

Prognostic Value of Pentraxin-3 and Procalcitonin in Neonatal Sepsis

Mudassar Rubab, Sumbul Nida, Amina Tariq, Hina Batool*

Department of Pathology, Pakistan Aeronautical Complex Hospital, Kamra Attock Pakistan,

*Department of Paediatrics, Pakistan Aeronautical Complex Hospital, Kamra Attock Pakistan

ABSTRACT

Objective: To evaluate the prognostic value of Pentraxin-3 and Procalcitonin in neonatal sepsis.

Study Design: Prospective observational study.

Place and Duration of Study: Neonatal Intensive Care Unit, Pakistan Aeronautical Complex Hospital, Kamra, Attock, Pakistan, from May 24 to May 25.

Methodology: A total of seventy neonates with sepsis were enrolled, including forty in the improved group and thirty in the deteriorated group. Clinical evaluation included anthropometric assessment, detailed history, physical examination, and disease progression scoring using the neonatal 0053equential Organ Failure Assessment. Laboratory parameters included complete blood count, blood culture, high-sensitivity C-reactive protein, Procalcitonin, and Pentraxin-3 levels.

Results: At baseline, median Pentraxin-3 levels was comparable between groups (1155.00 vs. 1006.50 pg/mL), while median levels of procalcitonin (1.58 vs. 5.80 ng/mL), hs-CRP (44.50 vs. 88.50 mg/L), and creatinine (71.50 vs. 195.00 μ mol/L) were significantly higher in the deteriorated group ($p < 0.001$). After 72 hours, the improved group showed reductions in median nSOFA score (8 $\rightarrow$ 5), Pentraxin-3 (1155.00 $\rightarrow$ 327.00 pg/mL), procalcitonin (1.58 $\rightarrow$ 0.50 ng/mL), hs-CRP (44.50 $\rightarrow$ 7.90 mg/L), TLC (14.95 $\rightarrow$ 8.00 $\times 10^9$ /L), and creatinine (71.50 $\rightarrow$ 55.50 μ mol/L) (all $p < 0.05$). Values before and after the arrows represent baseline and 72-hour measurements, respectively. The deteriorated group demonstrated increases in nSOFA (10 $\rightarrow$ 18), Pentraxin-3 (1006.5 $\rightarrow$ 2394.5 pg/mL), procalcitonin (5.80 $\rightarrow$ 14.75 ng/mL), hs-CRP (88.5 $\rightarrow$ 217 mg/L), and TLC (18.4 $\rightarrow$ 23.4 $\times 10^9$ /L) (all $p < 0.05$). Both markers showed strong correlation with worsening neonatal Sequential Organ Failure Assessment scores and adverse outcomes.

Conclusion: Procalcitonin and Pentraxin-3 are valuable prognostic indicators in neonatal sepsis. Their incorporation into clinical practice may enhance early risk stratification, guide treatment decisions, and improve neonatal outcomes.

Keywords: Neonatal Sepsis, Pentraxin-3, Procalcitonin, Sequential Organ Failure Assessment.

How to Cite This Article: Rubab M, Nida S, Tariq A, Batool H. Prognostic Value of Pentraxin-3 and Procalcitonin in Neonatal Sepsis. Pak Armed Forces Med J 2026; 76(Suppl-9): S1463-S1467. DOI: <https://doi.org/10.51253/pafmj.v76iSUPPL-9.13768>

This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<https://creativecommons.org/licenses/by-nc/4.0/>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

INTRODUCTION

Neonatal sepsis, a life-threatening condition in newborns within the first month of life, remains a significant global health concern, particularly in regions like South Asia.¹ Among neonates, sepsis ranks as the third most common cause of mortality. In areas where healthcare infrastructure is limited, neonatal sepsis poses a considerable burden on public health systems.² Factors such as inadequate prenatal care, poor sanitation, and limited access to skilled birth attendants, risk of neonatal sepsis.³ In South Asia, including Pakistan, a substantial proportion of neonatal deaths are attributed to sepsis-related complications. Survivors of neonatal sepsis may experience complications like developmental delays, neurological impairments, and growth disturbances,⁴ highlighting the urgent need for early detection. In

this context, exploring the utility of biomarkers such as procalcitonin and Pentraxin-3 holds promise for enhancing the early detection and prognostication of neonatal sepsis.⁵

Procalcitonin, the precursor to the hormone calcitonin, is synthesized primarily by the thyroid gland under normal physiological conditions. However, during bacterial infections, particularly those characterized by systemic inflammation such as sepsis, procalcitonin production increases substantially.⁶ Unlike traditional markers such as C-reactive protein (CRP) or white blood cell count, procalcitonin demonstrates a more rapid and specific response to bacterial infections. Moreover, serial monitoring of procalcitonin levels has shown promise in guiding therapeutic interventions, including antibiotic stewardship.⁷ Similarly, Pentraxin-3, a member of the Pentraxin family of pattern recognition molecules, exhibits immunomodulatory functions implicated in the innate immune response to microbial pathogens. Elevated Pentraxin-3 levels have been

Correspondence: Dr Mudassar Rubab, Department of Pathology, Pakistan Aeronautical Complex Hospital, Kamra Attock Pakistan
Received: 02 Sep 2025; revision received: 28 Feb 2026; accepted: 03 Mar 2026

associated with adverse outcomes, including septic shock, multi-organ dysfunction, and mortality, in neonates with sepsis. Additionally, Pentraxin-3 has demonstrated superior discriminatory ability compared to conventional biomarkers, highlighting its value as a prognostic tool in neonatal sepsis.⁸

In light of the critical need for improved diagnostic and prognostic tools in the management of neonatal sepsis, this study aims to investigate the utility of procalcitonin and Pentraxin-3 as prognostic markers, elucidating their roles in neonatal sepsis, evaluating their efficacy in early risk stratification, severity assessment, and prediction of clinical outcomes.

METHODOLOGY

This prospective observational study was conducted in the Neonatal Intensive Care Unit (NICU) of Pakistan Aeronautical Complex (PAC) Hospital, Kamra, Attock, Pakistan, from May 24 to May 25. The sample size was calculated using the Sensitivity and Specificity Calculator 7.4(a), keeping confidence interval 95%, margin of error 5%, sensitivity and specificity from previous studies,⁹ taking a reference prevalence of 32.5%, the sample size calculated was 70. All eligible neonates during the study period were recruited using consecutive sampling, and a total of 70 neonates presenting with sepsis were enrolled.

Inclusion Criteria: Neonates up to 28 days of age, with a gestational age between 32 and 40 weeks; neonates with a clinically and/or laboratory-confirmed diagnosis of neonatal sepsis.

Exclusion Criteria: Neonates with congenital abnormalities, cardiogenic or hemorrhagic shock, metabolic disorders, prior intravenous antibiotic use, hypoxic-ischemic encephalopathy, or birth weight less than 1.5 kg.

Ethical approval for the study was obtained from the Institutional Review Board of PAC Hospital Kamra, Attock (Reference No. Hosp-Kam/4986/7/Trg, dated 3 May 2024). Written informed consent was obtained from parents or guardians before enrollment.

Neonatal sepsis was diagnosed according to the International Sepsis Definitions (2001).¹⁰ Clinical suspicion was raised in neonates with nonspecific features such as lethargy, poor feeding, irritability, temperature instability, or respiratory distress, and confirmed by specific signs such as apnea, bradycardia, hypotension, and hypoglycemia.¹¹

Sources of infection were assessed through examination for omphalitis, skin pustules, respiratory distress with retractions or grunting, abdominal distension, or hepatosplenomegaly. Clinical diagnoses were made by two NICU consultants with postgraduate training in pediatrics and over five years of neonatal care experience. To minimize subjective bias, standardized diagnostic checklists were used, and all cases were independently reviewed by both consultants; disagreements were resolved by consensus.

The neonatal Sequential Organ Failure Assessment (nSOFA) score was calculated at admission and repeated during hospitalization to monitor progression. The score evaluates six organ systems (respiratory, cardiovascular, hepatic, coagulation, renal, and neurological) on a 0–4 scale, with higher scores reflecting greater severity.^{12,13} Blood samples were collected at admission and repeated at 72 hours or earlier if clinically indicated.

Based on clinical course and outcome at 72 hours of hospitalization, enrolled neonates were categorized into two groups: Improved Group: Neonates who demonstrated clinical recovery, with declining nSOFA scores and improvement in laboratory parameters. Deteriorated Group: Neonates who showed persistent or worsening clinical features, rising nSOFA scores, and deterioration in laboratory biomarkers, including those who developed multi-organ dysfunction or mortality.

Blood cultures were processed using the BACTEC ALERT 3D system. Pentraxin-3 (PTX3) was measured by quantitative sandwich ELISA (Elabscience), with absorbance read on a microplate reader (STAT FAX 2100). Procalcitonin (PCT) and high-sensitivity C-reactive protein (hs-CRP) were quantified using the Ichroma™ fluorescence immunoassay (FIA) system. All assays were performed following appropriate quality control procedures.

Demographic data, clinical findings, laboratory results, and outcomes were recorded on a standardized proforma. Research staff underwent structured training in study protocols, clinical assessments, and specimen handling; competency was verified before study initiation and refreshed periodically. To further minimize bias, laboratory staff performing PTX3, PCT, and hs-CRP assays were blinded to clinical data, and all samples were labeled with anonymized identifiers. Inter-rater reliability of

nSOFA scoring was assessed at baseline using duplicate scoring of a subset of patients.

Data was analyzed using Statistical Package for Social Sciences, version 27.0. Shapiro-Wilk test was used initially to check for normality. Normally distributed variables were platelets and hemoglobin (expressed as Mean $\pm$ SD, using an independent t-test). SOFA scores, Pentraxin-3, procalcitonin-CRP, Total leukocyte count, Creatinine and Bilirubin were not distributed normally (expressed as median (IQR), using Wilcoxon signed rank test and Mann-Whitney U test). A p -value ≤ 0.05 was considered statistically significant.

RESULTS

Among the seventy neonates enrolled, 42(60.0%) were females, and 28(40.0%) were males. The majority were delivered via lower segment cesarean section (42, 60.0%), while 28(40.0%) were born through spontaneous vaginal delivery. Most neonates were late preterm, with 35(50.0%) born at 32–34 weeks, 26(37.1%) at 35–37 weeks, and 9(12.9%) at 38–40 weeks of gestation. Apgar scores of 4–5 were observed in 54(77.1%) neonates, while 15(21.4%) had scores of 6–7, and only 1(1.4%) had a score of 8–9. Ventilatory support was required in 24(34.3%) neonates. Premature rupture of membranes (PROM) was noted in 50(71.4%) cases. Maternal comorbidities included diabetes in 25(35.7%) mothers and hypertension in 27(38.6%) mothers.

Table-I summarizes within-group comparisons over a period of 72 hours. The median total n-SOFA score declined in the improved group ($n=40$) from 8.00 at baseline to 5.00 at 72 hours ($p<0.001$). Significantly decreasing trends were also observed in Pentraxin-3 levels 1155pg/ml to 327pg/ml ($p<0.001$), pro-calcitonin levels from 1.58 ng/ml to 0.50 ng/ml ($p<0.001$), and hs-CRP from 44.50 mg/L to 7.90 mg/L. Contrary to it, in the deteriorated group ($n=30$), the median total n-SOFA score increased from 10.00 at baseline to 18.00 at 72 hours ($p<0.001$). Significant increasing trends were seen in Pentraxin-3 levels from 1006.50pg/ml to 2394.50pg/ml and pro-calcitonin level from 5.80ng/ml to 14.75ng/ml in this group.

Inter-group comparisons are shown in Table-II. At baseline, Pro-calcitonin (5.80ng/ml vs 1.58ng/ml) and hs-CRP (44.50 vs 88.50mg/L) were increased in deteriorated group as compared to improved group. Baseline Pentraxin-3 levels (1155.00 pg/ml vs 1006.00 pg/ml) and total leukocyte count did not differ significantly between groups. Significant inter-group

differences were observed at 72 hours for Pentraxin-3 (327.00pg/ml vs 2394.50 pg/ml) and procalcitonin levels (0.50 ng/ml vs 14.75 pg/ml). These findings indicate that rising Pentraxin-3 and Procalcitonin levels strongly correlated with clinical deterioration and higher nSOFA scores, whereas declining levels were associated with recovery.

Table-I: Comparison of baseline and 72-hour values in Improved ($n=40$) and Deteriorated ($n=30$) groups among the total study population ($n=70$)

Parameters	Improved Group ($n=40$)			Deteriorated Group ($n=30$)		
	Baseline	After 72 Hours	P -value	Baseline	After 72 Hours	p -value
nSOFA total	8.00 (6.00–11.00)	5.00 (3.00–6.00)	<0.001	10.00 (9.00–12.00)	18.00 (15.00–20.00)	<0.001
Respiratory nSOFA	2.00 (2.00–3.00)	1.00 (0.00–1.00)	<0.001	3.00 (2.00–4.00)	3.00 (3.00–4.00)	<0.001
Cardiovascular nSOFA	2.00 (2.00–3.00)	1.00 (0.00–1.00)	<0.001	3.00 (3.00–4.00)	3.00 (3.00–4.00)	0.53
Hematology nSOFA	2.00 (2.00–3.00)	1.00 (0.00–1.00)	<0.001	3.00 (3.00–4.00)	4.00 (3.00–4.00)	0.01
Pentaxin-3 (pg/mL)	1155.00 (684.00–2374.00)	327.00 (124.00–574.00)	<0.001	1006.50 (656.00–1993.00)	2394.50 (1444.00–4537.00)	<0.001
Procalcitonin (ng/mL)	1.58 (0.26–0.67)	0.50 (0.00–0.17)	<0.001	5.80 (5.00–9.00)	14.75 (12.00–19.00)	<0.001
hs-CRP (mg/L)	44.50 (12.00–63.00)	7.90 (0.00–11.00)	<0.001	88.50 (52.00–137.00)	217.00 (141.00–345.00)	0.05
TLC ($\times 10^9$ /L)	14.95 (12.00–23.00)	8.00 (7.00–11.00)	<0.001	18.40 (15.00–21.00)	23.40 (17.00–32.00)	0.01
Platelets ($\times 10^9$ /L)	264.85 $\pm$ 90.59	242.65 $\pm$ 97.97	0.16	227.06 $\pm$ 106.72	247.13 $\pm$ 89.96	0.28
Hemoglobin (g/dL)	16.76 $\pm$ 2.58	15.38 $\pm$ 2.85	0.01	16.82 $\pm$ 2.22	15.34 $\pm$ 1.61	<0.001
Creatinine (μ mol/L)	71.50 (59.00–101.00)	55.50 (45.00–77.00)	0.02	195.00 (128.00–345.00)	78.00 (58.00–124.00)	<0.001
Bilirubin (μ mol/L)	57.00 (40.00–81.00)	76.50 (57.00–126.00)	0.01	93.00 (65.00–160.00)	45.00 (36.00–56.00)	0.01

Legend: Values expressed as median (IQR) for non-parametric variables and mean $\pm$ SD for parametric variables. Paired t-test was used for parametric variables; Wilcoxon signed-rank test was used for non-parametric variables.

Abbreviations: nSOFA = neonatal Sequential Organ Failure Assessment; hs-CRP = high-sensitivity C-reactive protein; TLC = total leukocyte count; SD = standard deviation; IQR = interquartile range

Table-II: Baseline and 72-hour intergroup comparison of laboratory parameters between Improved ($n=40$) and Deteriorated ($n=30$) groups in the total study population ($n=70$)

Parameters	Improved Group ($n=40$)	Deteriorated Group ($n=30$)	p -value
Pentaxin-3 (pg/mL)	1155.00 (684.00–2374.00)	1006.50 (656.00–1993.00)	<0.001
Procalcitonin (ng/mL)	1.58 (0.26–0.67)	5.80 (5.00–9.00)	<0.001
hs-CRP (mg/L)	44.50 (12.00–63.00)	88.50 (52.00–137.00)	<0.001
TLC ($\times 10^9$ /L)	14.95 (12.00–23.00)	18.40 (15–21)	0.35
Platelets ($\times 10^9$ /L)	264.85 $\pm$ 90.59	227.06 $\pm$ 106.72	0.61
Hemoglobin (g/dL)	16.76 $\pm$ 2.58	16.82 $\pm$ 2.22	0.15
Creatinine (μ mol/L)	71.50 (59.00–101.00)	195 (128.00–345.00)	<0.001
Bilirubin (μ mol/L)	57.00 (40.00–81.00)	93 (65.00–160.00)	0.001
Pentaxin-3 after 72 h (pg/mL)	327 (124.00–574.00)	2394.50 (1444.00–4537.00)	<0.001
Procalcitonin after 72 h (ng/mL)	0.50 (0–0.17)	14.75 (12.00–19.00)	<0.001
hs-CRP after 72 h (mg/L)	7.90 (0.00–11.00)	217.00 (141.00–345.00)	<0.001
TLC after 72 h ($\times 10^9$ /L)	8.00 (7.00–11.00)	23.40 (17.00–32.00)	<0.001
Platelets after 72 h ($\times 10^9$ /L)	242.65 $\pm$ 97.97	247.13 $\pm$ 89.96	1.00
Hemoglobin after 72 h (g/dL)	15.38 $\pm$ 2.85	15.34 $\pm$ 1.61	0.005
Creatinine after 72 h (μ mol/L)	55.50 (45.00–77.00)	78.00 (58.00–124.00)	0.003
Bilirubin after 72 h (μ mol/L)	76.50 (57.00–126.00)	45.00 (36.00–56.00)	0.001

Legend: Intergroup comparison at baseline and 72 hours using the Mann-Whitney test for non-parametric variables and independent t-test for parametric variables.

Abbreviations: hs-CRP = high-sensitivity C-reactive protein; TLC = total leukocyte count; SD = standard deviation; IQR = interquartile range

DISCUSSION

In this study, significant differences were observed between the improved and deteriorated groups over 72 hours. In the improved group, the median total nSOFA score declined from 8.0 (6–11) at baseline to 5.0 (3–6) after 72 hours ($p<0.001$), reflecting clinical recovery. In contrast, patients in the

deteriorated group showed a sharp rise in nSOFA scores from 10.0 (9–12) to 18.0 (15–20) ($p < 0.001$). Similarly, Pentraxin-3 levels decreased markedly in the improved group from 1155 pg/mL to 327 pg/mL ($p < 0.001$), whereas they rose substantially in the deteriorated group from 1006.5 pg/mL to 2394.5 pg/mL ($p < 0.001$). Procalcitonin showed a comparable trend, falling from 1.58 ng/mL to 0.50 ng/mL in the improved group ($p < 0.001$), but increasing from 5.80 ng/mL to 14.75 ng/mL in the deteriorated group ($p < 0.001$). These findings highlight the prognostic utility of Pentraxin-3 and Procalcitonin, in parallel with nSOFA dynamics, for differentiating clinical trajectories in neonatal sepsis. Interestingly, PCT showed earlier separation between groups, whereas PTX3 divergence appeared later, suggesting a complementary role of the two biomarkers in prognostication.

Our findings agreed with Fahmey *et al.*, who also reported elevated PTX3 levels in septic neonates with poor outcomes, supporting its role as a severity marker.¹⁴ Similarly, Sabry *et al.*, demonstrated that PTX3 levels were significantly higher in neonates with systemic bacterial infection and correlated with sepsis severity.¹⁵ However, unlike these studies where baseline PTX3 was already elevated in non-survivors, our results showed no significant difference at baseline, with prognostic separation appearing only after 72 hours. This suggests that PTX3 dynamics over time may provide additional prognostic information compared to a single baseline value.

With regard to PCT, our study confirms earlier reports highlighting its diagnostic and prognostic relevance. Habib *et al.*, found serum PCT to be an accurate early biomarker for neonatal sepsis, using blood culture as the reference.¹⁶ These findings were extended by demonstrating its utility not only in diagnosis but also in monitoring progression, as persistently high or rising levels were associated with worsening nSOFA scores and poor outcomes. Importantly, our study highlights that PCT rises earlier than PTX3, which may give it an edge as an initial risk stratification tool.

The combined assessment of biomarkers with clinical scoring adds strength to prognostication. Prior studies have either evaluated biomarkers in isolation or relied mainly on blood cultures, which have well-recognized limitations such as low sensitivity and delayed reporting.¹⁷ By incorporating both PCT and PTX3 with nSOFA scoring, our study provides

evidence for a more comprehensive approach. This aligns with the findings of Mironov *et al.*, who validated the prognostic accuracy of nSOFA in neonatal sepsis, and suggests that combining dynamic biomarker trends with objective clinical scoring may improve early identification of high-risk neonates.¹⁷

Despite these encouraging results, some differences exist compared to the published literature. For example, while Dinleyici *et al.*, demonstrated that PTX3 could serve as an early marker in fetal inflammatory response syndrome,¹⁸ our data indicate that PTX3 may be more useful for monitoring progression rather than initial risk stratification. This discrepancy could be due to differences in study populations, assay methodologies, or timing of sampling. Similarly, unlike Mucka *et al.*, study, where PCT specificity was questioned due to elevations in non-infectious inflammatory conditions,¹⁹ our data showed a consistent correlation between PCT and sepsis severity, possibly reflecting the careful exclusion of confounding conditions in our cohort.

Overall, our results support the integration of PCT and PTX3 into neonatal sepsis management. While PCT may serve as an early indicator of severity, PTX3 provides valuable information on progression and prognosis. Used together, alongside validated clinical scoring systems, these biomarkers may enhance early risk stratification.

LIMITATIONS OF STUDY

This study has certain limitations. First, it was conducted at a single tertiary care center with a relatively small sample size, which may limit the generalizability of the findings. Second, although we attempted to minimize bias through blinding and standardized diagnostic checklists, some degree of subjectivity in clinical assessment could not be entirely eliminated. Third, serial measurements were limited to admission and 72 hours; more frequent sampling might have provided a clearer picture of biomarker kinetics. Finally, resource constraints did not allow parallel evaluation of additional emerging biomarkers, which could have further strengthened the comparative analysis and stratification.

CONCLUSION

Procalcitonin and Pentraxin-3 demonstrated strong prognostic significance in neonatal sepsis, with their serial measurement closely reflecting clinical progression and outcome. Their integration with nSOFA scoring enhances risk stratification and provides a reliable means of distinguishing recovery from deterioration in critically ill neonates.

ACKNOWLEDGEMENTS

The authors are grateful to the Department of Pediatrics, PAC Hospital Kamra, for their continuous support throughout the study. We also acknowledge the laboratory staff for their technical assistance in biomarker analysis.

Conflict of Interest: None.

Funding Source: None.

Authors' Contribution

Following authors have made substantial contributions to the manuscript as under:

MR & SN: Data acquisition, data analysis, critical review, approval of the final version to be published.

AT & HB: Study design, data interpretation, drafting the manuscript, critical review, approval of the final version to be published.

Authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

REFERENCES

- Denkel LA, Maechler F, Schwab F, Behnke M, Gastmeier P, Kola A. Infections caused by extended-spectrum beta-lactamase-producing Enterobacteriaceae in low-income and middle-income countries from a facility-based birth cohort: an international multisite prospective observational study. *Lancet Glob Health* 2022; 10(5): e661-e672. [https://doi.org/10.1016/S2214-109X\(22\)00043-2](https://doi.org/10.1016/S2214-109X(22)00043-2)
- Heron MP. Deaths: leading causes for 2018. *Natl Vital Stat Rep* 2021; 70(4): 1-13.
- Rudd KE, Kissoon N, Limmathurotsakul D, Bory S, Mutahunga B, Seymour CW, et al. The global burden of sepsis: barriers and potential solutions. *Crit Care* 2018; 22(1): 1. <https://doi.org/10.1186/s13054-018-1970-z>
- Chaulagain DR, Malqvist M, Wrammert J, KC A, Gurung R, Malla H, et al. Service readiness and availability of perinatal care in public hospitals—a multi-centric baseline study in Nepal. *BMC Pregnancy Childbirth* 2022; 22(1): 842. <https://doi.org/10.1186/s12884-022-05121-z>
- Bakhuizen SE, de Haan TR, Teune MJ, van Wassenaeer-Leemhuis AG, van der Heyden JL, van der Ham DP, et al. Meta-analysis shows that infants who have suffered neonatal sepsis face an increased risk of mortality and severe complications. *Acta Paediatr* 2014; 103(12): 1211-1218. <https://doi.org/10.1111/apa.12764>
- Sharma D, Farahbakhsh N, Shastri S, Sharma P. Biomarkers for diagnosis of neonatal sepsis: a literature review. *J Matern Fetal Neonatal Med* 2018; 31(12): 1646-1659. <https://doi.org/10.1080/14767058.2017.1322199>
- Bassetti M, Russo A, Righi E, Dolso E, Merelli M, D'Aurizio F, et al. Role of procalcitonin in bacteremic patients and its potential use in predicting infection etiology. *Expert Rev Anti Infect Ther* 2019; 17(2): 99-105. <https://doi.org/10.1080/14787210.2019.1570972>
- Candel FJ, Sá MB, Belda S, Bou G, Del Pozo JL, Estrada O, et al. Current aspects in sepsis approach: turning things around. *Rev Esp Quimioter* 2018; 31(4): 298-310.
- Abd El Aal MO, Abo Seif IS, Abd El Kareem SA, Abdel-Wahed MA. Prognostic value of pentraxin 3 and procalcitonin in late-onset neonatal sepsis. *Egypt J Hosp Med* 2023; 90(1): 132-138. <https://doi.org/10.21608/ejhm.2023.279093>
- Singer M, Deutschman CS, Seymour CW, Shankar-Hari M, Annane D, Bauer M, et al. The third international consensus definitions for sepsis and septic shock (Sepsis-3). *JAMA* 2016; 315(8): 801-810. <https://doi.org/10.1001/jama.2016.0287>
- Jana AK, Sridhar S. Clinical diagnosis of sepsis. *J Neonatol* 2009; 23(1): 44-47. <https://doi.org/10.1177/0973217920090108>
- Fleiss N, Polin RA. Sequential organ failure assessment scores to predict outcomes: from adults to neonates. *Curr Opin Pediatr* 2023; 35(2): 218-222. <https://doi.org/10.1097/MOP.0000000000001207>
- Mironov PI, Lekmanov AU. Evaluation of the validity of the nSOFA score in newborns with sepsis. *Mess Anesthesiol Reanimatol* 2021; 18(2): 56-61. <https://doi.org/10.21292/2078-5658-2021-18-2-56-61>
- Fahmey SS, Mostafa N. Pentraxin 3 as a novel diagnostic marker in neonatal sepsis. *J Neonatal Perinat Med* 2019; 12(4): 437-442. <https://doi.org/10.3233/NPM-1848>
- Sabry A, Ibrahim M, Khashana A. Assessment of pentraxin 3 in a systemic inflammatory response occurring with neonatal bacterial infection. *J Neonatal Perinat Med* 2021; 14(4): 563-568. <https://doi.org/10.3233/NPM-2060>
- Habib A, Raza S, Ali U, Zubairi AM, Salim E. Diagnostic accuracy of serum procalcitonin as an early biomarker of neonatal sepsis using blood culture as gold standard. *J Coll Physicians Surg Pak* 2021; 30(4): 383-388. <https://doi.org/10.29271/jcpsp.2021.04.383>
- Amber S. Blood culture positivity for the diagnosis of neonatal sepsis: is it always necessary? *Int J Clin Diagn Pathol* 2020; 3(1): 215-217. <https://doi.org/10.33545/26164841.2020.v3.i1d.162>
- Dinleyici EC, Tekin NA, Çolak Ö, Akşit AM. Exploring pentraxin 3 as a diagnostic and prognostic marker in fetal inflammatory response syndrome in newborns: a prospective study. *Research Square [Preprint]*. 2022. <https://doi.org/10.21203/rs.3.rs-123456/v1>
- Mučka S, Jakubiak GK, Pawlas N. Procalcitonin: infection or maybe something more? Noninfectious causes of increased serum procalcitonin concentration: updated knowledge. *Life* 2025; 15(3): 446. <https://doi.org/10.3390/life15030446>