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Original Research Article

Evaluation of Cord Blood Analytes and Serum Lactate in Term Babies Presenting with Meconium-Stained Amniotic Fluid

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Abstract

Background: Meconium-stained amniotic fluid (MSAF) is commonly encountered during labour and is often associated with concerns regarding fetal hypoxia. Objective biochemical markers such as umbilical cord blood gas analysis and lactate estimation can help differentiate physiological meconium passage from pathological fetal compromise.

Objectives: To evaluate cord blood analytes and serum lactate levels in term neonates born through MSAF with no additional risk factors and to analyze their correlation with neonatal outcomes.

Methods: A prospective observational study was conducted at tertiary care hospital Hospital, Gujarat, over 10 months (May 2015–March 2016) involving 264 term pregnant women with MSAF and no other risk factors. Umbilical arterial blood samples were analyzed for pH, pO_2, pCO_2, HCO_3 (actual and standard), base excess (BE, SBE), and lactate levels. Neonatal outcomes were assessed by Apgar scores, NICU admissions, and birth weight. Correlations between lactate and cord blood analytes were evaluated using Pearson's coefficient.

Results: Lactate values ranged from 16–55 mg% (mean 35.14 mg%), with most cases in the 36–40 mg% group. Lactate showed weak but significant correlations with pH ($r = -0.17$, $p = 0.0059$), H^+ ($r = 0.18$, $p = 0.0039$), pO_2 ($r = 0.16$, $p = 0.0091$), and pCO_2 ($r = -0.30$, $p < 0.00001$), and moderate negative correlations with HCO_3A ($r = -0.51$), HCO_3S ($r = -0.46$), BE ($r = -0.51$), and SBE ($r = -0.52$), all $p < 0.00001$. Apgar scores at 1 and 5 minutes were not significantly associated with lactate groups. NICU admission occurred in 11% of neonates, mostly for respiratory distress; there was one unrelated neonatal death.

Conclusion: Elevated cord blood lactate levels are common in term neonates with MSAF even in the absence of additional risk factors or adverse outcomes. Lactate correlates moderately with metabolic acid–base parameters and can serve as a simple adjunct to cord blood gas analysis for early identification of subclinical fetal hypoxia, aiding clinical decision-making and reducing unnecessary interventions.

Keywords: Meconium-Stained Amniotic Fluid, Umbilical Cord Blood, Lactate, pH, Base Excess, Fetal Hypoxia.

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Introduction

Intrapartum fetal surveillance aims to identify fetuses at risk of hypoxia and subsequent neurological injury, allowing timely intervention to reduce perinatal morbidity and mortality [1]. Traditional methods such as fetal heart rate monitoring, meconium staining of amniotic fluid (MSAF), and fetal scalp blood sampling are widely used but have limitations in sensitivity and specificity for detecting fetal acidaemia [1].

MSAF is observed in approximately 7–22% of term and 23–52% of post-term deliveries [1]. Although meconium passage may represent a physiological event related to fetal maturation, it may also be triggered by hypoxia, vagal stimulation, or umbilical cord compression, leading to anal sphincter relaxation and meconium passage into the amniotic cavity [1]. Approximately 5% of infants born through MSAF develop meconium aspiration syndrome (MAS), a major cause of respiratory morbidity and mortality in the neonatal period [2]. Around one-third of affected neonates require interventions such as intubation, mechanical ventilation, or advanced therapies, despite improvements in perinatal care [2].

The presence and thickness of meconium are considered important clinical markers. Thick and granular meconium is significantly associated with low cord pH and acidemia, adverse neonatal outcomes, and higher rates of operative delivery [3]. However, thin meconium may represent antepartum asphyxial events, as its dilution occurs gradually over hours to days [4]. Studies have demonstrated that thin meconium is associated with elevated urinary lactate:creatinine (L:C) ratios, indicating antepartum hypoxia, whereas thick meconium reflects intrapartum compromise [4].

Cord blood gas analysis provides an objective assessment of fetal acid–base status, and cord blood pH and base excess are traditionally used to document intrapartum hypoxia [1]. However, these parameters are influenced by both respiratory and metabolic factors. In contrast, lactate measurement reflects anaerobic metabolism and may provide a more direct and sensitive marker of fetal hypoxia [3]. Elevated lactate levels correlate with the severity of acidemia, low Apgar scores, and adverse neonatal outcomes, including respiratory distress, persistent pulmonary hypertension, and NICU admissions [2,3].

Recent evidence suggests that cord blood lactate levels may help predict which neonates born through MSAF are at higher risk of developing MAS and related complications, thereby guiding early intervention strategies [2,5]. Elevated lactate has also been identified as a potential adjunct to pH and base excess in the assessment of fetal well-being [4,3].

Given the limitations of conventional monitoring tools and the clinical burden of MSAF, evaluating cord blood analytes and serum lactate levels may improve early identification of at-risk neonates, enable targeted postnatal management, and potentially improve outcomes.

Materials and Methodology

Study Design and Setting: This prospective observational study was conducted at the Department of Obstetrics and Gynecology, at tertiary care hospital, Gujarat from May 2015 to March 2016. Ethical approval was obtained, and informed consent was secured from all participants.

Participants: A total of 264 term pregnant women with MSAF and no additional risk factors were included.

Inclusion Criteria

- Term pregnancy ($\geq$ 37 weeks)
- Singleton, cephalic presentation
- Meconium-stained amniotic fluid at delivery
- No fetal congenital anomalies or maternal comorbidities

Exclusion Criteria

- Clear amniotic fluid
- Preterm, multifetal or non-cephalic presentations
- Hypertension, diabetes, IUGR, or other risk factors

Data Collection: Immediately after delivery, the umbilical cord was double-clamped.

- 1 mL of arterial cord blood was collected in pre-heparinised syringes for ABG analysis
- 2 mL was collected in plain syringes for serum lactate estimation using the colorimetric method.

Lactate values were categorized in 5 mg% intervals (16–20, 21–25, 26–30, 31–35, 36–40, 41–45, 46–50, 51–55 mg%).

Neonatal Outcomes: Birth weight, Apgar scores at 1 and 5 minutes, NICU admission, and mortality were recorded.

Statistical Analysis: Data were analyzed using SPSS v20 and GraphPad Prism 5. Continuous variables were expressed as mean ± SD. Pearson's correlation was applied between lactate and cord blood analytes. $p \leq 0.05$ was considered statistically significant.

Result

A total of 264 term pregnant women with meconium-stained amniotic fluid (MSAF) and no additional risk factors were included in this prospective observational study.

Umbilical arterial cord blood was analyzed for pH, hydrogen ion concentration (H^+), partial pressure of oxygen (pO_2), partial pressure of carbon dioxide (pCO_2), actual bicarbonate (HCO_3A), standard bicarbonate (HCO_3S), base excess (BE), standard base excess (SBE), and lactate concentration. Neonatal outcomes were assessed by Apgar scores at 1 and 5 minutes, birth weight, NICU admission, and mortality.

Lactate concentrations ranged from 16 mg% to 55 mg%, with a mean of 35.14 mg%. For analysis, lactate values were grouped in 5 mg% intervals. The largest proportion of neonates (24.6%) fell within the 36–40 mg% category, followed by 26–30 mg% (21.6%) and 31–35 mg% (16.3%).

This distribution indicates a mild but consistent elevation in lactate levels in MSAF cases, reflecting a shift towards anaerobic metabolism even in the absence of other risk factors.

Table 1: Distribution of Lactate Groups in the Study Population (n = 264)

Lactate Range (mg%)	Frequency	Percentage (%)
16–20	10	3.8
21–25	19	7.2
26–30	57	21.6
31–35	43	16.3
36–40	65	24.6
41–45	33	12.5
46–50	29	11.0
51–55	8	3.0
Total	264	100.0

The majority of women (85.6%) delivered vaginally, 10.2% underwent lower segment cesarean section (LSCS), and 4.1% had instrumental deliveries (3.0% vacuum, 1.1% forceps). Among the LSCS group, 20 cases involved thick meconium. Instrumental deliveries included 5 cases with thick and 6 with thin meconium. Vaginal deliveries included 206 cases with thin and 23 with thick meconium. The mode of delivery was determined by fetal heart rate abnormalities and stage of labour, following standard intrapartum protocols, and showed no specific association with lactate groups.

Of the 264 neonates, 234 (88.6%) were transferred to their mothers, while 29 neonates (11.0%) were admitted to the NICU, predominantly for respiratory distress. There was one neonatal death, which occurred 48 hours after birth and was attributed to a probable congenital heart disease. The deceased neonate had elevated lactate and low Apgar scores (4 and 5 at 1 and 5 minutes, respectively), but this isolated event lacked statistical significance.

a) Lactate and pH: The mean pH was 7.26, with values ranging from 7.30 in the 21–25 mg% lactate group to 7.24 in the 51–55 mg% group. The correlation between lactate and pH was weak but statistically significant ($R = –0.1693$, $R^2 = 0.0287$, $p = 0.0059$).

b) Lactate and H^+: Mean H^+ increased from 50.16 mmol/L in the 21–25 mg% lactate group to 57.06 mmol/L in the 51–55 mg% group, with an overall mean of 54.29 mmol/L. The correlation was weak but significant ($R = 0.177$, $R^2 = 0.0313$, $p = 0.0039$).

c) Lactate and pO_2: Mean pO_2 values ranged from 20.93 mmHg (16–20 mg%) to 21.61 mmHg (51–55 mg%), with an overall mean of 22.25 mmHg. The correlation between lactate and pO_2 was weak but significant ($R = 0.1601$, $R^2 = 0.0256$, $p = 0.0091$).

d) Lactate and pCO_2: Mean pCO_2 decreased from 54.33 mmHg (16–20 mg%) to 42.41 mmHg (51–55 mg%), with an overall mean of 48.43 mmHg. A weak negative but significant correlation was found ($R = –0.2972$, $R^2 = 0.0883$, $p < 0.00001$).

e) Lactate and Bicarbonate Parameters

- **HCO_3A:** Mean = 21.27 mmol/L; moderate negative correlation with lactate ($R = –0.5113$, $R^2 = 0.2614$, $p < 0.00001$).
- **HCO_3S:** Mean = 18.38 mmol/L; negative correlation with lactate ($R = –0.4629$, $R^2 = 0.2143$, $p < 0.00001$).

Both bicarbonate parameters showed progressive decline with rising lactate, consistent with metabolic acidosis.

f) Lactate and Base Excess

- Base Excess (BE): Showed a moderate negative correlation with lactate ($R = –0.5112$, $R^2 = 0.2613$, $p < 0.00001$).
- Standard Base Excess (SBE): Also negatively correlated with lactate ($R = –0.5152$, $R^2 = 0.2654$, $p < 0.00001$).

Although BE and SBE decreased with increasing lactate, mean values remained above severe acidemia thresholds (–10 to –12 mmol/L).

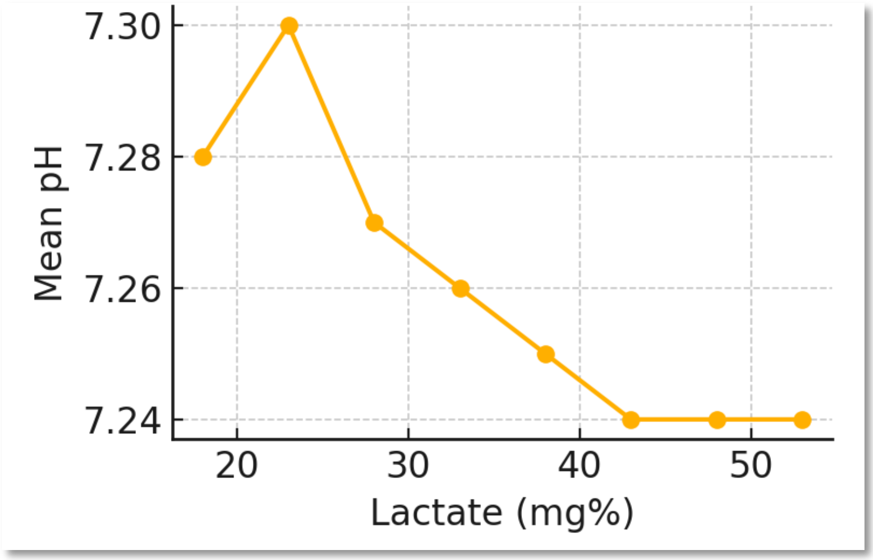

Figure 1: Correlation between Lactate and pH

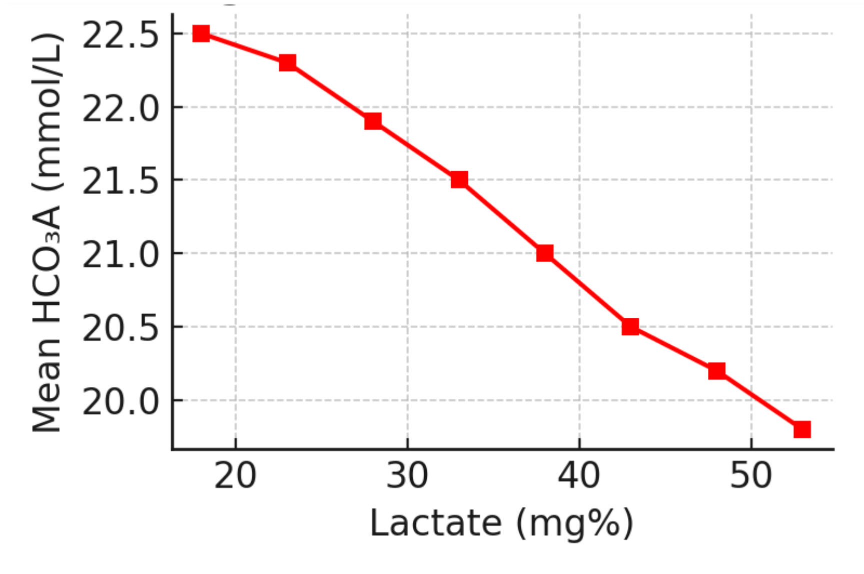

Figure 2: Correlation between Lactate and HCO_3A

Table 2: Comparison of Mean Umbilical Cord Blood Analytes Between NICU-Admitted Neonates and Interventional Deliveries

Parameter	NICU-Admitted Neonates (n = 29)	Interventional Deliveries (n = 38)
pH	7.25	7.26
H^+ (mmol/L)	56.13	54.76
pO_2 (mmHg)	22.09	21.46
pCO_2 (mmHg)	51.68	49.97
HCO_3A (mmol/L)	21.95	21.75
HCO_3S (mmol/L)	19.20	18.86
Base Excess (mmol/L)	–4.33	–4.64
Standard Base Excess (mmol/L)	–2.60	–2.73
Lactate (mg%)	38.57	34.89
Birth Weight (kg)	2.83	2.98

A Chi-square test comparing Apgar scores across lactate groups showed no significant association at 1 minute ($\chi^2 = 34.68$, $p = 0.4833$). Similarly, no statistically significant association was observed between 5-minute Apgar scores and lactate groups (χ^2 = 57.37, p = 0.0009; not significant). Most of neonates had Apgar scores ≥ 8 at 5 minutes, irrespective of lactate levels.

Discussion

This prospective study evaluated umbilical cord blood analytes and lactate levels in 264 term parturient with meconium-stained amniotic fluid (MSAF) and no additional risk factors. Lactate concentrations ranged from 16–55 mg%, with a mean of 35.14 mg% (3.89 ± 2.33 mmol/L). The largest proportion of neonates fell within the 36–40 mg% range, indicating a mild but consistent elevation, suggestive of a shift toward anaerobic metabolism even in low-risk settings. The observed mean lactate closely corresponds to previously established predictive cut-offs for neonatal morbidity: Tuuli et al. identified 3.9 mmol/L as the optimal threshold [6]; Geetha Damodaran et al. reported clinical decision limits of 27 mg% [7]; and Laila et al. found >3.6 mmol/L to have 93% diagnostic accuracy for hypoxia [8]. Lactate, a direct marker of anaerobic metabolism, rises earlier and persists longer than pH changes [9,10], offering superior predictive value compared to derived measures such as base excess.

Mode of delivery was predominantly vaginal (85.6%), with no significant association between delivery mode and lactate category, similar to prior observations [11,12].

The pH showed a weak but significant negative correlation with lactate (R = –0.17, p = 0.006), while H^+ correlated positively, consistent with physiological expectations [9,13,14]. There is no universal pH threshold for fetal acidosis, and its diagnostic utility is limited [15].

pO_2 and pCO_2 showed weak correlations with lactate, consistent with earlier reports indicating their limited role in predicting neonatal status [16-19]. Bicarbonate and base excess parameters showed moderate negative correlations with lactate (HCO_3A R = –0.51; BE R = –0.51; p < 0.00001), supporting the use of lactate as a more direct and discriminating marker of fetal metabolic status [9,20-22]. No statistically significant association was observed between lactate and Apgar scores, although higher lactate levels tended to be associated with lower scores, consistent with previous findings [23].

Subgroup analysis showed no significant deviations among NICU-admitted neonates or interventional deliveries. One neonatal death occurred, associated with high lactate and low Apgar, but attributable to probable congenital heart disease. Overall, cord blood lactate showed moderate correlations with metabolic parameters and weak correlations with pH, pO_2, and pCO_2, underscoring its value as a practical, early marker of fetal metabolic compromise in MSAF, even in low-risk labours. This study adds focused data to a limited body of literature by excluding high-risk factors, unlike previous studies [6,42].

Conclusion

Umbilical arterial cord blood lactate provides an objective and sensitive marker of fetal hypoxia and metabolic acidosis, even when conventional cord blood parameters appear normal. In meconium-stained amniotic fluid (MSAF) cases, lactate levels were consistently elevated, reflecting subtle metabolic compromise. Lactate measurement is simple, cost-effective, and requires minimal sample volume, making it a valuable adjunct in both clinical decision-making and medico-legal documentation. Larger prospective studies are warranted to further define its prognostic role, particularly in relation to long-term neonatal outcomes.

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