

Key: SVD- Spontaneous vertex delivery; C/S- Caesarean section; Moderate asphyxia = Apgar score 4 and 5; Severe Asphyxia = Apgar score ≤3

Table 2: Comparison of clinical profiles of babies who suffered from moderate and severe asphyxia.

Parameter	Moderate asphyxia N (% of 283)	Severe asphyxia N (% of 280)	Total N (% of 563)	χ^2	p value
Low birth weight	32 (11.3)	43(15.4)	75 (13.3)	2.0	NS
Macrosomia	12 (4.2)	29 (10.4)	41 (7.3)	7.8	S
Fever	6 (2.1)	32 (11.4)	38 (6.7)	17.9	S*
Hypothermia	24 (8.5)	61 (21.8)	85 (11.5)	19.4	S
Pallor	20 (7.1)	78 (27.9)	98 (17.4)	42.3	S
Cyanosis	33 (11.7)	68 (24.3)	101 (17.9)	15.2	S
Respiratory distress	33 (11.7)	89 (31.8)	122 (21.7)	33.4	S
Apnoea	15 (5.3)	73 (26.1)	88 (15.6)	46.0	S
Abdominal distension	10 (3.5)	51 (18.2)	61 (10.8)	31.4	S
Feed intolerance	28 (9.9)	95 (33.9)	123 (21.8)	47.6	S
Oliguria/Anuria	11(3.9)	61 (21.8)	72 (12.8)	40.4	S
Bleeding disorder	2 (0.7)	8 (2.9)	10 (1.8)	2.6	NS*
Jitteriness	15 (5.3)	72 (25.7)	87 (15.5)	44.9	S
Abnormal muscle tone	4 (1.4)	64 (22.9)	68 (12.1)	84.3	S*
Seizures	5 (1.8)	39 (13.9)	44 (7.8)	27.3	S*
Bulging frontannel	3 (1.1)	35 (12.5)	38 (6.7)	29.4	S*
Unconsciouness	5 (1.8)	92 (32.9)	97 (17.2)	93.2	S*

Yate's Corrections applied

Mortality and associated clinicolaboratory factors

Of 1607 total admissions during the period of study, 304 (18.9%) died while 128 (22.7%) of 563 babies with perinatal asphyxia died. Therefore, perinatal asphyxia accounted for 42.1% of the total mortality. Twenty-two (7.8%) of the 280 babies who suffered moderate asphyxia died, 106 (37.9%) of 283 babies who suffered severe asphyxia died. (χ^2 = 72.4, p=0.000).

Table 3 shows the comparison of some clinical profile between babies who survived and those who died from perinatal asphyxia. There was no gender variation. More males had asphyxia and higher proportion of females died. Thus, 54 (25.1%) of 215 females died while 74 (21.3%) of the 348 males died, p=0.3. Many of the asphyxiated babies had multisystemic adverse features. Significantly more babies who were outborn, low birth weight, macrosomic and hypothermic died. Also more babies with cyanosis, respiratory distress, apnoea, abdominal distension, feed intolerance, oliguria/anuria, bleeding disorder, abnormal muscle tone, seizures, bulging frontannel, and coma died, p at least 0.01.

Table 3: Comparison of some clinical profile between babies who survived and died from perinatal asphyxia.

Variable	No of babies affected n (%)	Babies who survived n = 435**	Babies who died n = 128**	χ^2	p value
Gender					
Male	348 (61.8)	274 (63.0)	74 (57.8)	1.1	0.3
Female	215 (31.2)	161 (37.0)	54 (42.2)		
Mode of delivery					
SVD	317 (56.3)	230 (58.9)	87 (68.0)	8.6	0.003
C/S	242 (43.0)	201 (46.2)	41(32.0)		
Vacuum	4 (0.7)	4 (0.9)	0 (0.0)		
Place of birth					
Inborn	339 (60.2)	293 (67.4)	46 (35.9)	40.8	0.000
Outborn	224 (39.8)	142 (32.6)	82 (64.1)		
Low birth weight	75 (13.3)	30 (6.9)	45 (35.1)	68.4	0.000
Macrosomia	41 (7.3)	19 (4.4)	22 (17.2)	24.1	
Fever	38 (6.7)	26 (6.0)	12 (9.4)	1.8	0.18
Hypothermia	85 (11.5)	48 (11.0)	37 (28.9)	24.6	0.000
Pallor	98 (17.4)	52 (12.0)	46 (35.9)	39.6	0.000
Cyanosis	101 (17.9)	49 (11.3)	52 (40.6)	57.9	0.000
Respiratory distress	122 (21.7)	58 (13.3)	64 (50.0)	78.3	0.000
Apnoea	88 (15.6)	29 (6.7)	59 (46.1)	116.6	0.000
Abdominal distension	61 (10.8)	28 (6.4)	33 (25.8)	38.3	0.000
Feed intolerance	123 (21.8)	55 (12.6)	68 (53.1)	94.9	0.000
Oliguria/Anuria	72 (12.8)	29 (6.7)	43 (33.6)	64.3	0.000
Bleeding disorder	10 (1.8)	4 (0.9)	6 (4.7)	6.0	0.01*
Jitteriness	87 (15.5)	51 (11.7)	36 (28.1)	20.4	0.000
Abnormal muscle tone	68 (12.1)	28 (6.4)	40 (31.3)	57.3	0.000
Seizures	44 (7.8)	12 (2.8)	32 (25.0)	67.9	0.000
Bulging frontannel	38 (6.7)	15 (3.4)	23 (18.0)	33.1	0.000
Unconsciouness	97 (17.2)	32 (7.4)	65 (50.8)	130.8	0.000

*Yate's correction applied; ** Multiple diagnoses occurred in some

Table 4 shows comparison of some laboratory findings between babies who survived and those who died from perinatal asphyxia. Among the babies who survived from asphyxia, mean haematocrit, plasma potassium and urea was significantly lower while plasma sodium was significantly higher p at least 0.001.

DISCUSSION

Perinatal asphyxia alone accounted for 35.0% of the admissions in the study period and 42.1% of neonatal mortality recorded during the study period. Among the asphyxiated, 22.7% died. The present study therefore, confirms perinatal asphyxia as a major cause of morbidity and mortality in the new-born period especially in

developing countries Nigeria inclusive. Previous studies undertaken in our centre had shown this high rate of morbidity and mortality from perinatal asphyxia.[4,8] Globally, about one quarter of all neonatal deaths are caused by birth asphyxia.[9] Also, according to World Health Organization estimates in the developing countries, 3% of all infants (3.6 million) suffer from moderate to severe birth asphyxia of which 23% (840,000) die and approximately the same number develop serious sequalae.[10] This is similar to what was obtained in the present study. However, Amritanshu, et al reported 10.94% prevalence of asphyxia among 5481 admissions in India.[11]

Table 4: Comparison of some laboratory findings between babies who survived and those who died from perinatal asphyxia.

Variable	Babies who survived n = 249	Babies who died n = 128	95% CI	t	p value
Haematocrit (%) Mean (SD)	46.57 (6.15)	53.04 (7.28)	-7.872 to -5.295	9.08	0.000
Blood glucose mmol/l Mean (SD)	3.54 (0.91)	3.21 (0.86)	0.139 to 0.521	3.40	0.001
Plasma HCO_3 Mean (SD)	18.11 (1.38)	18.21 (1.42)	-0.198 to 0.398	0.66	0.51
Plasma Na^+mmol/l Mean (SD)	134 (3.4)	128 (6.3)	3.02 to 4.98	8.01	0.000
Plasma K^+ mmol/l Mean (SD)	4.24 (0.38)	4.90 (0.55)	-0.755 to 0.549	13.64	0.000
Plasma Urea mg/dl Mean (SD)	39.8 (10.23)	49.50(16.8)	-12.445 to -6.955	6.95	0.000

SD = Standard deviation; CI = Confidence interval

In the present study, many of the systems were involved in varying degrees of severity. Significantly more babies who were outborn, low birth weight, macrosomic and hypothermic died. Also, more babies with cyanosis, respiratory distress, apnoea, abdominal distension, feed intolerance, oliguria/anuria, bleeding disorder, abnormal muscle tone, seizures, bulging frontannel, and coma died $p \leq 0.01$. Clearly, these features may be due to varying causes including and especially infections which may be commoner among outborns who have been delivered by unskilled birth attendants. Yelamali et al similarly found higher incidence of mortality among the outborns.[12] This can be explained on the basis of late presentation, unsatisfactory mode of transportation, higher incidence of hypothermia and hypoglycaemia which may have occurred in them. Amritanshu et al also in the study in India of asphyxiated babies found hypotension, hypoglycaemia, hypoxaemia, hypothermia as factors associated with poor outcome.[11] Anand-Babu et al recorded higher incidence of perinatal complications like convulsions, apnoea, feeds intolerance, necrotizing enterocolitis (NEC) among the asphyxiated babies.[13] In a previous retrospective study of 130 asphyxiated term infants, the proportion of those with organ dysfunction was: renal 70%, cardiovascular 62%, pulmonary 86%, hepatic 85%.[14] Jayshree et al observed incidence of ARF in 16% of asphyxiated babies while Finer et al observed Oliguria in 25% of cases.[15] Perinatal asphyxia has multisystemic or multiorgan adverse effects which contribute to high mortality among the asphyxiated babies. The observed pathologies could be explained based on pathophysiological processes in asphyxia. Multiorgan dysfunction is theorized to be secondary to the "diving reflex". It is known that perinatal asphyxia initially causes transient increase followed by a decrease in heart rate (HR), mild elevation in blood pressure (BP), increase in central venous pressure (CVP) and essentially no change in cardiac output (CO).[14] This is then accompanied by redistribution of CO with increased proportion of blood going to the brain, heart and adrenal glands (diving reflex). With prolonged asphyxia cerebral blood flow becomes dependent on systemic BP (loss of cerebral vascular autoregulation). Reduced cardiac output causes hypotension which subsequently causes multisystemic/multiultiorgan dysfunction in perinatal asphyxia with the kidney being most common organ affected during perinatal asphyxia. The proximal tubule of the kidney is especially affected by decreased perfusion, leading to acute tubular necrosis (ATN) resulting in oliguria. There may also be cardiac dysfunction caused by transient myocardial ischemia, reduced contractility.[14] Gastrointestinal effects include an increased risk of bowel ischemia, feed intolerance and necrotizing enterocolitis. Hematologic effects include bleeding disorders and reduced production of platelets by the bone marrow. Liver involvement may be manifested by isolated elevation of hepatocellular enzymes. Pulmonary effects include increased pulmonary vascular resistance leading to primary pulmonary hypertension and pulmonary hemorrhage, secondary respiratory distress syndrome due to failure of surfactant production, and meconium aspiration.[14]

In the present study, seizures occurred in 7.8% of the all asphyxiated babies studied; 1.8% in moderate asphyxia but in 13.9% of severe asphyxia. The cause of seizures in asphyxia is due to the same pathophysiological mechanisms. Hypoxic-ischemic encephalopathy could also disrupt the ATP-dependent sodium-potassium pump and cause excessive depolarization. This is an important cause of neonatal seizures.[16,17] Also, the role of hypoxic-

ischaemic encephalopathy was intertwined with those of hypoglycaemia, hypocalemia and infection. Perinatal asphyxia may also increase serum calcitonin which inhibits Ca release from bone and results in hypocalcemia.[18,19]

In the present study, the laboratory findings between babies who survived and those who died from perinatal asphyxia revealed significantly mean haematocrit, plasma potassium and urea were significantly higher among babies who died than those who survived asphyxia in the first 24 hours of life while plasma sodium wan glucose were significantly lower. Basu et al in India also found mean serum sodium level was significantly lower; mean serum potassium was higher than controls.[20]

Our findings have highlighted the major role of asphyxia in neonatal mortality and mulisystemic adverse effects as associated factors or early complications contributing to poor outcome among the asphyxiated babies. Mortality is unacceptably high even at centres where there are neonatal advanced life support facilities.[5] Also, the challenge to cope with late sequalae of asphyxia like cerebral palsy, seizure disorders, mental subnormality, microcephaly can be very daunting to families, communities and nations especially developing countries. It is therefore important that efforts at preventing perinatal asphyxia will be more rewarding. Such efforts include free and compulsory antenatal care, training of more skilled labour attendants and women empowerment.

CONCLUSION

Our findings have highlighted the major role of asphyxia in neonatal mortality and multisystemic morbidities or complications which contributed to death. It is therefore, likely that efforts at preventing perinatal asphyxia will be more rewarding. Such efforts include free and compulsory antenatal care, training of more skilled labor attendants and women empowerment.

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