RESEARCH PAPER

Clinical Profile of NICU Admitted Neonates Born to Mothers with Meconium-Stained Amniotic Fluid in a Tertiary Care Hospital: A Retrospective Observational Study

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ABSTRACT
Background: Meconium-stained amniotic fluid (MSAF), resulting from in utero passage of meconium, often indicates fetal distress and is associated with a range of maternal risk factors and serious neonatal complications, including meconium aspiration syndrome (MAS), frequently requiring neonatal intensive care unit (NICU) admission. This study describes the clinical profile of NICU-admitted neonates with MSAF in a rural tertiary care hospital, with a specific focus on MAS.
Methods: A retrospective observational study of 60 neonates admitted to NICU with MSAF was conducted at a rural tertiary care hospital. Maternal and neonatal data were analyzed with descriptive statistics, t-tests, Chi-square/Fisher's exact and regression analysis, with $p \leq 0.05$ considered statistically significant.
Results: Of 60 neonates with MSAF were included, 53.4% developed MAS. Maternal pregnancy-induced hypertension (25%) and anemia (35%) were significantly associated with lower neonatal birth weights ($p<0.05$). Most infants were term (85%) with 45% low birth weight. Respiratory distress was the strongest MAS predictor ($p<0.001$), with additional association with low 1-min APGAR ($p=0.011$), clinical sepsis ($p=0.031$), and need for oxygen therapy, CPAP, and caffeine ($p<0.05$). Mean NICU stay was 7.3 ± 7.2 days, with lower gestational age predicting longer stay ($p<0.001$).
Conclusions: High rates of MAS highlight the need for careful perinatal monitoring and timely respiratory support. Addressing maternal conditions such as anemia and PIH will reduce neonatal vulnerability and improve outcomes in such a high-risk MSAF cohort.
Keywords: *Meconium-Stained Amniotic Fluid, Meconium Aspiration Syndrome, Neonates, Neonatal Intensive Care Unit, Rural Hospital*
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1. INTRODUCTION

The first stool of life, meconium, is composed of sloughed-off cells and secretions from the gastrointestinal tract, and ingested waste products from the amniotic fluid. It begins forming in the fetal intestines as early as the second trimester of pregnancy. The term 'meconium' originates from the Greek word mekonion, which means thick, greenish-brown poppy juice, reflecting its appearance and the belief that fetal exposure to it would lead to neonatal sleepiness[(1)].

In healthy full-term neonates, meconium is typically passed within 24 to 48 hours after birth, making it a key physiological indicator of fetal maturity[(2)]. In preterm neonates, the first stool passage is often delayed due to gastrointestinal immaturity. Conversely, their post-term counterparts are more likely to pass meconium in utero, leading to meconium-stained amniotic fluid (MSAF) and potential perinatal complications[(3)].

MSAF and its poor outcomes are commonly associated with a range of risk factors, including pregnancy-induced hypertension (PIH), maternal diabetes, oligohydramnios, vaginal breech delivery and prolonged labor[(4–6)]. These factors often disrupt the typical progression of pregnancy and delivery, increasing the likelihood of fetal distress and the expulsion of meconium into the amniotic fluid.

Complications arising from MSAF can be severe for the neonate, encompassing meconium aspiration syndrome (MAS), birth asphyxia, persistent pulmonary hypertension of newborn (PPHN), and a heightened risk of sepsis[(7–9)]. Such adverse outcomes frequently necessitate neonatal intensive care unit (NICU) admissions for advanced

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interventions, including assisted ventilation, surfactant therapy, and antibiotics[10,11]. The widespread nature of these complications stresses the critical need to promptly identify and manage MSAF, particularly in high-risk pregnancies.

This study aims to provide a detailed clinical profile of neonates admitted with MSAF in a rural tertiary care hospital's NICU, focusing on MAS. By systematically analyzing the associated risk factors, complications, therapeutic interventions, and short-term outcomes of these neonates, the study will contribute to the existing body of knowledge and thereby help in reducing neonatal morbidity and mortality.

2. MATERIALS AND METHODS

2.1 Type of Study

This retrospective observational study was performed at a tertiary care hospital catering to the rural population. After obtaining requisite institutional approval and ethical clearances, the institutional Medical Records Department was approached to retrieve the necessary records. A waiver of informed consent was granted as the study involved no direct contact with the subjects. Subject identification was done based only on unique IDs assigned to them, ensuring confidentiality throughout the study.

2.2 Study Population and Selection Criteria

NICU records for a two-year period were screened for neonates born to mothers with MSAF.

Inclusion criteria:

i. the availability of complete maternal medical and obstetric data

ii. the availability of complete neonatal data at three time points: admission, during NICU stay, and discharge.

From 309 NICU records, 68 neonates with MSAF were identified. Of these, 60 cases met the inclusion criteria.

2.3 Sample Size Determination

The required sample size of 42 was calculated assuming a prevalence of 5% (based on the lower end of the prevalence range), a margin of error of 5%, and a 95% confidence level, and further applying the finite population correction for the identified population of 68 MSAF neonates. To ensure maximum representativeness and analytical power, all 60 eligible cases with complete data were included in the final study sample.

2.4 Data Collection

The exact requirements for data were decided using a pre-validated proforma:

The proforma consisted of two sections:

i. **Maternal Sheet:** Documented demographic details, obstetric history (parity, premature rupture of membranes, mode of delivery, gestational diabetes, pregnancy-induced hypertension, prolonged labor), laboratory investigations (e.g., maternal hemoglobin) among other relevant records.

ii. **Neonate Sheet:** Included demographic and delivery data, NICU information (at admission, during stay, and at discharge), and clinical parameters such as gestational age (calculated via first trimester ultrasound), APGAR (Appearance, Pulse, Grimace, Activity, Respiration) scores, vitals and biochemical parameters. Imaging (e.g., chest x-ray, 2-D echo) and interventions (e.g., assisted ventilation, phototherapy, inotropes, sildenafil) were recorded. Development of complications (e.g., respiratory distress, meconium aspiration syndrome) and neonatal outcomes (discharge, referral, DAMA, death) were also noted.

After compiling and entering the data into a Microsoft Excel sheet, a thorough review was conducted to ensure accuracy and completeness. The data was then subjected to statistical analysis for interpretation.

2.5 Statistical Methods

Statistical analyses were performed using IBM SPSS Statistics (Version 26). A p-value of ≤ 0.05 was considered statistically significant.

Continuous variables were summarized using descriptive statistics, while independent-samples t-tests were performed to compare means between groups. Associations between categorical variables were explored using the Chi-square test, or Fisher's exact test in instances of low expected cell counts. To identify predictors of outcomes, univariate and multivariate regression analyses were conducted.

Results are presented in tables and figures for clarity and ease of interpretation.

3. RESULTS

A total of 60 neonates born to mothers with meconium-stained amniotic fluid (MSAF) were included in the study.

Meconium Aspiration Syndrome (MAS) was the most notable complication, developing in 53.4% of neonates (n=32). Its associations are analysed in depth in the following text.

3.1 Maternal Factors

90% mothers (n=54) were between 20–34 years of age (24.87 ± 4.48 years). Primigravida status was noted in 58.3% (n=35) of the cohort. Common maternal comorbidities included anemia (35%, n=21), PIH (25%, n=15), and a history of previous poor obstetric outcomes (15%, n=9). Premature rupture of membranes (PROM) and oligohydramnios were each observed in 6.7% of cases (n=4). Lower Segment Caesarean delivery (LSCS) was the most common mode of delivery (65%, n=39), followed by normal vaginal delivery (26.7%, n=16) and assisted delivery (8.3%, n=5).

Associations between maternal risk factors and MAS were analyzed in detail (Table 1, Figure 1). Of note, mothers with breech presentation showed a statistically significant ($\chi^2(1)=7.62$, $p=0.006$) but negative relationship with the development of MAS in the neonates.

Neonatal birth weight was significantly associated with PIH and maternal anemia. An independent samples t-test assuming equal variances indicated that neonates born to mothers with PIH had a significantly lower mean birth weight (2.21 ± 0.61 kg, n=15) compared to those born to mothers without PIH (2.69 ± 0.51 kg, n=45; t(58)=-2.95, p=0.005; 95% CI [-0.79, -0.15] kg). Similarly, neonates of anaemic mothers had a lower mean birth weight (2.36 ± 0.54 kg, n = 21) compared to those of non-anaemic mothers (2.68 ± 0.56 kg, n = 39; t(58) = -2.13, p = 0.037; 95% CI [-0.62, -0.02] kg) (Figure 2).

3.2 Neonatal Demographic Profile

The male-to-female ratio among the neonates was approximately 1.07:1. The majority of infants were term, with 85% (n=51) born full term and only 15% (n=9) preterm (<37 weeks; mean gestational age: 38.56 ± 1.85 weeks).

45% (n=27) of the neonates had low birth weight (<2.5 kg), including 10% who were very low birth weight (<1.5 kg). Birth Injuries, congenital anomalies and intra-uterine growth restriction (IUGR) were found in 3.3% (n=2), 8.3% (n=5) and 11.7% (n=7) of cases, respectively (Table 2).

3.3 Neonatal Clinical Profile

At 1-minute of life, 21.7% (n=13) of neonates had APGAR scores <7, improving to 91.7% (n=55) with APGAR ≥7 at 5 minutes. Birth asphyxia was clinically diagnosed as per 1-minute APGAR in the 21.7% (n=13) neonates.

Resuscitation measures required following birth included bag and mask ventilation, delivery room oxygen and delivery room continuous positive airway pressure (CPAP) in 15% (n=9), 8.3% (n=5) and 21.7% (n=13), respectively.

Respiratory distress was observed in 61.7% (n=37) neonates while clinical sepsis was seen in 23.3% (n=14). Other clinically notable findings included PPHN and Hypoxic Ischemic Encephalopathy (HIE) in 6.7% (n=4) and 1.7% (n=1) of neonates, respectively.

Further analysis was performed to explore associations between MAS and neonatal clinical factors (Table 3, Figure 3). It showed that 83.8% of neonates with respiratory distress developed MAS versus 4.3% without. Respiratory distress predictably emerged as the single strongest predictor of MAS with Chi-Square test revealing its significant association ($\chi^2(1)=35.96$, $p<0.001$; OR=113.7, 95% CI [12.77, 1011.96]).

Additionally, clinical sepsis was more common in MAS cases (34% vs 11%, $\chi^2(1)=4.67$, p=0.031; Fisher's exact p=0.037). Low 1 minute APGAR (<7), or birth asphyxia, was also more common in MAS cases (34% vs 7%, $\chi^2(1)=6.53$, p=0.011; Fisher's exact p=0.013). Approaching significance was the relationship between the development of PPHN in those developing MAS ($\chi^2(1)=3.75$, p=0.053; Fisher's exact p=0.116).

3.4 Therapeutic Interventions

In the critical care setting of NICU, respiratory support was given in the form of oxygen therapy (51.7%, n=31), CPAP (45%, n=27) and mechanical ventilation (3.3%, n=2).

Phototherapy (81.7%, n=49) and antibiotics (25%, n=15) were frequently used adjuncts. Caffeine therapy was administered in 15% of neonates (n=9). Sildenafil and inotropes were used in 6.7% (n=4) neonates, and blood transfusions and intravenous immunoglobulin were employed in 1.7% (n=1).

Analysis of treatment modalities and their association with MAS was conducted (Table 4). It revealed statistically significant association between MAS and the use of oxygen therapy ($\chi^2(1)=11.21$, p=0.001), CPAP ($\chi^2(1)=42.96$, $p<0.001$) and caffeine therapy ($\chi^2(1)=5.38$, p=0.020; Fisher's exact p=0.029) The associations with antibiotics ($\chi^2(1)=3.21$, p=0.073), inotropes ($\chi^2(1)=3.75$, p=0.053; Fisher's exact p=0.116), and sildenafil ($\chi^2(1)=3.75$, p=0.053; Fisher's exact p=0.116) were marginal.

Prolonged hospitalisation (>7 days) was markedly more common among caffeine-treated neonates than among those not given caffeine ($\chi^2(1)=9.80$, p=0.002; Fisher's exact p=0.005).

3.5 Clinical Outcomes and Hospital Stay

Most neonates (83.3%, n=50) were discharged without disability (DWOD). Adverse outcomes occurred in 16.7% of neonates including discharge with disability (DWD) (13.3%, n=8) and discharge against medical advice (DAMA) (3.3%, n=2).

The mean duration of NICU stay was 7.32 ± 7.22 days, with 25% (n=15) of neonates staying in the NICU for more than 7 days. Clinical outcomes in MAS vs non-MAS neonates were explored, with no statistically significant results (Table 5).

Multiple linear regression (N=60) showed a one kg lower birth weight was associated with a 2.77 day longer stay, with marginal statistical significance (B=-2.77, 95% CI [-5.60, 0.06], p=0.055). Each one week decrease in gestational age prolonged the NICU stay by 2.13 days (B=-2.13, 95% CI [-3.01, -1.26], $p<0.001$) (Figure 4). Together, the two variables explained 46.6% of the variance in length of stay ($R^2=0.466$; adjusted $R^2=0.447$; $F(2, 57)=24.9$, $p<0.001$).

Adding the 5 minute APGAR score ($\Delta R^2=0.019$, p=0.16) or maternal age ($\Delta R^2\approx0$, p=0.84) did not improve model fit, so the two-predictor model was retained.

4. DISCUSSION

The clinical profile of a NICU-admitted neonate with a history of MSAF was elaborately explored in our study.

The large incidence of MAS in more than half of the neonates in our high-risk cohort (53.4%), as well as its powerful association with key clinical indicators, justified a deep focus on interpreting predictors and outcomes

related to this severe complication. This strikingly high incidence is comparable to the 34% reported by Singh et al. in a similar high-risk population[12], but is substantially higher than the 6.3% in the cohort studied by Tolu et al[9]. This variability highlights the influence of underlying population characteristics, the prevalence of risk factors, and the effectiveness of obstetric and neonatal care in preventing the complication.

4.1 Maternal Factors

The release of meconium in utero is most often a physiological response to fetal stress, frequently precipitated by pre-existing maternal conditions. Available literature reveals a consistent association between MSAF and a high incidence of a constellation of maternal and obstetric risk factors such as maternal anemia and PIH[4,13], as confirmed by their high prevalence of 35% and 25%, respectively, in our cohort as well. The link between PIH and fetal stress is well known, arising from endothelial dysfunction and poor placental perfusion that leads to chronic fetal hypoxia[14]. Similarly, maternal anemia reduces the oxygen-carrying capacity of the blood, predisposing the foetus to chronic hypoxic stress[15].

A critical finding in our study was that while these maternal conditions were significantly associated with lower neonatal birth weight (p=0.005 and p=0.037, respectively), as usually observed in neonates[16,17], they did not directly predict the development of MAS. This suggests an indirect but clinically relevant contribution to producing more vulnerable neonates, evidenced by the high incidence of low birth weight (LBW) in our cohort, with 45% of neonates weighing <2.5 kg. Such evidences of association of MSAF with LBW are also stressed upon by Jain et al[18]. This vulnerability possibly lowers the threshold for decompensation in the face of intrapartum stress, thereby increasing the risk for the series of events leading to aspiration.

Our analysis revealed a statistically significant negative association between breech presentation and the development of MAS (p=0.006), a finding that contradicts literature identifying breech as a risk factor[6,19]. A possible explanation is that the majority of breech presentations in our cohort were likely managed with planned Lower Segment Caesarean Sections (LSCS). This planned and controlled delivery would evade the prolonged labor and fetal distress, thus appearing protective in our statistical analysis. This interpretation is supported by the high rate of LSCS (65%) in our cohort, as has been the trend in MSAF afflicted neonates evidenced by studies by Mundhra et al, Shukla and Swapna and Jain et al[13,14,18]. This hypothesis, however, requires further investigation.

4.2 Neonatal Demographic Profile

Our findings were demographically similar to other studies[4,13] in terms of the mean maternal age of 24.87 ± 4.48 years and neonatal gender ratio of 1.07 female to 1 male. Our cohort lacked any documentation of post-term deliveries. This could be due to proactive obstetric management of post-term labor, likely reducing the contradictory well-noted incidence of severe MSAF associated with placental insufficiency in advancing gestational pregnancies as showcased by Balchin et al. and Hiersch et al[6,20].

4.3 Neonatal Clinical Profile

Our findings confirm that the key predictors of MAS are the clinical signs of perinatal compromise. Respiratory distress was observed in 61.7% of the cohort, much similar to significant findings of 58.2% and 79% in studies by Shukla and Swapna, and Rafia Rashid[13,21]. It also emerged as the single strongest predictor of MAS (OR=113.7, p<0.001). This finding aligns with the foundational diagnostic criteria for MAS, as discussed by Monfredini et al[10]. It impresses upon the critical importance of recognizing any disturbed breathing pattern in a neonate born through MSAF as possibly an indicative sign of MAS in order for us to intervene and prevent adverse outcomes[22].

Low 1-minute APGAR scores were also significantly associated with MAS (p=0.011), highlighting the role of immediate postnatal adaptation. This establishes a causal chain of an underlying pathology (e.g., uteroplacental insufficiency) that leads to fetal stress (manifesting as MSAF), which results in acute intrapartum decompensation (evidenced by low APGAR scores), with this acute event triggering the aspiration that presents as MAS.

The marked incidence of clinical sepsis in 23.3% neonates as seen in our cohort adheres to its higher occurrence in the MSAF cohort in a study by Tolu et al[9]. Furthermore, the association between clinical sepsis and MAS development (p=0.031) reiterates that MAS, in addition to its mechanical pathophysiology, carries a crucial inflammatory component. This concept of MAS as an inflammatory disease has significant implications for understanding its multi-organ effects and guiding future therapeutic strategies.

4.4 Therapeutic Interventions

The strong association between MAS and the need for respiratory support like oxygen (p=0.001) and CPAP (p<0.001) as its cornerstone of management was an expected confirmation of the syndrome's primary pathology and the high resource dependency required to manage these neonates[10,22].

The role of caffeine in neonates with MAS is complex. We found that caffeine use was associated with MAS (p=0.020) and prolonged hospitalization (p=0.002). We interpret this finding not as causal, but as a reflection of its use in more clinically complex cases requiring intensive intervention, a recognized phenomenon in clinical literature[23]. The crucial question of whether caffeine is therapeutic, and not just a marker of severity, is being addressed by the ongoing CAPUCINO clinical trial (ClinicalTrials.gov identifier:NCT06972108; first posted on 14th May 2025), which will evaluate its impact on respiratory outcomes in infants with MAS[24].

4.5 Clinical Outcomes and Hospital Stay

Our study reported a mean NICU stay of 7.32 ± 7.22 days.

Our multiple regression analysis showcased that decrease in gestational age was the strongest predictor of a longer stay ($p<0.001$), with LBW showing a strong trend ($p=0.055$). This is consistent and reaffirms the principle that even in the context of an acute issue like MAS, the underlying maturity and physiological reserve of the neonate remain fundamental determinants of the clinical course[6,25]. While our regression model explained a substantial portion of the variance in NICU stay (Adjusted $R^2=0.447$), over half remains unaccounted for. This suggests the influence of other factors not included, such as the consistency of meconium[14], and complications like PPHN[8].

Our study further demonstrates positive clinical outcomes, with 83.3% of neonates discharged without disability and, critically, zero in-hospital mortality. The remaining 16.7% with adverse outcomes consisted of eight neonates with significant morbidity and two cases of discharge against medical advice (DAMA). An analysis of the morbidity points to three main etiologies: severe congenital conditions (e.g., Pierre Robin Sequence), birth injuries (e.g., brachial plexus injury), and sequelae of critical cardiorespiratory illness (e.g., PPHN). Our zero percent mortality rate is substantially lower than the 5-20% range reported elsewhere[4,12,13,21]. However, this comparison is limited by the two DAMA cases whose outcomes were lost to follow-up, representing an important consideration for our findings.

4.6 Strengths and Limitations

A key strength of this study is the detailed data collection spanning maternal, neonatal, therapeutic, and outcome parameters, allowing for a comprehensive analysis. However, being a single-center study with a small sample size (N=60), affects the statistical power of our findings.

5. CONCLUSION

In conclusion, this study paints a detailed picture of a high-risk MSAF cohort where over half of neonates developed MAS, predicted most strongly by respiratory distress and low APGAR scores. This highlights the need for careful perinatal monitoring and timely respiratory support for these neonates. Maternal factors, anemia and PIH, contributed to neonatal vulnerability by being associated with lower birth weight. The duration of NICU stay was hugely determined by gestational age and birth weight. Hence, strengthening obstetric and neonatal care is necessary to improve outcomes.

There is need for future research to build upon these findings through more robust, larger-scale studies to further explore the relationships between MSAF and neonatal and maternal parameters.

Ethics approval and consent to participate

The study was approved by the Institutional Ethics Committee of Symbiosis International (Deemed University) (approval reference number: SIU/IEC/373). A waiver of consent was granted by the committee, as this was a retrospective analysis of anonymized patient data.

Competing interests

The authors declare that they have no competing interests.

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FIGURE TITLES AND LEGENDS

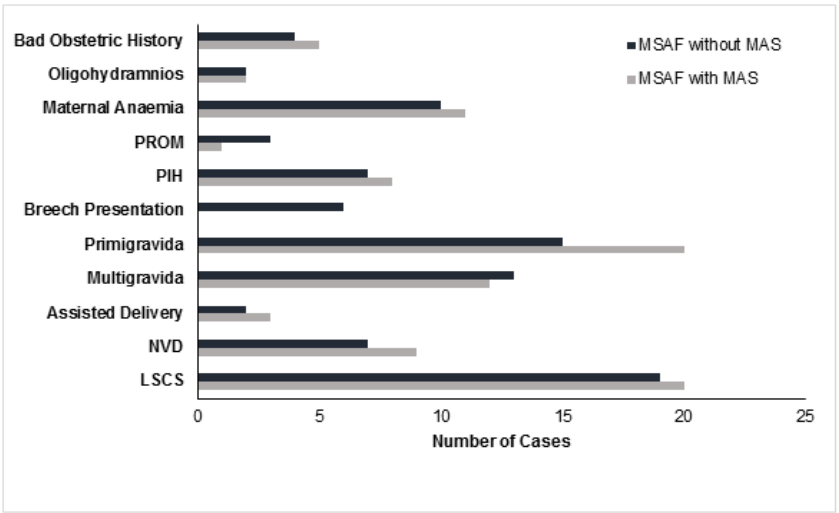

Figure 1. Maternal risk factors associated with meconium aspiration syndrome (MAS)

Legend: Horizontal bar graph showing maternal risk factors in neonates with and without MAS. Factors include bad obstetric history, oligohydramnios, maternal anemia, premature rupture of membranes (PROM), pregnancy-induced hypertension (PIH), breech presentation, parity (primigravida/multigravida), and mode of delivery [lower segment cesarean section (LSCS), normal vaginal delivery (NVD), assisted]. Grey and black bars represent MAS and non-MAS groups, respectively.

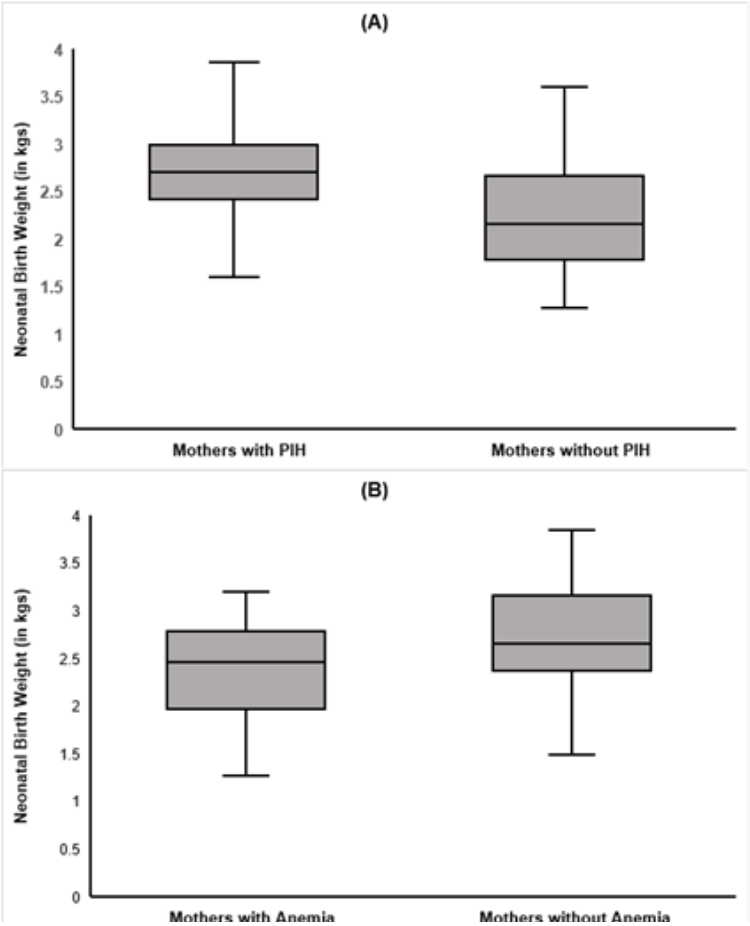

Figure 2. Effect of maternal comorbidities on neonatal birth weights

Legend: Box plots comparing neonatal birth weight distributions according to maternal conditions. Panel A: birth weights of neonates born to mothers with and without pregnancy-induced hypertension (PIH). Panel B: birth weights of neonates born to mothers with and without anemia. Boxes represent interquartile ranges, horizontal lines show medians, and whiskers indicate ranges.

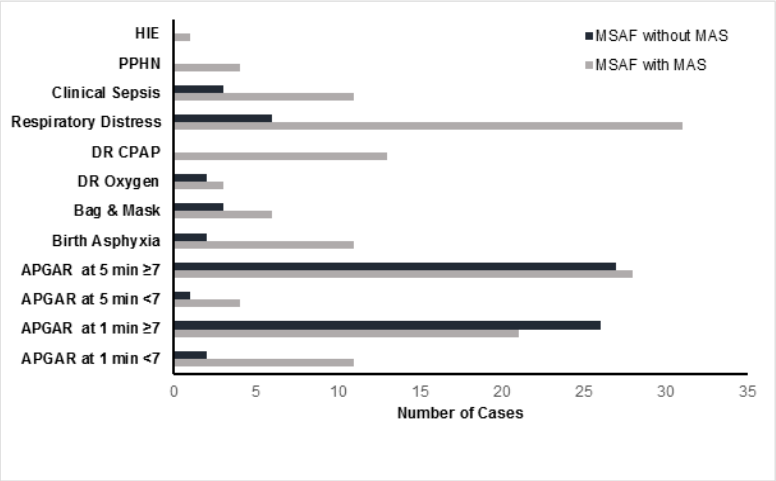

Figure 3. Neonatal clinical factors in MAS versus non-MAS groups

Legend: Horizontal bar graph illustrating neonatal clinical factors in MAS and non-MAS groups. Parameters include APGAR scores at 1 and 5 minutes, birth asphyxia, resuscitation measures (bag-mask ventilation, delivery room oxygen, CPAP), respiratory distress, clinical sepsis, persistent pulmonary hypertension of the newborn (PPHN), and hypoxic-ischemic encephalopathy (HIE). Grey and black bars represent MAS and non-MAS groups, respectively.

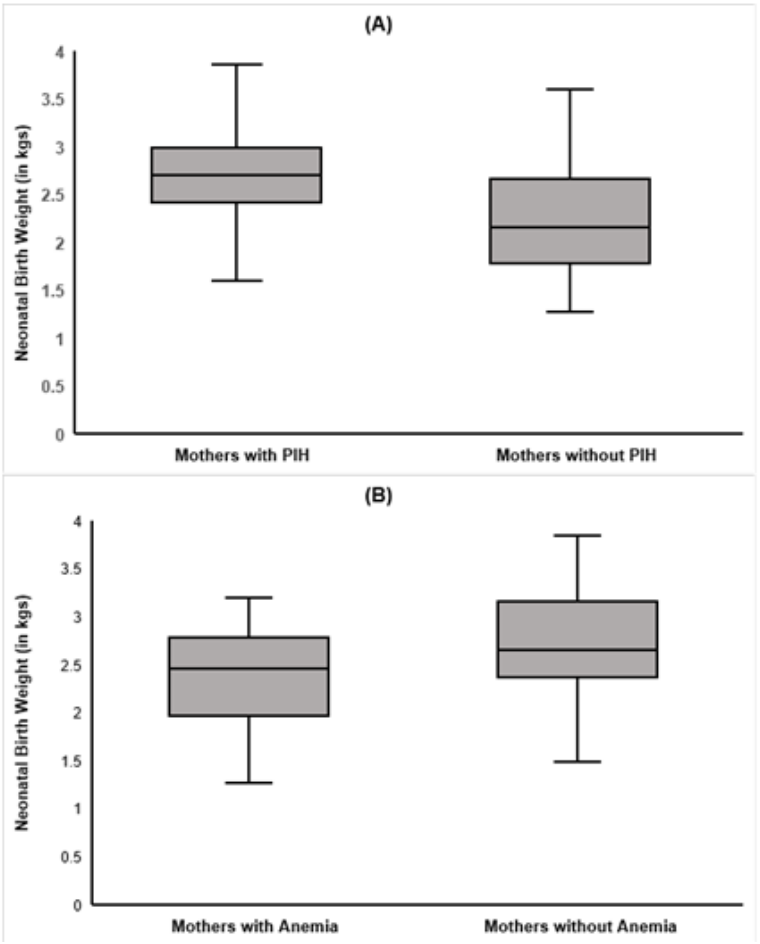

Figure 4. Predictors of NICU duration of stay in MSAF neonates

Legend: Scatterplots showing predictors of NICU stay duration. Panel A: neonatal birth weight (kg) versus NICU days. Panel B: gestational age (weeks) versus NICU days. Each dot represents an individual neonate; regression line demonstrates trend. Lower birth weights and gestational ages were associated with longer stays.

TABLES

Table 1. Maternal Risk Factors Associated with Meconium Aspiration Syndrome (MAS)

Maternal Risk Factors		MSAF (N = 60)		P-Value
		With MAS (n=32)	Without MAS (n=28)	
Mode of Delivery	LSCS	20	19	.900
	NVD	9	7	
	Assisted	3	2	
Gravida	Multigravida	12	13	.484
	Primigravida	20	15	
Breech Presentation		0	6	**.006***
PIH		8	7	1.000
PROM		1	3	.240
Maternal Anemia		11	10	.914
Oligohydramnios		2	2	.890
Bad Obstetric History		5	4	.885

Legend: Values are presented as n (number of neonates). p values derived from Chi-square test unless otherwise indicated. Abbreviations: LSCS - Lower Segment Caesarean Section, NVD - Normal Vaginal Delivery, PIH - Pregnancy-Induced Hypertension, PROM - Premature Rupture of Membranes. $p < 0.05$ considered statistically significant.

Table 2. Neonatal Demographic Factors

Demographic Factors		Frequency (n)	Percentage (%)	Mean ± Std. Dev. Or Ratio
Sex of Neonate	Male	31	51.7	1.07:1
	Female	29	48.3	
Gestational Age (in weeks)	<37 (Preterm)	9	15	38.56 ± 1.85
	37-42 (Full term)	51	85	
	>42 (Post term)	0	0	
Birth Weight (in Kgs)	1-1.5 (VLBW)	3	5	2.57 ± 0.57
	1.5-2.5 (LBW)	24	40	
	≥2.5 (NBW)	33	55	
APGAR (at 1 minute)	<7	13	21.7	6.82 ± 1.07 (Median/Mode = 7)
	≥7	47	78.3	
APGAR (at 5 minutes)	<7	5	8.3	7.88 ± 0.94 (Median/Mode = 8)
	≥7	55	91.7	
APGAR (at 10 minutes)	<7	0	0	8.88 ± 0.74 (Median/Mode = 9)
	≥7	**60**	**100**	

Legend: Values are presented as n (%) unless otherwise indicated. Ratio, mean ± standard deviation (SD), median/mode provided where applicable. Abbreviations: VLBW - Very Low Birth Weight, LBW - Low Birth Weight, NBW - Normal Birth Weight

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Table 3. Neonatal Clinical Factors Associated with Meconium Aspiration Syndrome (MAS)

Neonatal Factors		MSAF (N = 60)		P-Value
		With MAS (n=32)	Without MAS (n=28)	
APGAR (at 1 minute)	<7	11	2	**.011* (Fisher's exact p=0.013)**
	≥7	21	26	
APGAR (at 5 minute)	<7	4	1	.212
	≥7	28	27	
Birth Asphyxia		11	2	**.011* (Fisher's exact p=0.013)**
Resuscitation at Birth	Bag & Mask	6	3	.384
	DR Oxygen	3	2	.755
	DR CPAP	13	0	**.000***
Respiratory Distress		31	6	**.000***
Clinical Sepsis		11	3	**.031* (Fisher's exact p=0.037)**
PPHN		4	0	.053 (Fisher's exact p=0.116)
HIE		1	0	.346

Legend: Values are presented as n (number of neonates). p values derived from Chi-square test unless otherwise indicated. Fisher's exact test reported when expected cell counts <5. Abbreviations: DR - Delivery Room, CPAP - Continuous Positive Airway Pressure, PPHN - Persistent Pulmonary Hypertension of Newborn, HIE - Hypoxic Ischemic Encephalopathy. $p < 0.05$ considered statistically significant.

Table 4. Treatment Modalities Associated with Meconium Aspiration Syndrome (MAS)

Treatment Modalities	MSAF (N = 60)		P-Value
	With MAS (n=32)	Without MAS (n=28)	
Oxygen	23	8	**.001***
CPAP	27	0	**.000***
Mechanical Ventilation	2	0	.178
Antibiotics	11	4	.073
Inotropes	4	0	.053 (Fisher's exact p=0.116),
Sildenafil	4	0	.053 (Fisher's exact p=0.116),
Caffeine	8	1	**.020* (Fisher's exact p=0.029)**
IVIg	0	1	.281
Phototherapy	24	25	.154
Blood Transfusions	0	1	.281

Legend: Values are presented as n (number of neonates). p values derived from Chi-square test unless otherwise indicated. Fisher's exact test reported when expected cell counts <5. Abbreviations: CPAP - Continuous Positive Airway Pressure, IVIg - Intravenous Immunoglobulins. $p < 0.05$ considered statistically significant.

Table 5. Clinical Outcomes Associated with MAS

Clinical Outcomes		MSAF (N = 60)		P-Value
		With MAS (n=32)	Without MAS (n=28)	
Duration of Stay (in days)	≤7	21	24	.073
	>7	11	4	
Discharge Status	DWOD	28	22	.292
	DWD	4	4	
	DAMA	0	2	

Legend: Values are presented as n (number of neonates). p values derived from Chi-square test. Abbreviations: DWOD - Discharge Without Disability, DWD - Discharge With Disability, DAMA - Discharge Against Medical Advice. $p < 0.05$ considered statistically significant

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