
RESEARCH ARTICLE

Duration of Prelabor Rupture of Membranes and Early-Onset Neonatal Sepsis: A Retrospective Cohort with Risk Model

Huda Adnan Sahib^{1*} and Roua Hameed Kadhem²

¹ *Obstetrics and Gynecology Department, College of Medicine, University of Thi-Qar, Al-Nasiriyah, 64001, Iraq*

² *Department of Pediatrics, College of Medicine, Thi-Qar University, Al-Nasiriyah, 64001, Iraq*

Corresponding Author: Huda Adnan Sahib, **E-mail:** huda-abd@utq.edu.iq

ABSTRACT

Prelabor rupture of membranes (PROM) is an established risk factor for early-onset neonatal sepsis (EONS), but the quantitative relationship between rupture-to-delivery latency and EONS in resource-constrained settings without universal group B streptococcus (GBS) screening is incompletely characterized, and contemporary data from southern Iraq are absent from the indexed literature. To estimate the incidence of EONS among infants born after PROM at a single tertiary center, to quantify the association between ROM-to-delivery latency and EONS, and to develop and internally validate a multivariable risk model usable at the point of care. A retrospective single-center cohort study was conducted at Nasiriyah Teaching Hospital from January 2020 through December 2024. Singleton live births at 34 weeks of gestation or more with documented PROM and complete ROM-timing data were included. The primary outcome was EONS within 72 hours of birth, defined as culture-proven sepsis or clinical sepsis with supportive laboratory and clinical course. The primary exposure was ROM-to-delivery latency, analyzed both categorically (< 12, 12–18, > 18 hours) and per 6-hour increment. Multivariable logistic regression identified independent predictors; discrimination was assessed by the area under the receiver operating characteristic curve (AUC) with 1,000-resample bootstrap internal validation, and reporting followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) and Transparent Reporting of a Multivariable Prediction Model for Individual Prognosis or Diagnosis (TRIPOD) statements. Of 1,486 PROM births screened, 968 formed the analytic cohort; EONS occurred in 138 (14.3%, 95% confidence interval [CI] 12.1–16.7%), of which 37 (26.8%) were culture-proven, predominantly *Klebsiella* species and GBS. ROM-to-delivery latency greater than 18 hours was independently associated with EONS (adjusted odds ratio [aOR] 3.42, 95% CI 2.11–5.54), with risk rising approximately linearly (aOR 1.27 per 6-hour increment, 95% CI 1.14–1.42). The combined model achieved AUC 0.84 (95% CI 0.80–0.88); bootstrap-corrected AUC was 0.82. EONS risk rose progressively with ROM-to-delivery latency. A six-variable model identified high-risk newborns at the point of care and may inform surveillance and prophylaxis decisions where universal GBS screening is unavailable.

KEYWORDS

Prelabor rupture of membranes; early-onset neonatal sepsis; latency; chorioamnionitis; risk model; predictors; neonatal infection

ARTICLE INFORMATION

ACCEPTED: 21 April 2026

PUBLISHED: 20 May 2026

DOI: 10.32996/jmhs.2027.7.7.14

1. Introduction

Prelabor rupture of membranes (PROM) rupture of the amniotic and chorionic membranes before the onset of labor complicates approximately 5–10% of pregnancies and is present in 30–40% of preterm births [1,2,18]. The loss of the membrane barrier permits ascending colonization of the amniotic cavity, and the duration between rupture and delivery (the latency period) is a biologically plausible determinant of intra-amniotic infection and subsequent early-onset neonatal sepsis (EONS), defined as invasive bacterial infection presenting within the first 72 hours of life [3,4,13,22].

The quantitative relationship between latency and EONS has been examined in several large cohorts. A cohort of 113,568 singleton term infants reported that the risk of neonatal sepsis increased independently and nearly linearly with rupture duration up to 36 hours, with an odds ratio of approximately 1.29 for each 6-hour increase [5]. A multicenter prospective cohort of term PROM pregnancies found that rates of early-onset neonatal pneumonia and sepsis rose significantly beyond 16–18 hours of latency [6]. Case-cohort data have reported adjusted odds ratios for neonatal sepsis of approximately 3 to 7 across latency thresholds of 15–48 hours [7], and a five-year review of births after PROM longer than 18 hours reported a culture-proven EONS rate of approximately 4%, with *Klebsiella pneumoniae* and *Pseudomonas aeruginosa* predominating in that setting [8].

Two considerations limit the direct transfer of these estimates to the present setting. First, much of the foundational evidence derives from high-income systems with universal antenatal group B streptococcus (GBS) screening and intrapartum antibiotic prophylaxis; in settings where universal screening is not standard practice, the pathogen distribution and the latency–risk relationship may differ, and locally derived estimates are required to inform surveillance and empirical-antibiotic policy [8,9]. Second, contemporary cohort data from southern Iraq a region with a high birth rate and a resource-constrained perinatal pathway are essentially absent from the indexed international literature, leaving local clinicians without a calibrated, point-of-care risk estimate for the asymptomatic newborn after PROM [17].

This study had three objectives: to estimate the incidence and microbiology of EONS among infants born after PROM at 34 weeks of gestation or more at Nasiriyah Teaching Hospital; to quantify the association between ROM-to-delivery latency and EONS, modeled both categorically and as a continuous per-6-hour increment; and to develop and internally validate a parsimonious multivariable risk model for EONS usable at the point of care [16]. The contribution is the first contemporary single-center cohort from Thi-Qar Province to characterize the latency–EONS relationship and to provide a locally calibrated risk model in a setting without universal GBS screening.

2. Patients and methods

2.1 Study design and setting

A retrospective single-center cohort study was conducted at the Departments of Obstetrics and Gynecology and of Pediatrics/Neonatology of Nasiriyah Teaching Hospital, a regional referral maternity and neonatal center serving Thi-Qar Province in southern Iraq, from 1 January 2020 through 31 December 2024 (60 months). The study protocol was approved by the Institutional Review Board / Research Ethics Committee of [Institution] (approval number [XXXX/2024]); the requirement for individual informed consent was waived owing to the retrospective design and the absence of identifiable patient contact. Reporting followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement and the Transparent Reporting of a Multivariable Prediction Model for Individual Prognosis or Diagnosis (TRIPOD) statement [10,11].

2.2 Participants and eligibility

Eligible records were singleton live births at a gestational age of 34 completed weeks or more with documented PROM and complete documentation of the time of membrane rupture and the time of delivery. PROM was defined as rupture of membranes before the onset of regular uterine contractions, ascertained from the obstetric record by sterile speculum examination, pooling, or confirmatory testing. Exclusion criteria were: gestational age below 34 weeks; missing or unreliable ROM-timing data; multiple gestation; major congenital anomaly; maternal therapeutic antibiotic exposure for more than 48 hours before rupture (as distinct from intrapartum prophylaxis); incomplete neonatal records; and neonatal transfer before 72 hours precluding outcome ascertainment. Cohort selection is summarized in Figure 1.

2.3 Exposure definition

The primary exposure was the ROM-to-delivery latency, defined as the interval in hours between documented membrane rupture and delivery. Latency was analyzed both as a categorical variable (< 12 hours, 12–18 hours, > 18 hours, with < 12 hours as the reference) and as a continuous variable expressed per 6-hour increment, mirroring the metric used in the principal published cohorts to facilitate comparison [5]. The 18-hour threshold was pre-specified as the principal clinical cut-off because it is widely used to define prolonged rupture in EONS risk assessment.

2.4 Outcome definition

The primary outcome was EONS within 72 hours of birth. Culture-proven EONS was defined as the isolation of a recognized bacterial pathogen from blood or cerebrospinal fluid. Clinical EONS was defined, in the absence of a positive culture, as a compatible clinical course requiring at least 5 days of antibiotic therapy together with at least two supportive abnormal laboratory findings (C-reactive protein, immature-to-total neutrophil ratio, or abnormal absolute neutrophil count) adjudicated by two clinicians (one neonatologist and one obstetrician) blinded to the exposure classification. Infants not meeting either definition and with an uncomplicated course were classified as no EONS.

2.5 Candidate predictors

Pre-specified candidate predictors were drawn from the published literature and from variables routinely recorded in the linked obstetric and neonatal records: ROM-to-delivery latency category and per-6-hour increment; maternal intrapartum fever (temperature ≥ 38.0 °C); maternal C-reactive protein at admission; maternal leukocytosis (white blood cell count $> 15 \times 10^9/L$); number of digital vaginal examinations after rupture; gestational age category (34–36 versus ≥ 37 weeks); meconium-stained amniotic fluid; and 5-minute Apgar score below 7. Variables were extracted from the institutional records by two reviewers using a standardized form, with discrepancies resolved by consensus.

2.6 Sample size

Sample size was governed by the multivariable model using the events-per-variable principle of at least 10 outcome events per candidate predictor. With 10 candidate predictors and an anticipated EONS incidence of approximately 12–15%, a minimum of approximately 700 mother–infant pairs was required to provide adequate events. The achieved analytic cohort of 968 with 138 EONS events provided 13.8 events per variable, exceeding the conventional threshold.

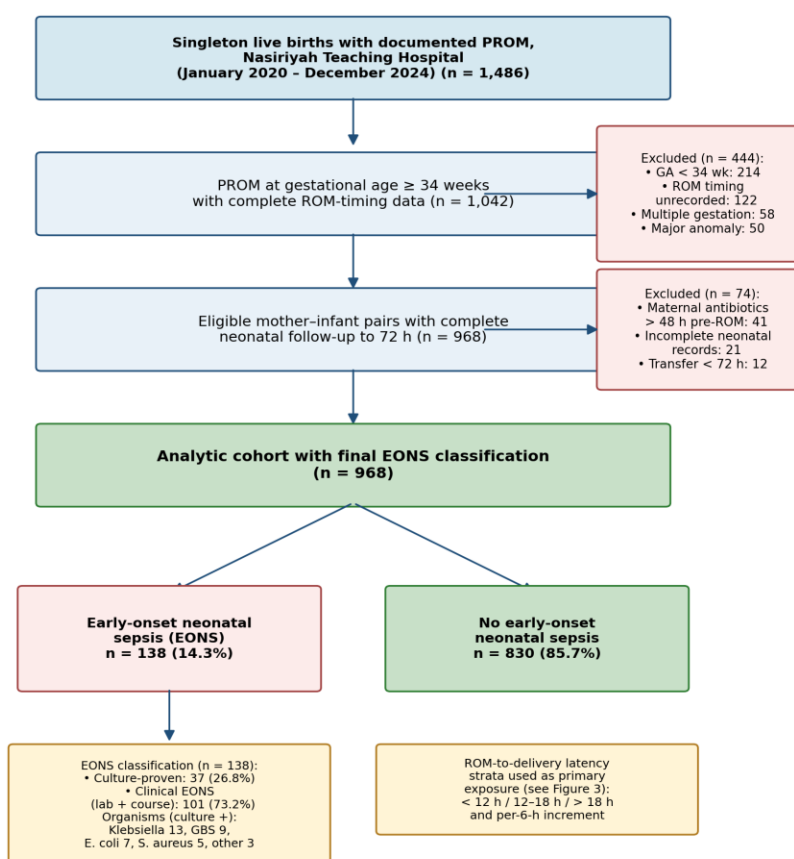
2.7 Statistical analysis

Continuous variables were summarized as mean $\pm$ standard deviation (SD) or median with interquartile range (IQR); categorical variables as counts and percentages. Univariable comparisons between EONS and no-EONS groups used the chi-squared or Fisher exact test for categorical variables and the Student t test or Mann–Whitney U test for continuous variables. Candidate variables with univariable $p < 0.20$ were entered into multivariable logistic regression with backward elimination retaining variables at $p < 0.05$. Adjusted odds ratios (aORs) with 95% confidence intervals (CIs) are reported. Discrimination was assessed by the AUC with 95% CI by the DeLong method, and AUCs were compared between the combined model and single predictors using DeLong's test [12]. Calibration was assessed by the Hosmer–Lemeshow goodness-of-fit test, and multicollinearity by variance inflation factors (VIF; threshold > 5). Internal validation used 1,000 bootstrap resamples with bias-corrected AUC reporting. Records with greater than 10% missingness on the primary predictors were excluded; a multiple-imputation sensitivity analysis ($m = 10$) was pre-specified. Analyses used IBM SPSS Statistics version 27.0 (IBM Corp., Armonk, NY) and R version 4.3 (R Foundation for Statistical Computing, Vienna, Austria) with the pROC and rms packages. Two-sided $p < 0.05$ was considered significant.

3. Results

3.1 Cohort assembly and baseline characteristics

During the 60-month study period, 1,486 singleton live births with documented PROM were screened. After exclusion of 444 records (214 below 34 weeks, 122 with unrecorded ROM timing, 58 multiple gestations, and 50 with major anomaly) and 74 further exclusions (41 maternal antibiotic exposure exceeding 48 hours before rupture, 21 incomplete neonatal records, and 12 transferred before 72 hours), 968 mother–infant pairs formed the analytic cohort (**see Figure 1**). Baseline characteristics are summarized in **Table 1**. The mean maternal age was 27.4 ± 5.9 years and the mean gestational age was 38.1 ± 1.8 weeks, with 17.6% delivering at 34–36 weeks. The median ROM-to-delivery latency was 10.4 hours (IQR 5.8–19.2); 28.1% of pregnancies had a latency exceeding 18 hours. Maternal intrapartum fever was documented in 8.9% and maternal admission C-reactive protein was 20 mg/L or higher in 22.3%.

Figure 1. Study cohort flow: PROM duration and early-onset neonatal sepsis, Nasiriyah Teaching Hospital, 2020-2024**Table 1. Baseline characteristics of the analytic cohort (n = 968).**

Characteristic	Value
Maternal age, mean ± SD (years)	27.4 ± 5.9
Nulliparous, n (%)	402 (41.5%)
Gestational age, mean ± SD (weeks)	38.1 ± 1.8
Gestational age 34–36 wk, n (%)	170 (17.6%)
ROM-to-delivery latency, median (IQR) (h)	10.4 (5.8–19.2)
Latency < 12 h, n (%)	515 (53.2%)
Latency 12–18 h, n (%)	181 (18.7%)

Latency > 18 h, n (%)	272 (28.1%)
Maternal intrapartum fever $\geq 38^{\circ}\text{C}$, n (%)	86 (8.9%)
Maternal CRP ≥ 20 mg/L at admission, n (%)	216 (22.3%)
Maternal leukocytosis $> 15 \times 10^9/\text{L}$, n (%)	198 (20.5%)
≥ 4 digital vaginal examinations, n (%)	241 (24.9%)
Cesarean delivery, n (%)	339 (35.0%)
Meconium-stained liquor, n (%)	142 (14.7%)
5-minute Apgar < 7, n (%)	97 (10.0%)
Early-onset neonatal sepsis, n (%)	138 (14.3%)

CRP = C-reactive protein; IQR = interquartile range; ROM = rupture of membranes; SD = standard deviation.

3.2 Incidence and microbiology of EONS

EONS occurred in 138 of 968 infants (14.3%, 95% CI 12.1–16.7%). Of these, 37 (26.8%) were culture-proven and 101 (73.2%) were clinical EONS. Among culture-proven cases, the predominant organisms were *Klebsiella* species (n = 13, 35.1%), GBS (n = 9, 24.3%), *Escherichia coli* (n = 7, 18.9%), *Staphylococcus aureus* (n = 5, 13.5%), and other organisms (n = 3, 8.1%). The predominance of *Klebsiella* species over GBS is consistent with the reported microbiology of EONS after prolonged rupture in settings without universal antenatal GBS screening and intrapartum prophylaxis.

3.3 Latency and univariable predictors

EONS incidence rose monotonically across latency strata: 6.8% for latency below 12 hours, 13.1% for 12–18 hours, and 27.9% for latency exceeding 18 hours ($p < 0.001$ for trend) (see Table 2). In univariable analysis, EONS was significantly associated with ROM-to-delivery latency exceeding 18 hours, maternal intrapartum fever, maternal C-reactive protein of 20 mg/L or higher, maternal leukocytosis, four or more digital vaginal examinations, gestational age 34–36 weeks, and a 5-minute Apgar score below 7. Maternal age, mode of delivery, and meconium-stained liquor did not reach the pre-specified univariable threshold.

Table 2. Early-onset neonatal sepsis incidence by ROM-to-delivery latency stratum.

Latency stratum	n	EONS, n (%)	Crude OR (95% CI)
< 12 hours (reference)	515	35 (6.8%)	1.00
12–18 hours	181	24 (13.3%)	2.10 (1.21–3.63)
> 18 hours	272	79 (29.0%)	5.62 (3.66–8.63)
Per 6-hour increment	968	—	1.34 (1.22–1.47)

3.4 Multivariable analysis and risk model

Multivariable logistic regression retained six independent predictors of EONS (see Figure 3 and Table 3). ROM-to-delivery latency exceeding 18 hours, relative to less than 12 hours, was the strongest predictor (aOR 3.42, 95% CI 2.11–5.54, $p < 0.001$); latency of 12–18 hours conferred intermediate risk (aOR 1.86, 95% CI 1.18–2.93, $p = 0.007$). Modeled continuously, each 6-hour increment in latency was associated with an aOR of 1.27 (95% CI 1.14–1.42, $p < 0.001$), indicating an approximately linear dose-response relationship consistent with the principal published term cohort [5]. Maternal intrapartum fever (aOR 2.98, 95% CI 1.74–5.11),

maternal C-reactive protein of 20 mg/L or higher (aOR 2.41, 95% CI 1.46–3.98), maternal leukocytosis (aOR 2.05, 95% CI 1.21–3.47), and gestational age 34–36 weeks (aOR 2.33, 95% CI 1.42–3.82) were independently associated with EONS. Meconium-stained liquor did not retain independent significance after adjustment. All variance inflation factors were below 2.0, and the Hosmer–Lemeshow test was non-significant ($\chi^2 = 6.8$, $p = 0.56$), indicating acceptable calibration.

1) Figure 3. Adjusted odds ratios for early-onset neonatal sepsis.

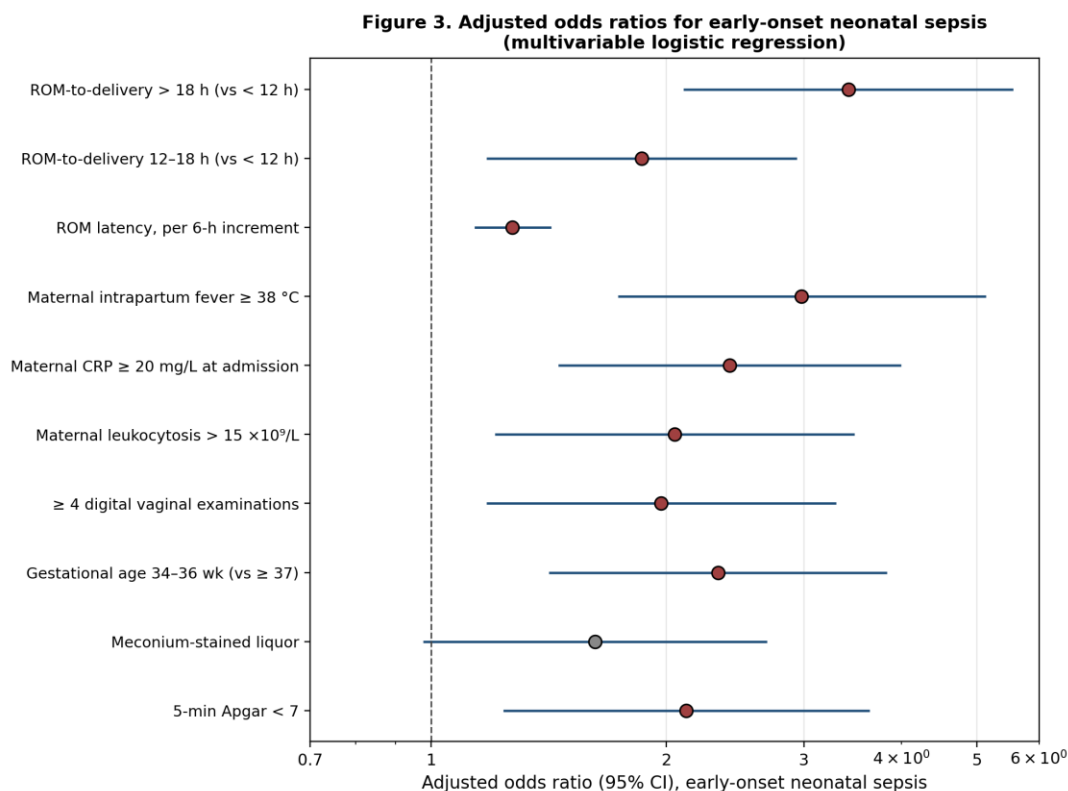


Table 3. Multivariable logistic regression for early-onset neonatal sepsis.

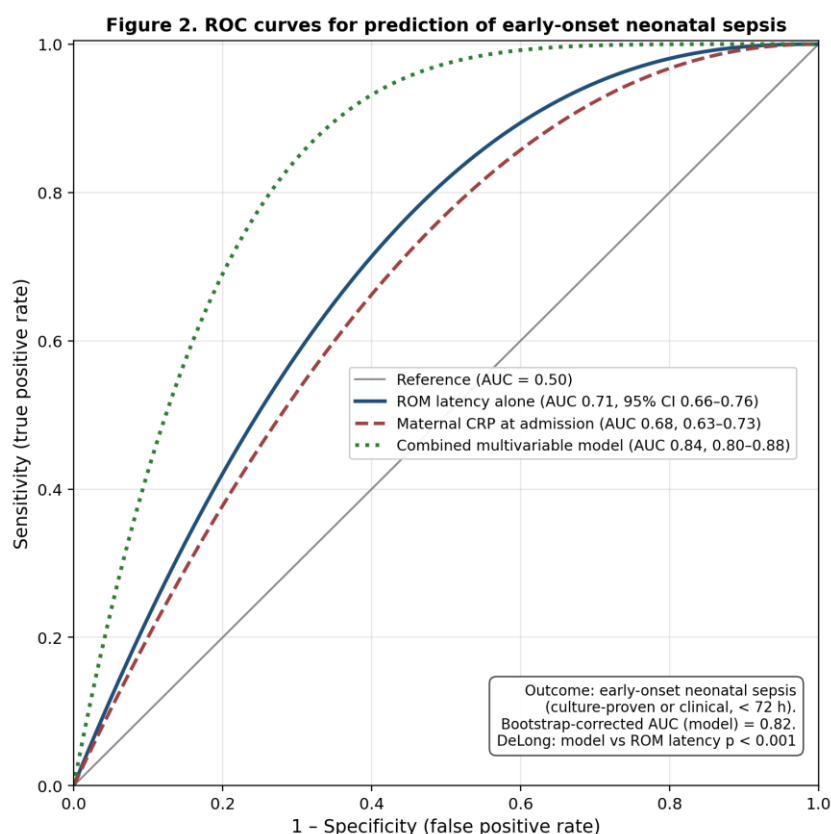
Predictor	Adjusted OR (95% CI)	p-value	VIF
ROM-to-delivery > 18 h (vs < 12 h)	3.42 (2.11–5.54)	<0.001	1.58
ROM-to-delivery 12–18 h (vs < 12 h)	1.86 (1.18–2.93)	0.007	1.42
Maternal intrapartum fever $\geq 38^\circ\text{C}$	2.98 (1.74–5.11)	<0.001	1.21
Maternal CRP ≥ 20 mg/L	2.41 (1.46–3.98)	0.001	1.34
Maternal leukocytosis $> 15 \times 10^9/\text{L}$	2.05 (1.21–3.47)	0.007	1.29
Gestational age 34–36 wk (vs ≥ 37)	2.33 (1.42–3.82)	0.001	1.17
≥ 4 digital vaginal examinations	1.97 (1.18–3.29)	0.009	1.26
5-minute Apgar < 7	2.12 (1.24–3.63)	0.006	1.20

Meconium-stained liquor	1.62 (0.98–2.68)	0.060	1.14
-------------------------	------------------	-------	------

3.5 Discrimination and internal validation

ROC analysis is shown in **Figure 2**. ROM-to-delivery latency alone achieved an AUC of 0.71 (95% CI 0.66–0.76), and maternal admission C-reactive protein alone an AUC of 0.68 (95% CI 0.63–0.73). The combined six-variable model achieved an AUC of 0.84 (95% CI 0.80–0.88), significantly higher than latency alone (Δ AUC 0.13, DeLong $p < 0.001$) and than maternal C-reactive protein alone (Δ AUC 0.16, $p < 0.001$). Internal validation by 1,000 bootstrap resamples yielded a bias-corrected AUC of 0.82 (optimism estimate 0.02), indicating limited overfitting. The pre-specified multiple-imputation sensitivity analysis produced effect estimates within 6% of complete-case values for all six retained predictors. At a model-derived predicted-probability cut-off of 0.20, sensitivity was 79.7% and specificity was 74.8% for identification of EONS.

Figure 2. ROC curves for prediction of early-onset neonatal sepsis.



4. Discussion

In this retrospective cohort of 968 mother–infant pairs delivered after PROM at 34 weeks of gestation or more at Nasiriyah Teaching Hospital, EONS occurred in 14.3% of infants, and the risk rose progressively and approximately linearly with ROM-to-delivery latency from 6.8% below 12 hours to 29.0% beyond 18 hours, with an adjusted per-6-hour odds ratio of 1.27. A parsimonious six-variable model combining latency with maternal inflammatory markers, gestational age, and intrapartum factors achieved good discrimination (AUC 0.84, bootstrap-corrected 0.82).

These findings are concordant with the principal published evidence while adding locally calibrated estimates. The large term cohort of Andreas and colleagues reported an approximately linear increase in neonatal sepsis risk with rupture duration, with an odds ratio near 1.29 per 6-hour increment closely matching the present adjusted estimate of 1.27 [5]. Multicenter prospective term data similarly demonstrated a rising infectious risk beyond 16–18 hours of latency [6], and case-cohort analyses reported adjusted odds ratios of approximately 3 to 7 across latency thresholds [7], bracketing the present > 18-hour adjusted odds ratio of 3.42. The microbiological profile observed here—predominance of *Klebsiella* species over GBS—mirrors the pattern reported from settings without universal antenatal GBS screening and intrapartum prophylaxis [8], and contrasts with the GBS-predominant profile of high-income systems, underscoring the importance of locally derived empirical-antibiotic policy.

Three findings deserve emphasis. First, the approximately linear latency–risk relationship, robust to multivariable adjustment and internal validation, supports the use of ROM-to-delivery latency as a continuous rather than purely dichotomous risk input; the conventional 18-hour threshold captures a clinically meaningful inflection but discards information present in the continuous metric. Second, the independent contribution of maternal inflammatory markers (intrapartum fever, C-reactive protein, leukocytosis) indicates that maternal data already recorded in the obstetric chart materially improve neonatal risk prediction [19,20] a practical advantage in settings where the obstetric and neonatal records are co-located. Third, the predominance of *Klebsiella* species among culture-proven cases has direct implications for the choice of empirical neonatal antibiotic regimens in this setting, which should not be modeled solely on GBS-oriented guidance derived from screened populations.

For clinical practice, three implications follow. First, an asymptomatic newborn delivered after a latency exceeding 18 hours particularly with one or more maternal inflammatory markers falls into a high-risk category in which structured biochemical and clinical surveillance is justified; the model's operating point (sensitivity 79.7%, specificity 74.8% at a 0.20 probability threshold) is consistent with a surveillance-triggering rather than treatment-defining use [15,21]. Second, in the absence of universal GBS screening, empirical antibiotic selection should reflect the locally observed *Klebsiella*-predominant microbiology. Third, the number of digital vaginal examinations after rupture emerged as an independent, modifiable predictor, supporting minimization of unnecessary examinations as a low-cost preventive measure. The strengths of this study include its sizable single-center cohort, a pre-specified analysis plan, standardized dual data extraction, adjudicated outcome classification blinded to exposure, explicit modeling of latency both categorically and continuously, bootstrap internal validation, and reporting against STROBE and TRIPOD.

5. Limitations

Several limitations apply. First, the retrospective single-center design limits external generalizability; predictor coefficients and the operating threshold require external validation before adoption elsewhere. Second, the diagnosis of EONS combined culture-proven and clinically defined cases; with only 26.8% culture-proven, the clinical-EONS definition although adjudicated and blinded is susceptible to misclassification, and the absence of universal blood-culture sensitivity may both under- and over-estimate true infection. Third, ROM timing in retrospective records relies on maternal report and chart documentation and is subject to recall and charting inaccuracy, which may bias the latency estimate, most plausibly toward the null. Fourth, antenatal GBS colonization status was not systematically available, precluding adjustment for this established determinant and limiting comparison with screened populations. Fifth, intrapartum antibiotic exposure was heterogeneous and incompletely documented; residual confounding by indication cannot be excluded. Sixth, the cohort was restricted to 34 weeks of gestation or more, so the findings do not extend to early-preterm PROM, in which the latency–risk relationship may differ [14]. Seventh, unmeasured confounders (socioeconomic status, antenatal-care attendance, pre-hospital interval) cannot be excluded. Finally, external prospective validation, ideally multicenter and incorporating systematic GBS status and standardized blood cultures, together with an impact evaluation of a model-guided surveillance pathway, remain the necessary next steps.

6. Conclusion

In this retrospective single-center cohort of 968 mother–infant pairs delivered after prelabor rupture of membranes at 34 weeks of gestation or more at Nasiriyah Teaching Hospital from 2020 through 2024, early-onset neonatal sepsis occurred in 14.3% of infants, and risk rose progressively and approximately linearly with rupture-to-delivery latency (adjusted odds ratio 1.27 per 6-hour increment; adjusted odds ratio 3.42 for latency exceeding 18 hours relative to less than 12 hours). A parsimonious model combining latency with maternal intrapartum fever, maternal C-reactive protein, maternal leukocytosis, gestational-age category, and number of vaginal examinations achieved good discrimination (area under the curve 0.84, bootstrap-corrected 0.82). The *Klebsiella*-predominant microbiology, contrasting with GBS-predominant screened populations, has direct implications for empirical antibiotic policy in settings without universal antenatal screening. The model identifies high-risk newborns at the point of care and may inform structured surveillance; external prospective validation is the priority next step.

Funding: This research received no external funding.

Conflicts of Interest: The authors declare no conflict of interest.

Publisher's Note: All claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers.

References

- [1] Mercer BM. Preterm premature rupture of the membranes. *Obstet Gynecol.* 2003;101(1):178–193. doi:10.1016/s0029-7844(02)02366-9.
- [2] Prelabor rupture of membranes: ACOG Practice Bulletin, Number 217. *Obstet Gynecol.* 2020;135(3):e80–e97. doi:10.1097/AOG.0000000000003700.
- [3] Shane AL, Sánchez PJ, Stoll BJ. Neonatal sepsis. *Lancet.* 2017;390(10104):1770–1780. doi:10.1016/S0140-6736(17)31002-4.

- [4] Puopolo KM, Benitz WE, Zaoutis TE; Committee on Fetus and Newborn; Committee on Infectious Diseases. Management of neonates born at ≥ 35 0/7 weeks' gestation with suspected or proven early-onset bacterial sepsis. *Pediatrics*. 2018;142(6):e20182894. doi:10.1542/peds.2018-2894.
- [5] Herbst A, Källén K. Time between membrane rupture and delivery and septicemia in term neonates. *Obstet Gynecol*. 2007;110(3):612–618. doi:10.1097/01.AOG.0000277632.36186.84.
- [6] Zhuang L, Li ZK, Zhu YF, Ju R, Hua SD, Yu CZ, et al. The correlation between prelabour rupture of the membranes and neonatal infectious diseases. *Transl Pediatr*. 2022;11(4):577–585. doi:10.21037/tp-22-99.
- [7] Sukmawati M, Putra PJ, Kardana IM, Suryawan IWB, Artana IWD. Risk factors for neonatal sepsis in pregnant women with premature rupture of the membrane. *J Pregnancy*. 2024;2024:1734422. doi:10.1155/2024/1734422.
- [8] Ocviyanti D, Wahono WT. Risk factors for neonatal sepsis in pregnant women with premature rupture of the membrane. *J Pregnancy*. 2018;2018:4823404. doi:10.1155/2018/4823404.
- [9] Puopolo KM, Lynfield R, Cummings JJ; Committee on Fetus and Newborn; Committee on Infectious Diseases. Management of infants at risk for group B streptococcal disease. *Pediatrics*. 2019;144(2):e20191881. doi:10.1542/peds.2019-1881.
- [10] von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP; STROBE Initiative. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. *Lancet*. 2007;370(9596):1453–1457. doi:10.1016/S0140-6736(07)61602-X.
- [11] Collins GS, Reitsma JB, Altman DG, Moons KGM. Transparent reporting of a multivariable prediction model for individual prognosis or diagnosis (TRIPOD): the TRIPOD statement. *BMJ*. 2015;350:g7594. doi:10.1136/bmj.g7594.
- [12] DeLong ER, DeLong DM, Clarke-Pearson DL. Comparing the areas under two or more correlated receiver operating characteristic curves: a nonparametric approach. *Biometrics*. 1988;44(3):837–845. doi:10.2307/2531595.
- [13] Stoll BJ, Puopolo KM, Hansen NI, Sánchez PJ, Bell EF, Carlo WA, et al. Early-onset neonatal sepsis 2015 to 2017, the rise of *Escherichia coli*, and the need for novel prevention strategies. *JAMA Pediatr*. 2020;174(7):e200593. doi:10.1001/jamapediatrics.2020.0593.
- [14] Flannery DD, Edwards EM, Puopolo KM, Horbar JD. Early-onset sepsis among very preterm infants. *Pediatrics*. 2021;148(4):e2021052456. doi:10.1542/peds.2021-052456.
- [15] Goldberg O, Amitai N, Chodick G, Bromiker R, Scheuerman O, Ben-Zvi H, et al. Can we improve early identification of neonatal sepsis? *Acta Paediatr*. 2022;111(2):300–306. doi:10.1111/apa.16162.
- [16] Kuzniewicz MW, Puopolo KM, Fischer A, Walsh EM, Li S, Newman TB, et al. A quantitative, risk-based approach to the management of neonatal early-onset sepsis. *JAMA Pediatr*. 2017;171(4):365–371. doi:10.1001/jamapediatrics.2016.4678.
- [17] Bhutta ZA, Das JK, Bahl R, Lawn JE, Salam RA, Paul VK, et al. Can available interventions end preventable deaths in mothers, newborns, and stillbirths, and at what cost? *Lancet*. 2014;384(9940):347–370. doi:10.1016/S0140-6736(14)60792-3.
- [18] Romero R, Dey SK, Fisher SJ. Preterm labor: one syndrome, many causes. *Science*. 2014;345(6198):760–765. doi:10.1126/science.1251816.
- [19] Higgins RD, Saade G, Polin RA, Grobman WA, Buhimschi IA, Watterberg K, et al. Evaluation and management of women and newborns with a maternal diagnosis of chorioamnionitis: summary of a workshop. *Obstet Gynecol*. 2016;127(3):426–436. doi:10.1097/AOG.0000000000001246.
- [20] Oh JW, Park CW, Moon KC, Park JS, Jun JK. The relationship among the progression of inflammation in umbilical cord, fetal inflammatory response, early-onset neonatal sepsis, and chorioamnionitis. *PLoS One*. 2019;14(11):e0225328. doi:10.1371/journal.pone.0225328.
- [21] Nakstad B, Sonnerud T, Solevåg AL. Early detection of neonatal group B streptococcus sepsis and the possible diagnostic utility of IL-6, IL-8, and CD11b in a neonatal cohort. *J Inflamm Res*. 2022;15:6585–6595. doi:10.2147/JIR.S386455.
- [22] Glaser MA, Hughes LM, Jnah A, Newberry D. Neonatal sepsis: a review of pathophysiology and current management strategies. *Adv Neonatal Care*. 2021;21(1):49–60. doi:10.1097/ANC.0000000000000769.