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# Elevated Maternal Procalcitonin and Neonatal Outcomes in Preterm Premature Rupture of Membranes: A Prospective Cohort Study

**Alif Laila[1*], Quazi Rubyata Sania[2], Nasrin Akter[3], Shahzadi Afruza[4], Sanjana Rahman[5], Mohammad Rafiqul Islam Khan[6]**

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*Corresponding Author

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***ABSTRACT***

***Background****: Preterm premature rupture of membranes (PPROM) is a leading cause of neonatal morbidity and mortality. Maternal procalcitonin (PCT) has emerged as a potential biomarker for intra-amniotic infection, but its association with comprehensive neonatal outcomes beyond early-onset sepsis remains incompletely characterized.* ***Objective****: To evaluate the association between elevated maternal serum procalcitonin levels and adverse neonatal outcomes in women with PPROM.* ***Methods & Materials****: This prospective cohort study was conducted at Dhaka Medical College Hospital from January to December 2022. A total of 99 women with PPROM (24–34 weeks of gestation) were enrolled. Maternal serum PCT was measured on admission. Neonates were followed for clinical signs, laboratory parameters, blood culture-proven sepsis, NICU admission, and mortality within 3 postnatal days.* ***Results****: Elevated maternal PCT (>0.5 ng/ml) occurred in 61.6% of patients. The elevated PCT group showed significantly higher rates of lethargy (27.3% vs. 2.9%, p=0.003), respiratory distress (16.4% vs. 0.0%, p=0.010), fever (38.2% vs. 8.6%, p=0.002), and hypotonia (14.5% vs. 0.0%, p=0.017). Mean neonatal CRP was higher (4.0 vs. 2.9 mg/L, p=0.001) and ANC lower (4794.5 vs. 5489.9 cells/μL, p=0.047) in the elevated PCT group. Blood culture positivity (p=0.016), NICU admission (24.2%), and mortality (9.1%) occurred exclusively in the elevated PCT group.* ***Conclusion****: Elevated maternal serum procalcitonin in PPROM is significantly associated with adverse neonatal outcomes, including clinical sepsis, positive blood culture, NICU admission, and mortality. Maternal PCT is a valuable non-invasive predictor of poor neonatal prognosis.*

***Keywords:*** *C-reactive protein, Mortality, Neonatal outcomes, NICU admission, Preterm premature rupture of membranes, Procalcitonin*

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1. *Registrar, Department of Feto Maternal Medicine, Dhaka Medical College Hospital, Dhaka, Bangladesh (ORCID: 0009-0004-9400-3364)*
2. *Junior Consultant, Department of Gynecology & Obstetrics, Shaheed Suhrawardy Medical College Hospital, Dhaka, Bangladesh*
3. *Assistant Professor, Department of Feto Maternal Medicine, Shaheed Suhrawardy Medical College Hospital, Dhaka, Bangladesh*
4. *Assistant Professor, Department of Feto Maternal Medicine, Shaheed Suhrawardy Medical College Hospital, Dhaka, Bangladesh*
5. *Assistant Professor, Department of Feto Maternal Medicine, Dhaka Medical College Hospital, Dhaka, Bangladesh*
6. *Assistant Professor (Arthroplasty), Dhaka Medical College Hospital, Dhaka, Bangladesh*

## INTRODUCTION

Preterm premature rupture of membranes (PPROM), defined as the spontaneous rupture of the amniotic membranes before 37 weeks of gestation and before the onset of labor, affects approximately 2-4% of all pregnancies and accounts for nearly 30-40% of all preterm deliveries [1]. This obstetric emergency disrupts the protective barrier against ascending microbial invasion from the lower genital tract, predisposing both the mother and fetus to a cascade of infectious and inflammatory complications [2]. The consequences of PPROM extend far beyond the immediate risk of preterm birth, encompassing a spectrum of adverse neonatal outcomes including respiratory distress syndrome, intraventricular hemorrhage, necrotizing enterocolitis, and most notably, early-onset neonatal sepsis [3,4]. Neonatal sepsis remains one of the most devastating complications following PPROM, affecting 4% to 33% of preterm infants depending on gestational age at membrane rupture and latency period [5,6]. The pathophysiology of PPROM-related neonatal morbidity is primarily driven by ascending bacterial infection, which triggers a robust inflammatory response characterized by the release of pro-inflammatory cytokines such as interleukin-6, interleukin-8, and tumor necrosis factor-alpha [7]. This inflammatory cascade, while intended to combat infection, often results in collateral tissue damage, including injury to the fetal lungs, brain, and other developing organs [3]. Beyond culture-proven sepsis, neonates born following PPROM face a myriad of other adverse outcomes. Respiratory distress syndrome occurs in up to 50-60% of very preterm infants due to surfactant deficiency compounded by inflammation-induced lung injury [8]. Intraventricular hemorrhage, particularly severe grades III and IV, affects 15-25% of extremely preterm infants following PPROM and is associated with long-term neurodevelopmental impairment [9]. Furthermore, the need for neonatal intensive care unit (NICU) admission is nearly universal for preterm infants delivered after PPROM, with length of stay directly correlating with gestational age at delivery [4,10]. Given the substantial burden of PPROM-related neonatal morbidity, there is a critical need for early and reliable biomarkers that can identify pregnancies at the highest risk for adverse neonatal outcomes. Procalcitonin (PCT), a 116-amino acid precursor of the hormone calcitonin, has emerged as a promising biomarker for bacterial infection and sepsis [11]. Unlike C-reactive protein

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(CRP), which rises slowly over 12-24 hours following infection onset, PCT increases rapidly within 2-4 hours of bacterial endotoxin exposure and demonstrates superior specificity for bacterial versus viral or inflammatory conditions [12]. In neonatal populations, PCT has shown excellent diagnostic accuracy for early-onset sepsis, with sensitivity and specificity exceeding 85% in multiple studies [13,14]. However, most existing literature has focused on neonatal or cord blood PCT levels as predictors of EONS. The utility of maternal serum PCT as a predictor of broader neonatal outcomes—including clinical signs of sepsis, laboratory abnormalities, NICU admission requirements, and mortality—remains substantially underexplored [15]. Since ascending infection from the maternal genital tract is the primary driver of both maternal inflammation and neonatal morbidity, maternal PCT levels may serve as an early window into the severity of intra-amniotic infection and the consequent risk to the fetus [16]. Therefore, this prospective cohort study was designed to evaluate the association between elevated maternal serum procalcitonin levels and comprehensive neonatal outcomes in women with PPROM, including clinical signs of sepsis, laboratory parameters, blood culture positivity, NICU admission, and neonatal mortality.

## METHODS & MATERIALS

This prospective cohort study was conducted at the Fetal-Maternal Medicine unit of the Department of Obstetrics and Gynaecology, Dhaka Medical College Hospital, Bangladesh, from January 2022 to December 2022. A total of 99 pregnant women diagnosed with PPROM were enrolled using purposive sampling.

**Inclusion criteria:** Women aged 18-40 years with a singleton pregnancy, gestational age between 24 and 34 weeks confirmed by first-trimester ultrasonography, duration of membrane rupture to delivery less than 18 hours, and those providing written informed consent were included.

**Exclusion criteria:** Patients with pre-existing medical disorders (diabetes mellitus, hypertension, renal disease, chronic liver disease), COVID-19 positive status, fetal distress (heart rate <110 or >160 bpm), fetal anomalies, clinical evidence of chorioamnionitis, or neonatal death within 3 days of delivery were excluded.

**Study procedure:** On admission, 3 ml of maternal venous blood was collected aseptically. Serum procalcitonin was measured using the chemiluminescence sandwich technique on an immunochemistry autoanalyzer. Patients were followed until delivery. Neonates were examined within 72 hours of birth for clinical signs of sepsis. Laboratory parameters, including C-reactive protein, absolute neutrophil count, and blood culture, were obtained. NICU admission and mortality were recorded.

**Data analysis:** Data were analyzed using SPSS version 26.0. Continuous variables were compared using the unpaired t-test and presented as mean ± standard deviation. Categorical variables were compared using the chi-square test. A p-value <0.05 was considered statistically significant.

## RESULT

A total of 99 women with preterm premature rupture of membranes were enrolled in this study. The mean age of participants was 25.2±5.1 years, with the majority (53.5%) belonging to the 18-25 year's age group. Most patients were housewives (92.9%) and Muslim (96.0%), with a primary education level (55.6%) and a monthly income below 10,000 Taka (48.5%). The mean BMI was 23.6±3.5 kg/m$^2$ (*Table I*).

**Table I: Sociodemographic and baseline clinical profile of participants (*n*=99)**

| Variable | Category | n | % |
|---|---|---|---|
| Maternal age (years) | 18–25 | 53 | 53.5 |
| | 26–34 | 40 | 40.4 |
| | 35–40 | 6 | 6.1 |
| Education | Illiterate | 1 | 1.0 |
| | Primary | 55 | 55.6 |
| | SSC | 25 | 25.3 |
| | HSC | 14 | 14.1 |
| | Graduate | 4 | 4.0 |
| Religion | Islam | 95 | 96.0 |
| | Hindu | 4 | 4.0 |
| Occupation | Housewife | 92 | 92.9 |
| | Service | 7 | 7.1 |
| Monthly family income (Taka) | <10,000 | 48 | 48.5 |
| | 10,000–25,000 | 37 | 37.4 |
| | >25,000 | 14 | 14.1 |
| Body mass index (kg/m$^2$) | <18.5 (Underweight) | 4 | 4.0 |
| | 18.5–22.9 (Normal) | 52 | 52.5 |
| | 23–24.9 (Overweight) | 43 | 43.4 |

*Mean BMI: 23.6±3.5 kg/m$^2$ (range: 18.1–29.7)*

The two groups were comparable regarding baseline demographic and clinical characteristics, including age, education, occupation, income, parity, pulse, blood pressure, and BMI (all p>0.05) *Table II*.

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**Table II: Comparability of groups by maternal procalcitonin level at baseline**

| Parameter | Group A (>0.5 ng/ml) (n=61) | Group B (≤0.5 ng/ml) (n=38) | p-value |
|---|---|---|---|
| Age (years), mean ± SD | 25.1 ± 5.2 | 25.2 ± 5.0 | 0.977 |
| BMI (kg/m$^2$), mean ± SD | 22.2 ± 2.0 | 21.8 ± 2.0 | 0.357 |
| Primiparous, n (%) | 33 (54.1) | 21 (55.3) | 0.910 |
| Pulse rate (beats/min), mean ± SD | 80.8 ± 4.0 | 79.8 ± 3.4 | 0.209 |
| Systolic BP (mmHg), mean ± SD | 114.3 ± 9.0 | 113.9 ± 11.7 | 0.880 |
| Diastolic BP (mmHg), mean ± SD | 75.7 ± 5.5 | 75.8 ± 7.1 | 0.968 |

*Note: p-value derived from unpaired t-test for continuous variables and chi-square test for categorical variables*

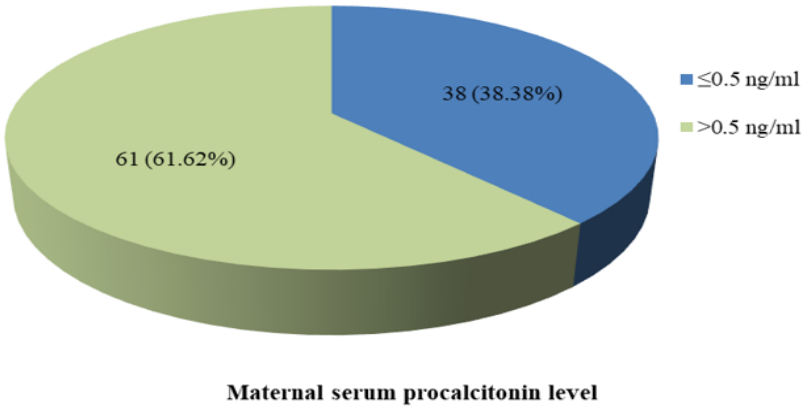


**Figure 1: Distribution of maternal serum procalcitonin level**

Elevated maternal serum procalcitonin level (>0.5 ng/ml) was observed in 61.6% of patients (Group A), while 38.4% had normal or decreased levels (Group B) *Figure 1*.

Regarding neonatal outcomes, 90 babies (90.9%) were alive after the 3rd postnatal day, while 9 (9.1%) died. Among the 90 live neonates, clinical signs of sepsis were significantly more frequent in Group A compared to Group B. Lethargy, poor cry, and refusal to suck occurred in 27.3% of Group A versus 2.9% of Group B (p=0.003). Respiratory distress, apnea, and gasping were observed in 16.4% of Group A and none in Group B (p=0.010). Fever was present in 38.2% of Group A versus 8.6% of Group B (p=0.002). Hypotonia with absent neonatal reflexes occurred in 14.5% of Group A and none in Group B (p=0.017) *Table III*.

**Table III: Neonatal clinical manifestations stratified by maternal procalcitonin level (n=90*)**

| Clinical sign | Group A (>0.5 ng/ml) (n=55 | Group B (≤0.5 ng/ml) (n=35) | p-value† |
|---|---|---|---|
| Lethargy, poor cry, refusal to suck | 15 (27.3) | 1 (2.9) | 0.003 |
| Respiratory distress, apnea, gasping | 9 (16.4) | 0 (0.0) | 0.010 |
| Fever | 21 (38.2) | 3 (8.6) | 0.002 |
| Hypotonia, absent neonatal reflexes | 8 (14.5) | 0 (0.0) | 0.017 |
| Poor perfusion, prolonged capillary refill time | 4 (7.3) | 0 (0.0) | 0.139 |

*Data presented as n (%) unless otherwise specified, †p-value from chi-square test, 9 babies died within 3 days of delivery and were excluded from this analysis*

Laboratory analysis revealed that mean neonatal CRP was significantly higher in Group A (4.0±1.6 mg/L) compared to Group B (2.9±1.2 mg/L, p=0.001). Mean absolute neutrophil count was significantly lower in Group A (4794.5±1753.4 cells/μL) than in Group B (5489.9±1318.3 cells/μL, p=0.047). No significant differences were observed for hemoglobin, WBC, ESR, platelet count, or immature-to-total neutrophil ratio (*Table IV*).

**Table IV: Laboratory parameters of neonates by maternal procalcitonin group (*n*=90*)**

| Laboratory parameter | Group A | Group B | p-value‡ |
|---|---|---|---|
| C-reactive protein (mg/L) | 4.0 ± 1.6 | 2.9 ± 1.2 | 0.001 |
| Absolute neutrophil count (cells/μL) | 4794.5 ± 1753.4 | 5489.9 ± 1318.3 | 0.047 |
| Hemoglobin (g/dL) | 14.6 ± 1.2 | 14.7 ± 1.2 | 0.628 |
| Total leukocyte count ($10^3$/m$^3$) | 9.2 ± 2.6 | 10.2 ± 1.9 | 0.060 |
| ESR (mm in 1st hour) | 12.8 ± 4.2 | 11.5 ± 3.5 | 0.117 |
| Platelet count ($10^3$/m$^3$) | 228.8 ± 88.3 | 250.1 ± 62.9 | 0.218 |
| Immature-to-total neutrophil ratio | 0.16 ± 0.06 | 0.14 ± 0.05 | 0.355 |

*Data presented as mean ± SD, ‡p-value from unpaired t-test, 9 babies died within 3 days of delivery and were excluded from this analysis*

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NICU admission was required for 24 babies (24.2%). All 8 cases of early-onset neonatal sepsis (100%) and all 9 deaths (100%) occurred in Group A. A significant positive correlation was observed between maternal procalcitonin and neonatal CRP (r=0.502, p=0.001), while a significant negative correlation was found with absolute neutrophil count (r=-0.362, p=0.001) *Table V*.

**Table V: Major neonatal outcomes according to maternal procalcitonin level (*n*=99)**

| Neonatal outcome | Group (n=61) | Group B (n=38) | p-value§ |
|---|---|---|---|
| Positive blood culture, n (%) | 8 (13.1) | 0 (0.0) | 0.016 |
| Early-onset neonatal sepsis, n (%) | 8 (13.1) | 0 (0.0) | 0.016 |
| NICU admission required, n (%) | 24 (39.3) | 0 (0.0) | <0.001 |
| Neonatal death (within 72 hours), n (%) | 9 (14.8) | 0 (0.0) | 0.011 |

*§p-value from chi-square test, Relative risk for EONS: 1.74 (95% CI: 1.45–2.10)*

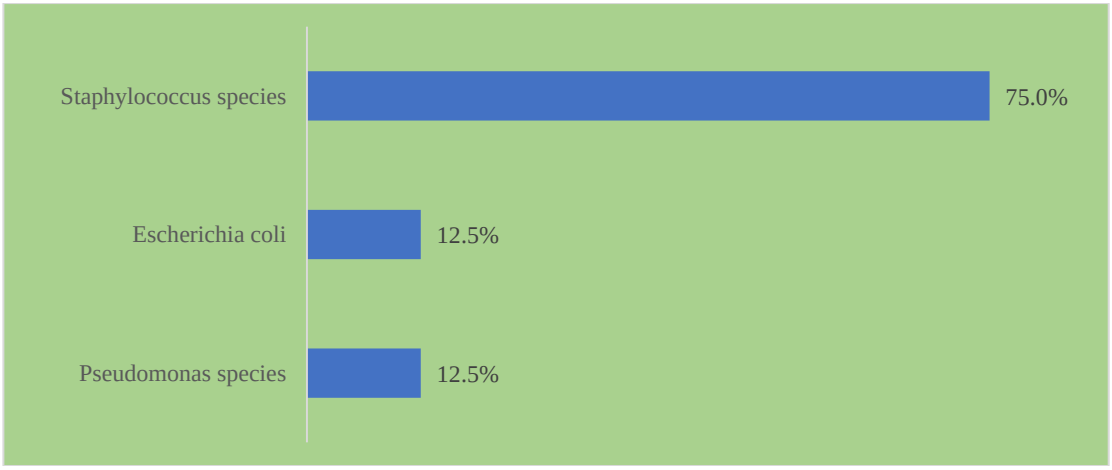


**Figure 3: Distribution of bacterial isolates from neonatal blood culture (*n*=8)**

Blood culture positivity was found in 8 neonates (8.9% of total, 14.5% of Group A), occurring exclusively in the elevated maternal PCT group (p=0.016). Among isolates, Staphylococcus was the most common (75.0%), followed by Pseudomonas (12.5%) and Escherichia coli (12.5%) *Figure 3*.

## DISCUSSION

In this prospective cohort study of 99 women with preterm premature rupture of membranes, elevated maternal serum procalcitonin (>0.5 ng/ml) was significantly associated with a spectrum of adverse neonatal outcomes, including clinical signs of sepsis, abnormal laboratory parameters, positive blood culture, NICU admission, and neonatal mortality. The elevated PCT group constituted 61.6% of the cohort, and all adverse outcomes occurred exclusively or predominantly within this group, underscoring the predictive value of this biomarker. The finding that elevated maternal PCT was associated with significantly higher rates of neonatal clinical signs-lethargy (27.3% vs. 2.9%, p=0.003), respiratory distress (16.4% vs. 0.0%, p=0.010), fever (38.2% vs. 8.6%, p=0.002), and hypotonia (14.5% vs. 0.0%, p=0.017)-is clinically important. These nonspecific manifestations of neonatal sepsis are often the earliest indicators of systemic infection, yet they are easily overlooked or attributed to prematurity [3,7]. These data suggest that maternal PCT elevation should prompt heightened vigilance for these signs, potentially enabling earlier intervention. Previous research has emphasized that up to 50% of culture-proven EONS cases may be asymptomatic at birth, making risk-stratification tools like maternal PCT invaluable [3]. The laboratory findings further support the association between maternal PCT and neonatal inflammatory status. Neonates in the elevated maternal PCT group had significantly higher mean CRP levels (4.0±1.6 vs. 2.9±1.2 mg/L, p=0.001) and a positive correlation between maternal PCT and neonatal CRP (r=0.502, p=0.001). This correlation suggests that maternal inflammatory response directly reflects or precedes neonatal systemic inflammation. Earlier studies have reported similar findings, demonstrating that the combination of PCT and CRP improves diagnostic accuracy for EONS [14,15]. The lower absolute neutrophil count observed in the elevated PCT group (4794.5 vs. 5489.9 cells/µL, p=0.047) is consistent with neonatal neutropenia in severe sepsis, resulting from increased neutrophil consumption and depletion of the bone marrow storage pool [16,17]. Blood culture positivity occurred exclusively in the elevated maternal PCT group (14.5% vs. 0.0%, p=0.016), with a relative risk of 1.74 for EONS. This finding aligns with previous reports that maternal inflammatory markers are strong predictors of culture-proven neonatal sepsis following PPROM [5,6]. The predominance of Staphylococcus species (75%) in this study differs from Western literature, where Group B Streptococcus and Escherichia coli are more common [17]. This variation likely reflects regional differences in microbial epidemiology, antibiotic resistance patterns, and possibly hospital-acquired colonization, highlighting the need for local surveillance data. The finding that NICU admission was required in 39.3% of neonates in the elevated maternal PCT group compared to none in the normal PCT group (p<0.001) has significant resource implications. Previous research has reported that NICU admission is nearly universal for preterm infants following PPROM, with length of stay inversely correlated with gestational age [4,10]. These data suggest that maternal PCT may help identify which PPROM pregnancies are at the highest risk, allowing for optimized

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resource allocation and advanced preparation for neonatal intensive care. Neonatal mortality within 3 days occurred in 9.1% of the cohort, exclusively in the elevated maternal PCT group (14.8% vs. 0.0%, p=0.011). This mortality rate is comparable to the 15-25% case fatality rate reported for culture-proven EONS in very low birth weight infants [3,6]. Previous work has demonstrated that extreme prematurity combined with sepsis carries a particularly high mortality risk [18]. These findings suggest that maternal PCT may serve as an early warning signal for potentially fatal neonatal outcomes. The biological plausibility of these findings is strong. Ascending bacterial infection following membrane rupture triggers the release of bacterial endotoxins and pro-inflammatory cytokines, including interleukin-6 and tumor necrosis factor-alpha, which induce PCT production [19,20]. This maternal inflammatory response precedes and predicts the severity of fetal and neonatal involvement [21,22]. Limitations of this study include the single-center design, modest sample size, exclusion of patients with prolonged rupture-to-delivery intervals (>18 hours), and lack of long-term neurodevelopmental follow-up. Future multicenter studies with larger cohorts are needed to validate optimal PCT cutoff values across different gestational age strata [23,24].

## LIMITATIONS

This study has several limitations: single-center design, modest sample size, exclusion of patients with prolonged rupture-to-delivery intervals (>18 hours), lack of serial maternal PCT measurements, and absence of long-term neurodevelopmental follow-up data.

## CONCLUSION

This study demonstrates that elevated maternal serum procalcitonin (>0.5 ng/ml) in PPROM is significantly associated with adverse neonatal outcomes, including clinical sepsis, positive blood culture, NICU admission, and mortality. Maternal PCT is a valuable, non-invasive predictor of poor neonatal prognosis. Routine maternal PCT measurement at admission may guide risk stratification, optimize resource allocation, and improve neonatal outcomes in resource-limited settings.

## RECOMMENDATION

Routine maternal serum procalcitonin measurement should be integrated into PPROM management protocols. Multicenter prospective studies with larger samples are needed to validate optimal cutoff values across gestational ages and establish PCT-guided neonatal care algorithms.

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