Original Article

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Correlation of Metabolic Acidosis in Neonate with Morbidity, Mortality and Neurodevelopmental Outcome.

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Abstract

Background: In metabolic acidosis there is risk of neonatal encephalopathy with symptoms varying from lethargy to coma. The neonates with encephalopathy usually present with decreased level of consciousness with abnormality in neuromotor tone and associated with seizure like activity, hypoventilation or apnea. **Subjects and Methods:** In the present study the children were followed upto 9 months of age and assessment of neurodevelopment was done and the correlation of acidosis with neurodevelopment outcome was analyzed. **Results:** Of 100 neonates with metabolic acidosis, 26 babies had necrotizing enterocolitis, 20 had seizures, 15 had intraventricular/periventricular hemorrhage and 33 babies had renal dysfunction. Out of 100 neonates with metabolic acidosis, 10 babies died and 23 had good outcome. There exists correlation between pH and neurodevelopmental outcome. The p value is 0.000 i.e it is significant. Thus with decreasing pH, there is increased incidence of neurodevelopmental delay. The incidence of developmental delay is more in neonates with pH <7.20, 4 (21.1%) babies had transient abnormalities, 2 (10.5%) had persistent abnormalities. There exists correlation between base deficit and neurodevelopmental outcome. The p value is 0.000 i.e it is significant. Thus with increasing base deficit, there is increased incidence of neurodevelopmental delay. **Conclusion:** With based deficit of >12 mmol/l, there is increased incidence of neurodevelopmental delay i.e 25% had persistent abnormality and 10% transient abnormality hence it can be used as one of the early predictors of neurodevelopmental delay.

Keywords: Base deficit, Metabolic Acidosis, Neurodevelopment, Neonates.

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Introduction

Acidemia is common in neonates especially in association with prematurity and perinatal asphyxia. Metabolic acidosis in preterm infants may be associated with hypoxemia, hypotension or poor tissue perfusion, anemia, infection or sepsis, or strenuous activity (respiratory distress). There is an association between acidosis, acute physiological dysfunction in the neonate and longer term neurodevelopmental abnormalities. Whether the acidosis is causative or only associated with acute organ dysfunction and abnormal neurodevelopment is less certain.[1,2]

Metabolic Acidosis results from either inability of the kidney to excrete the dietary H+ load or increase in the generation of H+ or loss of bicarbonate. Base Excess is a useful parameter which reflects metabolic component of abnormality under steady state conditions. The presence or absence of increased anion gap is useful for determining the cause of metabolic acidosis.[3]

Metabolic acidosis associated with normal anion gap results from buffer loss through the renal or gastro intestinal systems. Bicarbonate is replaced by chloride and serum chloride is elevated. Increased production of non-volatile acids in the body such as phosphate, sulfate or organic acids titrates bicarbonate and leads to metabolic acidosis. Subsequently, inadequate hydrogen ion excretion by premature kidney results in acidosis. Metabolism of sulfur containing aminoacids in casein and increased hydrogen ion release due to rapid mineralization of bone cause an increased acid load.[4]

Birth asphyxia is most common cause of metabolic acidosis. In asphyxia initially there is redistribution of cardiac output with an increased proportion going to brain, heart and adrenal glands. Impaired cerebral blood flow results in anaerobic metabolism and eventually intracellular energy failure due to increase in the utilization of glucose in the brain and fall in the concentration of glycogen, phosphocreatine and ATP. This energy failure impairs ion pump function resuling in accumulation of intracellular Na+, Cl, HCO3 & Ca+2, excitatory aminoacid neurotransmitters like glutamate.[5,6]

Failure of Na+/K+ ATPase pump leads to influx of Na+ and Ca+2 with osmotic influx of water causing neuronal edema. Activation of receptor subtypes such as kainate, NMDA, AMPA by glutamate released allow Ca+2 to enter the

neurons. Ca+2 ions inside the cells cause activation of intracellular proteases and lipases with generation of O2 free radicals causing further damage to cellular membranes exacerbating neuronal damage. Thus neonatal acidosis and hypoxia have their main effect on the central nervous system and in preterm infants leads to periventricular leukomalacia.[6-8]

Evaluation of neuromotor function is of paramount importance in establishing links between perinatal events and late outcome. Changes in neuromotor function observed during first year of life seem closely linked with maturation of central nervous system and with presence or absence of brain damage. Moreover these changes are relatively independent of familial and sociocultural background and quality of parent-infant interaction. Therefore it is useful to obtain information on the neuromotor function to document perinatal origins of a later dysfunction or to demonstrate the effect of early intervention and recovery from injury. Neuromotor retardation may occur due to gestational immaturity, perinatal hypoxia, birth trauma, metabolic disorders, hypoglycemia, Kernicterus, intra-uterine infections, postnatal CNS infections, hypothyroidism, developmental and chromosomal disorders.[9,10]

In hypoxic neonates due to anaerobic metabolism lactic acid accumulation results in metabolic acidosis. In metabolic acidosis there is risk of neonatal encephalopathy with symptoms varying from lethargy to coma. The neonates with encephalopathy usually present with decreased level of consciousness with abnormality in neuromotor tone and associated with seizure like activity, hypoventilation or apnea, depressed primitive reflexes like moro's reflex, suck reflex etc.[11]

In the present study we are using AMIEL-TISON.C, a method for neurological evaluation within the first year of life. It helps to identify at the end of the first year the children with no abnormality, patterns of transient abnormalities and patterns of persistent abnormalities.

There are many studies done on neonatal acidemia and base deficit by collecting umbilical cord arterial blood and correlation of neonatal acidosis with morbidity and mortality but there are only few studies done by collecting arterial blood sample. This is a follow up study where the children were followed upto 9 months of age and assessment of neurodevelopment was done and the correlation of acidosis with neurodevelopment outcome was analyzed.

Objectives:

1. To study the correlation between metabolic acidosis and morbidity in neonates.
2. To study the correlation between metabolic acidosis in neonate and neurodevelopmental outcome.
3. To study the correlation between severity of acidosis and occurrence of neurodevelopmental delay.

Subjects and Methods

Neonates below 48hrs of life admitted to NICU of Pragna Children's Hospital with symptoms of acidosis and in whom ABG done at the time of admission showing pH<7.35 and base excess <-4 mmol/l or base deficict >4mmol/l were included in the study group. The base excess is used for the assessment of the metabolic component of acid-base disorders, and indicates whether the patient has metabolic acidosis or metabolic alkalosis.

A typical reference range for base excess is −2 to +2 mmol/l. A base deficit (a below-normal base excess), thus indicates metabolic acidosis.

The neonates with respiratory acidosis are excluded. Respiratory acidosis defined as pCO2 >50mmHg and PH<7.35.

In NICU detailed history and thorough clinical examination was done.1ml of Arterial blood is collected in heparinised syringe and sent to the lab. Other investigations like Bloodurea, S.creatinine , S.electrolytes, CBP, CRP were done along with certain special investigations like NSG, 2DECho, EEG , CT Scan Brain as and when required to establish the final diagnosis. The neonates, then were followed up, at 3months, 6months and 9 months of age to assess the neurodevelopment by detailed neurological examination and Amiel Tison method of neurological assesment and find out any neuro developmental delay and its correlation with metabolic acidosis.

All neonates presenting with clinical features of acidosis and ABG done at admission showing pH of <7.35 and base deficit of >4mmol/l were includes and Neonates with Congenital anomalies, Inborn errors of metabolism, Congenital heart disease, ELBW<1kg, Gestational age of <30 wks were excluded from the present study.

Arterial blood is collected from peripheral artery after cleaning the site with povidone iodine and spirit. 1ml of blood is collected in heparinised syringe and sent immediately to the lab. The outcome measures in the present study was morbidity, neurodevelopmental outcome, mortality and good outcome

Results

In the present study a total of 110 neonates were enrolled. Of 110 neonates, 10 babies lost follow up. Thus in the final study, a sample of 100 neonates were analyzed.

The babies who presented with clinical features of acidosis within 48hrs of life were selected. In these babies ABG was done at the time of admission. The babies in whom ABG showed pH<7.35 and base deficit of >4mmol/l were selected for the study.

Out of 100 neonates , 19 babies had pH<7.20 , 46 babies had pH 7.20-7.25 and 35 babies had pH 7.25.

Out of 100 neonates , 50 babies had base deficit of 4 – 8 mmol/l , 30 babies had base deficit of 8 – 12 mmol/l and 20 babies had base deficit of >12 mmol/l.

Table 1: Distribution based on pH.

pH	Frequency
<7.20	19
7.20-7.25	46
>7.25	35
Total	100

Table 2: Distribution based on base deficit

Base deficit	Frequency
4-8 mmol/l	50
8-12 mmol/l	30
>12 mmol/l	20
Total	100

Of total 100 neonates with metabolic acidosis , 54 (54%) were male babies and 46 (46%) were female babies.

Table 3: Distribution as per gender based on pH

		Gender		Total
		Female	Male	
pH	<7.20	6 (31.6%)	13 (68.4%)	19
	7.20-7.25	18 (39.1%)	28 (60.9%)	46
	>7.25	22 (62.9%)	13 (37.1%)	35
Total		46	54	100

Of 19 babies who had pH<7.20, 6 (31.6%) babies were female and 13 (68.4%) babies were male. Of 46 babies who had pH 7.20-7.25, 18 (39.1%) were female and 28 (60.9%) were male. Of 35 babies who had pH >7.25 , 22 (62.9%) were female babies and 13 (37.1%) were male babies. There is no correlation between pH and gender. The p value is 0.039 i.e it is not significant.

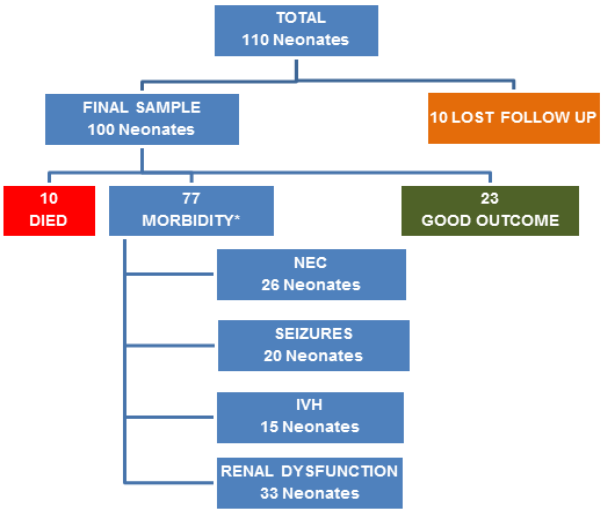
Table 4: Distribution as per gender based on base deficit

		Gender		Total
		Female	Male	
Base Deficit	4-8mmol/l	23 (46%)	27 (54%)	50
	8-12mmol/l	17 (56.7%)	13 (43.3%)	30
	>12mmol/l	6 (30%)	14 (70%)	20
Total		46	54	100

Of 100 neonates with metabolic acidosis, 26 babies had necrotizing enterocolitis, 20 had seizures, 15 had intraventricular/periventricular hemorrhage and 33 babies had renal dysfunction. Out of 100 neonates with metabolic acidosis, 10 babies died and 23 had good outcome.

Around 14 babies with morbidity had more than one outcome, 3 babies had more than two outcomes and 1 baby had more than three outcomes Of 19 babies who had pH<7.20, 7 (36.8%) babies had seizures. Of 46 babies who had pH 7.20-7.25, 10 (21.7%) babies had seizures. .Of 35 babies who had pH >7.25, 3 (8.6%) babies had seizures. There is no correlation between pH and seizures. The p value is 0.043 i.e it is not significant.

Of 50 babies who had base deficit of 4 – 8 mmol/l, 3 (6%) babies had seizures. Of 30 babies who had base deficit of 8 – 12 mmol/l, 10 (33.3%) babies had seizures. Of 20 babies who had base deficit of >12 mmol/l, 7 (35%) babies had seizures. There exists correlation between base deficit and seizures. The p value is 0.001 i.e it is significant.



Of 50 babies who had base deficit of 4 – 8 mmol/l, 2 (4%) babies had intraventricular/periventricular hemorrhage. Of 30 babies who had base deficit of 8 – 12 mmol/l, 9 (30%) babies had intraventricular/periventricular hemorrhage. Of 20 babies who had base deficit of >12 mmol/l, 4 (20%) babies had intraventricular/periventricular hemorrhage.

There is no correlation between base deficit and intraventricular / periventricular hemorrhage. The p value is 0.005 i.e it is not significant

Of 19 babies who had pH<7.20, 3 (15.8%) babies had necrotizing enterocolitis. Of 46 babies who had pH 7.20-7.25, 9 (19.6%) babies had necrotizing enterocolitis. Of 35 babies who had pH >7.25 ,.14 (40%) babies had necrotizing enterocolitis. There is no correlation between pH and necrotizing enterocolitis. The p value is 0.061 i.e it is not significant.

Of 19 babies who had pH <7.20, 3 (15.8%) babies had renal dysfunction. Of 46 babies who had pH 7.20-7.25, 21 (45.7%) babies had renal dysfunction . Of 35 babies who had pH >7.25, 9 (25.7%) babies had renal dysfunction.

Of 19 babies who had pH <7.20, not even a single baby had good outcome. Of 46 babies who had pH 7.20-7.25, 8 (17.4%) babies had good outcome . Of 35 babies who had pH >7.25, 15 (42.9%) babies had good outcome.

Of 50 babies who had base deficit of 4 – 8 mmol/l, not even a single baby died. Of 30 babies who had base deficit of 8 – 12 mmol/l, 3 (10%) babies died. Of 20 babies who had base deficit of >12 mmol/l, 7 (35%) babies died.

Discussion

In the present study, 100 neonates were enrolled. Both term and preterm neonates were included. Preterm babies were more in each group. But the pvalue (0.649/0.630) is not

significant. There is no correlation between gestational age and pH & base deficit.

In the study conducted by Victory R et al,[12] had concluded that umbilical cord pH and B.E are related to subsequent adverse outcome events for infants delivered very preterm. Worsening acidosis is associated with progressively greater increase in these outcomes. In the study conducted by Salhab and Perlman,[13] had concluded that the larger depressed preterm infant is at increased risk for moderate to severe encephalopathy.

Table 5: Correlation between pH and neurodevelopmental outcome

		Neurodevelopment				Total
		Died	No Abn	PA	TA	
pH	<7.20	9	4	2	4	19
		47.4%	21.1%	10.5%	21.1%	100.0%
	7.20-7.25	1	37	6	2	46
		2.2%	80.4%	13.0%	4.3%	100.0%
	>7.25	0	33	1	1	35
		.0%	94.3%	2.9%	2.9%	100.0%
Total		10	74	9	7	100
		10.0%	74.0%	9.0%	7.0%	100.0%

*No Abn – No Abnormality, TA – Transient abnormality, PA – Persistent abnormality

Table 6: Correlation between base deficit and neurodevelopmental outcome.

		Neurodevelopment				Total
		Died	NoAbn	PA	TA	
Base Deficit	4-8mmol/l	0	47	2	1	50
		.0%	94.0%	4.0%	2.0%	100.0%
	8-12mmol/l	3	21	2	4	30
		10.0%	70.0%	6.7%	13.3%	100.0%
	>12mmol/l	7	6	5	2	20
		35.0%	30.0%	25.0%	10.0%	100.0%
Total		10	74	9	7	100
		10.0%	74.0%	9.0%	7.0%	100.0%

*No Abn – No Abnormality, TA – Transient abnormality, PA – Persistent abnormality

In the present study, there is no correlation between gender and pH & base deficit. Of total 100 neonates with metabolic acidosis, 54 were male babies and 46 were female babies. But in each group of pH and base deficit, the incidence varied. There is no correlation between pH , base deficit and gender. The p-value for the gender and pH & base deficit is 0.039 and 0.179 respectively i.e it is not significant.

In a study conducted by Anne Lisbeth Hoffmann et al,[14] a total of 2778 infants born at term were studied to determine relationship between APGAR scores after 1 min, umbilical artery pH values , mode of delivery , diagnosis of fetal distress leading to operative delivery and sex of the neonate. A significantly higher incidence of operatively delivered for fetal distress and acidosis was found in boys i.e 58.4%.

In a study conducted by Hassan et al,[15] in which nucleated red blood cell was used for early diagnosis of perinatal asphyxia had the incidence of acidosis more in male babies i.e 63% .

In the present study, there is increased incidence of seizures in neonates with base deficit >8mmol/l (35%) and pH<7.20 (36.8%). There is correlation between base deficit and seizures. The p value for seizures and base deficit is 0.001 i.e it is significant.

There is correlation between pH, base deficit and mortality. The p value for the mortality and pH & base deficit is 0.000 and 0.000 respectively i.e it is not significant. Of 19 babies who had pH <7.20, 9 (47.4%) babies died. Of 46 babies who had pH 7.20-7.25, 1 (2.2%) baby died .Of 35 babies who had pH >7.25, not even a single baby died. Of 50 babies who had base deficit of 4 – 8 mmol/l, not even a single baby died. Of 30 babies who had base deficit of 8 – 12 mmol/l, 3 (10%) babies died. Of 20 babies who had base deficit of >12 mmol/l, 7 (35%) babies died.

R.L.Andres et al,[16] conducted a study that concluded that the metabolic component of fetal academia i. e umbilical artery pH<7.0, base deficit, and bicarbonate are most important variables in subsequent neonatal morbidity i.e IVH, RDS, NEC, sepsis. Low umbilical artery pH<7.0 associated with HIE, cardiopulmonary resuscitation, intubation and IUGR. Greater mean base deficit was associated with seizure, cardiopulmonary resuscitation, IUGR and HIE.

Sehdev HM et al,[17] conducted a case-control study in which both of them had umbilical artery pH<7. Cases were defined as those who had seizures, IVH, RDS, sepsis, GI complications and death. Controls had no complications. Out of 35 cases 3 neonatal deaths, 2 had IVH, 5 had GI dysfunction, 4 had neonatal seizures. Neonatal morbidity in neonates with umbilical artery cord pH<7 can be predicted by a high arterial base deficit value and low 5 minute APGAR score.

Belai et al had reported that an arterio-venous pCO2 difference >25 torr was a highly sensitive parameter to identify asphyxiated infants with seizures, HIE, cardio – pulmonary and renal dysfunction.[18]

There exists correlation between pH and neurodevelopmental outcome. The p value is 0.000 i.e it is significant .Thus with decreasing pH, there is increased incidence of neurodevelopmental delay. The incidence of developmental delay is more in neonates with pH <7.20, 4 (21.1%) babies had transient abnormalities, 2 (10.5%) had persistent abnormalities.

There exists correlation between base deficit and neurodevelopmental outcome. The p value is 0.000 i.e it is significant. Thus with increasing base defict, there is increased incidence of neurodevelopmental delay. With based deficit of >12 mmol/l, there is increased incidence of neurodevelopmental delay i.e 25% had persistent abnormality and 10% transient abnormality.

Helene T et al conducted neurodevelopmental assessment in children born with an umbilical artery pH<7. 0 infants were enrolled. From this group 23 were admitted in NICU and 8 required intubation. 28 survived neonatal period, 3 children

experienced an episode of mild hypertonia, and one child had mild motor developmental delay.[19]

Lavrijsen et al had conducted a study to determine the effects of academia at birth on neurodevelopment in preterm and fullterm neonates . Acidemia at birth increased the occurrence of severe intraventricular hemorrhage in preterm neonates and seizures in both preterm and full term neonates. However no significant effect of acidemia on longterm outcome could be demonstrated.[20]

Wildschut J et al had conducted study to assesss the relationship between acid-base status and quality and quantity of general movements at birth and at age 3 months and motor, cognitive and behavioural functioning at age of 4 yrs. The study concluded that in a sample of infants with a large variation in umbilical artery pH and without severe neonatal neurological abnormalities, acid base status at birth and quality of general movements at 3 months of age is not predictive for motor milestone achievement, cognitive and behavioural functioning at 4 yrs but these parameters are related to a less optimal condition of nervous system.[21]

Limitations of the Study

In the present study the babies were followed upto 9 months of age using AMIEL-TISON method of neurodevelopmental assessment. Hence only motor developmental delay could be assessed. Thereby the neonates need to be followed for longer period, atleast for 2 years of age using another scale for neuro development assessment so that even cognitive, language, behavioural developmental delay can be assessed.

Conclusion

Neuro developmental delay is difficult to predict at birth sometimes inspite of signs of neurological injury. Many parameters are used to predict so that early intervention can be started. Acid base imbalance at birth can be one of the important tools to predict neurodevelopmental delay as base deficit of >12mmol/L is found to have increased incidence i.e 25% had persistent abnormality and 10% had transient abnormality.

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