

Original Research Article

Correlation between umbilical cord blood pH and meconium stained deliveries

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ABSTRACT

Background: Among all live births approximately 13% neonates are born through meconium-stained amniotic fluid and out of these 5-10% developed MAS, which increases neonatal morbidity and mortality. The incidence increases as the gestational age advances with reported frequencies at 37, 40, and >42 weeks being 3%, 13%, and 18% respectively. Although there is a significant decrease in the occurrence of MAS and associated mortality in developed countries, MAS remains a major problem in developing countries. The objective was to study the correlation between umbilical cord blood PH and Meconium stained amniotic fluid.

Methods: Observational study done in KIMS hospital Bangalore, Karnataka, India in a study period of 18 months on a sample size of 100. Within 30 sec of delivery a segment of umbilical cord was clamped at both ends. Cord blood was collected in heparinised syringe. It was then transported with cold ice packs and blood pH, pCO₂, pO₂ were measured.

Results: In present study population, among those with MSAF, 72% had acidemia and 28% did not have acidemia. The mean (SD) of pH in the group with MSAF was 7.16 (0.10). The median (IQR) of pH in the group with MSAF was 7.14 (0.12). There was no significant difference between the groups (those with MSAF and those without MSAF but other risk factors) in terms of pH ($W = 867.500$, $p = 0.580$).

Conclusions: The presence of acidosis in the umbilical cord blood, used as a biochemical marker for perinatal asphyxia can be used to evaluate the significance of intrauterine passage of meconium. But a normal acid-base status at delivery present in many cases of MSAF, suggests that either a pre-existing injury or a non-hypoxic mechanism is often involved. MSAF is not always secondary to an acute hypoxic event.

Keywords: ABG, Acidosis, Hypoxia, Meconium stained amniotic fluid

INTRODUCTION

Among all live births approximately 13% neonates are born through meconium-stained amniotic fluid and out of these 5-10% developed MAS, which increases neonatal morbidity and mortality.^{1,2} The incidence increases as the gestational age advances with reported frequencies at 37, 40, and >42 weeks being 3%, 13%, and 18% respectively.³ In early 2000, the prevalence of meconium aspiration syndrome (MAS) ranged from 0.20% to 0.54%

in the general population and from 1.0% to 6.8% in infants born through meconium-stained amniotic fluid (MSAF).^{4,5}

Thick meconium staining is considered to be a marker of more prolonged or severe asphyxial episodes. Presence of thick meconium in the amniotic fluid is a predisposing factor towards adverse perinatal outcome mainly in the form of its aspiration and further sequelae. Intrauterine passage of meconium may occur as a physiological event

or secondary to fetal hypoxia and acidosis.⁶ Maturation of gastrointestinal tract in post-dated babies may account for the large number of cases with meconium stained amniotic fluid.

In the end it is concluded that the knowledge of Antenatal factors associated with MSAF provide a way of early identification of high risk cases in resource poor setup where facilities like electronic fetal monitoring are not available, who can be managed by optimal timely intervention in order to avoid severe asphyxia and meconium aspiration and its complications.⁷

MSAF is associated with a significant risk of caesarean delivery and prolonged labor. Also, MSAF was associated with the following fetal and neonatal complications; fetal heart rate abnormalities, low Apgar score at the 5th minute, need for neonatal resuscitation, neonatal asphyxia and neonatal infection.⁸

Intrauterine hypoxia causes fetal gasping and alters the direction of fluid flow. This allows meconium to be passed and aspirated. Model studies on guinea pigs suggest that extent of meconium aspiration is not related to amount of meconium in the amniotic fluid but to the length and degree of asphyxia.⁹

The presence of acidosis in the umbilical cord blood, used as a biochemical marker for perinatal asphyxia may provide a useful tool to evaluate the significance of intrauterine passage of meconium. It can be used as an indicator of early onset fetal hypoxia. But not all cases of Meconium stained amniotic fluid are secondary to fetal distress it can be secondary to a preexisting injury or a non-hypoxic mechanism.

The significance of the passage of meconium may be due to the fact that the pathogenesis of fetal and neonatal morbidity is multifactorial. There are two main theories on the etiology for the passage of meconium.¹⁰ First, it is thought by many to be a normal maturational event. As the fetus matures, it becomes more responsive to exogenous stimuli.

The second theory suggests that meconium passage is in response to fetal hypoxia or distress. Regardless of the initiating event (hypoxic insult or normal maturation of the intestinal tract), exposure of the umbilical vessels to meconium may result in further hypoxia. Pathologic evaluation of the umbilical vessels suggests that meconium induces vasoconstriction with resultant fetal hypoperfusion.

METHODS

Materials and method

It is an observational study conducted in the Department of paediatrics, KIMS hospital, Bangalore, Karnataka, India. Study subjects were the neonates delivered at

Kempegowda Institute of Medical Sciences. Study period was of 18 months, along with sample size 100.

Inclusion criteria

- Neonates of mothers diagnosed with
 - Eclampsia and preeclampsia.
 - Complicated pregnancies like cord prolapse, chorioamnionitis, abruption, cord compression
 - NST showing abnormal changes
 - Meconium stained amniotic fluid
 - Prolonged labour
 - Neonates with antenatal history of foetal distress
- Neonates with normal antenatal history and low APGAR score at birth.

Exclusion criteria

Normal antenatal history with normal Apgar score.

Newborns fulfilling the inclusion criteria are selected after taking informed consent from the mother. The standard technique of sampling cord blood for gas and acid-base analysis comprises three steps:

- Clamping a segment of the cord
- Removing the clamped cord segment
- Needle aspiration of venous blood sample from the excised clamped cord segment was done and later transferred into preheparinized syringes.

Immediately after birth, an approximate 20-cm segment of cord must be isolated between two sets of two clamps and 0.5ml of the venous blood is aspirated and transferred to preheparinized syringe. The sample is transported with ice packs. Delay in clamping by as little as 45 seconds after birth results in significant change in acid-base parameters the longer the delay, the greater is the change. The change is a progressive decrease in pH and base excess and increase in pCO₂ and lactate.

Statistical analysis

Data were coded and recorded in MS Excel spreadsheet program. SPSS v23 was used for data analysis. Descriptive statistics were elaborated in the form of means and standard deviations for continuous variables, and frequencies and percentages for categorical variables. Group comparisons were made using independent sample t-test for continuously distributed data, and chi-squared test for categorical data. Pearson's correlation coefficient was calculated to explore the linear correlation between two continuous variables. Appropriate non-parametric tests (Wilcoxon test/Kruskal Wallis Test/Spearman Correlation) were used when the data were non-normally distributed. ROC analysis was performed to find the best cut-off for diagnosing the outcome condition. Level of significance was taken as p<0.05.

RESULTS

In the study conducted in our hospital, in a sample size of 100, 25.0% of the participants had MSAF, 75.0% did not have MSAF. Cord blood ABG was done in all these patients to find the association between MSAF and umbilical cord blood pH.

Table 1: Distribution of the participants in terms of MSAF (n = 100).

MSAF	Frequency	Percentage
Present	25	25.0%
Absent	75	75.0%
Total	100	100.0%

Table 2: Association between acidemia and MSAF (n = 100).

MSAF	Acidemia			Chi-Squared Test	
	Present	Absent	Total	χ^2	p-value
Present	18(72.0%)	7(28.0%)	25(100.0%)	0.377	0.539
Absent	49(65.3%)	26(34.7%)	75(100.0%)		
Total	67(67.0%)	33(33.0%)	100(100.0%)		

Chi-squared test was used to explore the association between acidemia and MSAF. Among those with MSAF, 72% had acidemia and 28% did not have acidemia. Among those without MSAF, 65.3% had acidemia and 34.7% did not have acidemia.

Table 2 shows that there was no significant difference between the various groups in terms of distribution of Acidemia ($\chi^2=0.377$, p=0).

The variable pH was not normally distributed in the 2 subgroups of the variable MSAF. Thus, non-parametric

tests (Wilcoxon Test) were used to make group comparisons. The mean (SD) of pH in the group with MSAF was 7.16 (0.10). The mean (SD) of pH in the group without MSAF was 7.15 (0.15). The median (IQR) of pH in the group with MSAF was 7.14 (0.12). The median (IQR) of pH in the group without MSAF was 7.18 (0.18). The pH in the group with MSAF ranged from 7-7.34. The pH in the group without MSAF ranged from 6.56-7.35.

In table 3, there was no significant difference between the groups in terms of pH (W=867.500, p=0.580).

Table 3: Comparison of the 2 subgroups of the variable MSAF in terms of pH (n=100)

pH	MSAF		Wilcoxon Test	
	Present	Absent	W	p-value
Mean (SD)	7.16(0.10)	7.15(0.15)	867.500	0.580
Median (IQR)	7.14(0.12)	7.18(0.18)		
Range	7-7.34	6.56-7.35		

DISCUSSION

In present study done in KIMS hospital Bangalore, Karnataka, India, in a sample size of 100, it was noted that, among those with MSAF, 72 % had acidemia and 28% did not have acidemia.

There was no significant difference between the various groups (those with MSAF and those without MSAF) in terms of distribution of acidemia. Acidemia was present in most of the babies with MSAF but it was not statistically significant in comparison with others without MSAF which had other risk factors. There was no significant difference between the groups (those with

MSAF, those without MSAF) in terms of pH (W=867.500, p=0.580).

The mean (SD) of pH in the group with MSAF was 7.16 (0.10). The median (IQR) of pH in the group with MSAF was 7.14 (0.12).

The pH in the group with MSAF ranged from 7-7.34.

In a study done by Blackwell SC, done during a 4-year study period, Forty-eight cases met all study criteria, and pH values at delivery were as follows: pH \geq 7.20, n=29, and pH<7.20, n=19. There were no differences between groups in the incidence of clinical chorioamnionitis, in the presence of meconium below the vocal cords, or in

birth weight. Neonates with meconium aspiration syndrome and umbilical pH \geq 7.20 at delivery developed seizures as often as those with pH <7.20.⁵

Recent data also indicates that some fetuses with Meconium stained liquor will be compromised but most will not be unless associated with other abnormalities such as type II deceleration and fetal heart rate arrhythmias.¹¹

In a study done by Scott H et al on the significance of meconium stained amniotic fluid on preterm population it was seen that the incidence of MSAF in preterm deliveries is relatively low and there was no evidence that the presence of meconium is a marker for acute hypoxia.¹²

Similarly on our study, MSAF babies had acidemia but not all indicating that there are other causes for meconium stained amniotic fluid and not just intra uterine hypoxia.

CONCLUSION

The presence of acidosis in the umbilical cord blood, used as a biochemical marker for perinatal asphyxia can be used to evaluate the significance of intrauterine passage of meconium. But normal acid-base status at delivery present in many cases of MSAF, suggests that either a pre-existing injury or a no hypoxic mechanism is often involved.

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