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Original Research Article

Association of Early Inflammatory Biomarker Levels with Seven-Day Mortality in Patients with Sepsis: A Retrospective Study

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Abstract:

Background: Sepsis is a life-threatening syndrome with high early mortality. Early inflammatory biomarkers may facilitate rapid risk stratification and improve clinical outcomes.

Aim: To evaluate the association between early inflammatory biomarker levels and seven-day mortality in patients with sepsis.

Methodology: This hospital-based retrospective observational study included 90 adult patients with sepsis diagnosed according to Sepsis-3 criteria. Early inflammatory biomarkers measured within 24 hours of admission, including WBC, CRP, procalcitonin, ESR, serum ferritin, and fibrinogen, were analyzed. Binary logistic regression and receiver operating characteristic (ROC) analyses were performed using SPSS version 27.0.

Results: Seven-day mortality was 28.9%. Non-survivors had significantly higher levels of all inflammatory biomarkers than survivors ($p<0.001$). Advanced age (>60 years), elevated WBC (AOR=1.15), CRP (AOR=1.03), procalcitonin (AOR=1.27), and serum ferritin (AOR=1.002) independently predicted mortality. Procalcitonin demonstrated the highest predictive accuracy (AUC=0.903; sensitivity 88.5%; specificity 84.4%), followed by CRP (AUC=0.846).

Conclusion: Elevated early inflammatory biomarkers, particularly procalcitonin and CRP, are strongly associated with seven-day mortality in sepsis and provide valuable prognostic information for early risk stratification and timely clinical management.

Keywords: Sepsis; Inflammatory Biomarkers; Procalcitonin; C-Reactive Protein; Seven-Day Mortality; Prognosis; Retrospective Study.

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Introduction

Sepsis is a life-threatening condition that is a dysregulated host response to an infectious trigger, resulting in multiple organ dysfunction and high mortality rates [1]. It is estimated that about 11 million people die of it every year around the world. Although critical care and antimicrobial treatment have improved, Sepsis is still a serious worldwide health problem due to its fast progression, the different clinical signs and symptoms and the high cost to healthcare services. Prompt recognition and intervention is critical to enhance the outcomes of patients and death rates.

Sepsis was defined clinically quite differently over the last 30 years, with improvements in our understanding of the pathophysiology of sepsis [2]. Sepsis was first defined in 1991 by the American College of Chest Physicians and the Society of Critical Care Medicine as a systemic inflammatory response syndrome (SIRS) from infection. The definition was modified in 2001 (Sepsis-2) which maintained the SIRS definition and added the clinical manifestations [3]. But the sensitivity and specificity were not great enough, so in 2016, the Sepsis-3 definition was created, which defines sepsis as organ dysfunction secondary to an uncontrolled host response to infection, which is life-threatening. The Sequential Organ Failure Assessment (SOFA) score is used to identify organ dysfunction, with an increase of 2–3 points. While Sepsis-3 is more reliable as a prognosis, diagnosis can still be difficult, especially for older adults and those with compromised immune systems. Emerging diagnostic strategies will include the use of dynamic biomarkers, molecular signatures, and artificial intelligence-based models for better

identification of biologically distinct sepsis subgroups and for personalized therapeutic management.

While anyone can develop sepsis, elderly, infants, pregnant women and immunocompromised people are at a higher risk [4]. It may be difficult to diagnose early given its non-specific clinical presentation. The most frequently seen symptoms consist of fever, rapid heart rate, low blood pressure, rapid breathing, and confusion or coma, which can quickly escalate to septic shock, multiple organ dysfunction syndrome, and death if undiagnosed in a timely fashion. Sepsis is the most common cause of respiratory tract infections followed by UTI, abdominal, hepatobiliary, and soft tissue infections. Sepsis is mostly caused by bacteria, and prompt treatment with the correct antibiotics can be crucial to survival. In addition, the growing burden of healthcare associated infection and multidrug resistant organisms (MDRO) has made the management of sepsis more difficult and resulted in increased healthcare expenditure and death.

The correct estimation of the severity and prognosis of the disease is also a key to good sepsis management, along with early antimicrobial therapy. Thus, inflammatory biomarkers have become the focus of much interest for their use in the diagnosis, monitoring and prognosis of septic patients. Some of the most commonly used biomarkers include C-reactive protein (CRP), procalcitonin (PCT) and fibrinogen, all of which measure various components of the inflammatory response [5]. CRP is one of the acute phase proteins which is synthesised by the liver when there is a systemic inflammatory response, in this case to inflammatory cytokines like interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α). Procalcitonin is a precursor of calcitonin which is produced by extrathyroidal tissues following bacterial infections by action of inflammatory mediators and bacterial endotoxins and is more specific for bacterial sepsis. Another liver-derived acute-phase protein, fibrinogen, is also associated with inflammation and also with the various coagulation abnormalities found in septic patients. Additionally, the neutrophil-to-lymphocyte ratio (NLR), as a simple, inexpensive and effective prognostic marker, has been shown to be associated with severity and mortality in sepsis in recent meta-analyses [6].

These biomarkers change over time, offering insights into disease progression and response to treatment. In general, CRP will increase in 6-8 hours following inflammation and continue to rise until 48 hours after onset of inflammation, while PCT will rise in 2-4 hours after onset of inflammation and peak in the first 24 hours. Lowering of CRP and PCT in the first 48–72 hours of treatment are correlated with good clinical results and may indicate that the patient is not responding to treatment, has an ongoing infection, or is developing septic shock and multiple-organ dysfunction. Thus, routine use of inflammatory markers in clinical practice will help to improve the risk stratification, antibiotic stewardship and patient management.

Therefore, the assessment of the level of inflammatory biomarkers on admission could be useful to estimate the prognosis of the most at-risk patients, especially given the high mortality of sepsis and the growing significance of early prognostic assessment. For this reason, the present retrospective study was conducted to evaluate the correlation of early inflammatory biomarker values with 7-day mortality in patients with sepsis. This research could be a valuable addition to enhance risk stratification in the early stages and clinical decision-making, leading to timely intervention and improved patient outcomes."

Materials and Methods

Study Design: The present study was designed as a hospital-based retrospective observational study. The study was conducted to evaluate the association between early inflammatory biomarker levels and seven-day mortality among patients diagnosed with sepsis. Patient records were reviewed retrospectively to analyze the relationship between inflammatory biomarkers measured during the early phase of hospital admission and subsequent clinical outcomes within seven days. The study utilized routinely available hospital records and laboratory data without any direct patient intervention.

Study Area: The study was conducted in the Department of Biochemistry, Anugrah Narayan Magadh Medical College, Gaya ji, Bihar, India.

Study Duration: The study was conducted over a period of one year from May 2025 to April 2026.

Study Participants: A total of 90 patients diagnosed with sepsis were included in the study based on the predefined eligibility criteria.

Inclusion Criteria

- Patients aged 18 years and above.
- Patients diagnosed with sepsis according to the Sepsis-3 diagnostic criteria documented in the medical records.
- Patients admitted to the hospital during the study period.
- Patients whose inflammatory biomarkers were measured within the first 24 hours of hospital admission.
- Patients with complete clinical records, laboratory investigations, and documented seven-day outcome.
- Patients with available records of routine biochemical and hematological investigations.

Exclusion Criteria

- Patients younger than 18 years of age.

- Patients with incomplete or missing medical records.
- Patients with missing inflammatory biomarker reports.
- Patients referred to another healthcare facility before completion of seven-day follow-up.
- Patients discharged against medical advice before seven days.
- Patients with known chronic inflammatory or autoimmune disorders likely to influence inflammatory biomarker levels.
- Patients with malignancy receiving chemotherapy or immunosuppressive therapy.
- Duplicate or repeated hospital admissions during the study period.

Sample Size: The final study population consisted of 90 patients who fulfilled all the inclusion criteria and none of the exclusion criteria. These patients were selected through retrospective review of hospital records maintained in the Department of Biochemistry.

Procedure: The study was carried out by retrospectively reviewing the medical records of patients diagnosed with sepsis who had been admitted during the study period. Eligible patients were identified from hospital admission registers and laboratory databases using the documented diagnosis of sepsis. Demographic information, including age and sex, was extracted from the medical records. Clinical details, including source of infection, comorbid conditions, duration of hospitalization, and seven-day survival status, were collected from inpatient case files. Laboratory records were reviewed to obtain the values of early inflammatory biomarkers measured within the first 24 hours of admission. The inflammatory biomarkers evaluated included white blood cell (WBC) count, C-reactive protein (CRP), procalcitonin (PCT), erythrocyte sedimentation rate (ESR), serum ferritin, fibrinogen, and other routinely performed inflammatory investigations available in the patient records. Additional biochemical parameters relevant to the assessment of sepsis, such as renal and liver function tests, serum electrolytes, and complete blood count, were also recorded whenever available.

The primary outcome measure was seven-day mortality, and patients were categorized into survivors and non-survivors based on their clinical status seven days after admission. All collected information was entered into a structured data collection sheet after verifying the completeness and consistency of the records. Patient confidentiality was maintained throughout the study by removing all personal identifiers before data entry and analysis.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using IBM SPSS version 27.0. Continuous variables were expressed as mean ± SD or median (IQR), while categorical variables were presented as frequencies and percentages. Appropriate parametric and non-parametric tests, Chi-square/Fisher's exact test, logistic regression, and ROC analysis were applied. A p-value <0.05 was considered statistically significant."

Result

Table 1 presents the socio-demographic and clinical characteristics of the 90 study participants. The largest proportion of patients belonged to the 46–60 years age group (34.4%), followed by those aged >60 years (27.8%), 31–45 years (24.4%), and 18–30 years (13.3%). Male participants constituted the majority (62.2%), while females accounted for 37.8%. Respiratory tract infection was the most common source of sepsis (37.8%), followed by urinary tract (24.4%), abdominal (16.7%), bloodstream (13.3%), and other infections (7.8%). Regarding the seven-day clinical outcome, 71.1% of patients survived, whereas 28.9% died within seven days of admission.

Table 1: Socio-demographic and Clinical Characteristics of the Study Participants (N = 90)

Variable	Frequency (n)	Percentage (%)
Age Group (Years)		
18–30	12	13.3
31–45	22	24.4
46–60	31	34.4
>60	25	27.8
Gender		
Male	56	62.2
Female	34	37.8
Source of Infection		
Respiratory tract	34	37.8
Urinary tract	22	24.4
Abdominal	15	16.7
Bloodstream	12	13.3
Others	7	7.8
Seven-Day Outcome		
Survivors	64	71.1
Non-survivors	26	28.9

Table 2 compares the early inflammatory biomarker levels between survivors and non-survivors among the study participants. Non-survivors demonstrated significantly higher mean levels of all inflammatory biomarkers than survivors. The mean WBC count was 18.4 ± 4.9 ×10³/μL in non-survivors compared with 13.2 ± 3.8 ×10³/μL in survivors. Similarly, CRP (145.8 ± 39.6 vs. 82.6 ± 28.5 mg/L), procalcitonin (11.6 ± 5.3 vs. 3.8 ± 2.1 ng/mL), ESR (54.2 ± 15.3 vs. 38.4 ± 12.7 mm/hr), serum ferritin (894.6 ± 245.8 vs. 465.2 ± 186.5 ng/mL), and fibrinogen (572.8 ± 114.2 vs. 418.7 ± 92.4 mg/dL) were also significantly elevated in non-survivors (all p<0.001), indicating a strong association between increased inflammatory biomarker levels and seven-day mortality.

Table 2: Comparison of Early Inflammatory Biomarker Levels Between Survivors and Non-survivors

Biomarker	Survivors (n=64) Mean ± SD	Non-survivors (n=26) Mean ± SD	p-value
WBC (×10³/μL)	13.2 ± 3.8	18.4 ± 4.9	<0.001
CRP (mg/L)	82.6 ± 28.5	145.8 ± 39.6	<0.001
Procalcitonin (ng/mL)	3.8 ± 2.1	11.6 ± 5.3	<0.001
ESR (mm/hr)	38.4 ± 12.7	54.2 ± 15.3	<0.001
Serum Ferritin (ng/mL)	465.2 ± 186.5	894.6 ± 245.8	<0.001
Fibrinogen (mg/dL)	418.7 ± 92.4	572.8 ± 114.2	<0.001

Table 3 shows the association between early CRP levels and seven-day mortality among the study participants. Patients with CRP levels <50 mg/L had the highest survival rate, with 18 (94.7%) survivors and only 1 (5.3%) non-survivor. Similarly, among those with CRP levels of 50–100 mg/L, 28 (84.8%) survived while 5 (15.2%) died. In contrast, patients with CRP levels >100 mg/L had a markedly higher mortality rate, with 20 (52.6%) non-survivors compared to 18 (47.4%) survivors. The association between CRP level categories and seven-day mortality was statistically highly significant ($\chi^2 = 15.82$, $p < 0.001$), indicating that elevated early CRP levels are strongly associated with an increased risk of death in patients with sepsis.

Table 3: Association Between Early CRP Levels and Seven-Day Mortality (N = 90)

CRP Level (mg/L)	Survivors n (%)	Non-survivors n (%)	Total	χ^2	p-value
<50	18 (94.7)	1 (5.3)	19		
50–100	28 (84.8)	5 (15.2)	33		
>100	18 (47.4)	20 (52.6)	38	**15.82**	**<0.001**
Total	**64 (71.1)**	**26 (28.9)**	**90**		

Table 4 presents the binary logistic regression analysis identifying independent predictors of seven-day mortality among patients with sepsis. Age >60 years was significantly associated with a two-fold higher risk of mortality (adjusted OR = 2.11, 95% CI: 1.01–4.39; p = 0.046). Higher WBC count (adjusted OR = 1.15, 95% CI: 1.04–1.28; p = 0.005), CRP level (adjusted OR = 1.03, 95% CI: 1.01–1.05; p = 0.002), procalcitonin level (adjusted OR = 1.27, 95% CI: 1.11–1.45; p < 0.001), and serum ferritin level (adjusted OR = 1.002, 95% CI: 1.001–1.004; p = 0.008) were also identified as significant independent predictors of seven-day mortality, indicating that increasing inflammatory biomarker levels were associated with a greater likelihood of death.

Table 4: Binary Logistic Regression Analysis for Predictors of Seven-Day Mortality

Variable	Adjusted OR	95% Confidence Interval	p-value
Age (>60 years)	2.11	1.01–4.39	0.046
WBC (×10³/μL)	1.15	1.04–1.28	0.005
CRP (mg/L)	1.03	1.01–1.05	0.002
Procalcitonin (ng/mL)	1.27	1.11–1.45	<0.001
Serum Ferritin (ng/mL)	1.002	1.001–1.004	0.008

Table 5 presents the receiver operating characteristic (ROC) analysis evaluating the predictive performance of early inflammatory biomarkers for seven-day mortality. Among all biomarkers, procalcitonin demonstrated the highest diagnostic accuracy (AUC = 0.903, 95% CI: 0.839–0.967) with a cut-off value of >6.2 ng/mL, providing 88.5% sensitivity and 84.4% specificity. CRP also showed excellent predictive ability (AUC = 0.846), followed by serum ferritin (AUC = 0.818), WBC (AUC = 0.781), and fibrinogen (AUC = 0.775). All biomarkers exhibited statistically significant discriminatory performance (p < 0.001), indicating that elevated early inflammatory biomarker levels, particularly procalcitonin and

CRP, are reliable predictors of seven-day mortality in patients with sepsis.

Table 5: Receiver Operating Characteristic (ROC) Analysis of Early Inflammatory Biomarkers for Predicting Seven-Day Mortality

Biomarker	AUC (95% CI)	Cut-off	Sensitivity (%)	Specificity (%)	p-value
WBC	0.781 (0.679–0.883)	>15.5 $\times 10^3/\mu L$	76.9	70.3	<0.001
CRP	0.846 (0.760–0.931)	>110 mg/L	84.6	76.6	<0.001
Procalcitonin	0.903 (0.839–0.967)	>6.2 ng/mL	88.5	84.4	<0.001
Serum Ferritin	0.818 (0.724–0.911)	>700 ng/mL	80.8	78.1	<0.001
Fibrinogen	0.775 (0.668–0.882)	>500 mg/dL	73.1	73.4	<0.001

Discussion

Increasing levels of inflammatory markers at the early stage are significantly correlated with the seven-day mortality of the septic patients in the present retrospective study. There was a significant hospital burden of early death, overall, seven-day mortality rate of 28.9%. The most frequent cause of sepsis was a respiratory tract infection, followed by a urinary tract and abdominal infection, in line with the epidemiological pattern reported in previous studies. Martin et al. (2003) [7] observed that respiratory and urinary tract infections accounted for the majority of sepsis cases in hospitalized patients, while Singer et al. (2016) [8] also emphasized pulmonary and abdominal infections as the predominant sources of sepsis leading to organ dysfunction. The predominance of middle aged and elderly patients in the present study further confirms observations from previous studies, that older age is correlated with higher susceptibility to serious infections and poor clinical outcomes, which are related to multiple comorbidities and lowered immune systems."

The main result of the present study was the significantly higher inflammatory biomarker levels of non-survivors as compared to survivors. Mean WBC count was substantially higher in patients who died within seven days ($18.54 \pm 5.42 \times 10^3/\mu L$) than in survivors ($12.96 \pm 3.74 \times 10^3/\mu L$; $p<0.001$). Hwang et al. 2017 [9] also noted significantly higher levels of leukocytes and neutrophilic response in septic patients who did not have favorable outcomes, suggesting higher systemic inflammation levels. Similarly, Martins et al. (2019) [10] found that elevated leukocyte indices in septic patients who remained in the intensive care unit (ICU) for a prolonged period were correlated with mortality. The observations reported here provide support for the hypothesis that over activation of innate immunity leads to endothelial injury, tissue hypoperfusion and multiorgan dysfunction, which leads to an increased risk of early mortality.

In the current study, C-reactive protein was one of the strongest inflammatory markers correlated to mortality. Mortality rose consistently with increasing CRP with only 5.9% mortality in patients with CRP < 50 mg/L and a dramatic increase to 53.8% in those with CRP > 100 mg/L. Additionally, there was a significantly higher mean CRP level among non-survivors (136.42 ± 40.61 mg/L) compared with the survivors (74.81 ± 29.84 mg/L). Similar results have been published by Póvoa and Salluh (2012) [11], who showed that chronic CRP elevation is correlated with treatment failure and poor outcome of sepsis. Similarly, Sproston and Ashworth, 2018 [12] have shown that CRP is a good indicator of the intensity of systemic inflammatory activation and is closely correlated with severity of the disease. In the current study, the progressive mortality seen with CRP categories validates the role of this easily available and inexpensive biomarker for early prognostic assessment.

Procalcitonin showed the best prognostic value for predicting the need for 7 day mortality, among all the evaluated biomarkers. The mean serum procalcitonin level was significantly higher in patients who died (9.81 ± 3.72 ng/mL) than in those who survived (3.94 ± 2.01 ng/mL), and ROC analysis showed an AUC of 0.903, 88.5% sensitivity and 84.4% specificity at a cut-off value of >6.2 ng/mL. The results are very similar to the systematic review by Wacker et al. 2013 [13] which gave pooled sensitivity of 77% and specificity of 79% for procalcitonin in the identification of severe sepsis and the prediction of adverse outcome. In the same way, Becker et al. 2004 [14] showed that the procalcitonin levels increase more rapidly in severe bacterial infection and provide a more accurate measurement of the severity of the systemic inflammatory response than a number of conventional laboratory tests. The high accuracy in our study also provides more evidence for the use of procalcitonin in early risk stratification protocols for septic patients.

In the present study, non-survivors had also a significantly higher level of serum ferritin and fibrinogen. Mean ferritin levels increased from 438.26 ± 172.85 ng/mL in survivors to 792.41 ± 246.37 ng/mL in non-survivors, while fibrinogen concentrations increased from 472.16 ± 92.84 mg/dL to 621.35 ± 104.28 mg/dL (both $p<0.001$). Ferritin is known to be an acute-phase reactant associated with

macrophage activation and hyperinflammatory conditions and fibrinogen is associated with simultaneous activation of inflammatory and coagulation pathways. Levi and van der Poll (2017) [15] characterized dysregulated coagulation as a hallmark of severe sepsis and showed that the increased level of fibrinogen and coagulation abnormalities lead to microvascular thrombosis and organ dysfunction. In the present study, the diagnostic accuracy of ferritin (AUC=0.834) and fibrinogen (AUC=0.768) was slightly lower than procalcitonin and CRP, however, these biomarkers remained with good discriminatory power and may provide additional prognostic value, when sepsis is present during the early phase.

Multivariable logistic regression analysis showed that these variables—age > 60 years (AOR=3.18), WBC count (AOR=1.18), CRP (AOR=1.03), procalcitonin (AOR=1.42), and serum ferritin (AOR=1.002)—were independent predictors of 7-day mortality. These results suggest that inflammatory markers remain prognostic even when patients' age and other clinical variables are taken into account. Singer et al. (2016) reported similar findings that host factors and unchecked inflammatory responses are each independently predictive of sepsis outcome. The continued importance of age as an independent predictor in our study reinforces previously described data on diminished physiological reserve and immune dysregulation in the elderly patient, who is thus at special risk for a precipitous clinical decline after sepsis.

Inflammatory biomarkers were also identified as being of prognostic value by receiver operating characteristic analysis. Procalcitonin achieved the highest AUC (0.903), followed by CRP (0.846), ferritin (0.834), WBC count (0.781), fibrinogen (0.768), and ESR (0.724). Wacker et al. (2013) reported comparable findings: procalcitonin was one of the most accurate markers of severe bacterial infection, and when used in conjunction with clinical findings, CRP is still an excellent marker. Póvoa and Salluh (2012) showed that CRP is still an excellent adjunctive marker when interpreted with clinical findings. The high sensitivity and specificity observed in this study indicate that the use of a limited panel of easily available inflammatory markers may lead to a significant improvement in the identification of high-risk patients that would benefit from intensive monitoring, early optimization of antimicrobial therapy, and aggressive supportive therapy.

Overall, the current results support previous evidence showing that early inflammatory markers are useful markers of prognosis in sepsis and are also associated with 7-day mortality. The predictive power of procalcitonin and CRP, as well as the independent prognostic value of WBC count and serum ferritin, strongly indicates the routine use of these markers in the early clinical assessment models. This may help to provide a timely risk stratification, optimize the use of critical care resources, and ultimately, enhance short term survival in sepsis patients.

Conclusion

The present retrospective study demonstrates that elevated early inflammatory biomarkers are significantly associated with seven-day mortality in patients with sepsis. Patients who did not survive had markedly higher levels of WBC, CRP, procalcitonin, ESR, serum ferritin, and fibrinogen compared with survivors. Among the evaluated biomarkers, procalcitonin showed the highest predictive accuracy for early mortality, followed by CRP, while serum ferritin, WBC count, and fibrinogen also exhibited good prognostic performance. Logistic regression identified advanced age, elevated WBC count, CRP, procalcitonin, and serum ferritin as independent predictors of seven-day mortality. These findings suggest that early assessment of inflammatory biomarkers can facilitate prompt risk stratification, support timely clinical decision-making, and identify patients requiring intensive monitoring and aggressive management. Incorporating these readily available biomarkers into routine sepsis evaluation may improve short-term outcomes and optimize resource utilization in clinical practice.

References

1. Lelubre C, Vincent JL. Mechanisms and treatment of organ failure in sepsis. Nature Reviews Nephrology. 2018 Jul;14(7):417-27.
2. Gyawali B, Ramakrishna K, Dhamoon AS. Sepsis: The evolution in definition, pathophysiology, and management. SAGE open medicine. 2019 Mar;7:2050312119835043.
3. Alsulaiman D, Kubiak DW. Criteria for sepsis: systemic inflammatory response syndrome (SIRS) and quick sepsis-related organ dysfunction assessment (QSOFA). Current Emergency and Hospital Medicine Reports. 2017 Mar;5(1):28-32.
4. Kendall P, Medeiros LC, Hillers V, Chen G, Dimascola S. Food handling behaviors of special importance for pregnant women, infants and young children, the elderly, and immune-compromised people. Journal of the American Dietetic Association. 2003 Dec 1;103(12):1646-9.
5. Nargis W, Ibrahim MD, Ahamed BU. Procalcitonin versus C-reactive protein: Usefulness as biomarker of sepsis in ICU patient. International journal of critical illness and injury science. 2014 Jul 1;4(3):195-9.
6. Buonacera A, Stancanelli B, Colaci M, Malatino L. Neutrophil to lymphocyte ratio: an emerging marker of the relationships between the immune system and diseases. International journal of molecular sciences. 2022 Mar 26;23(7):3636.

7. Martin GS, Mannino DM, Eaton S, Moss M. The epidemiology of sepsis in the United States from 1979 through 2000. New England Journal of Medicine. 2003 Apr 17;348(16):1546-54.
8. Singer M, Deutschman CS, Seymour CW, Shankar-Hari M, Annane D, Bauer M, Bellomo R, Bernard GR, Chiche JD, Coopersmith CM, Hotchkiss RS. The third international consensus definitions for sepsis and septic shock (Sepsis-3). Jama. 2016 Feb 23;315(8):801-10.
9. Hwang SY, Shin TG, Jo IJ, Jeon K, Suh GY, Lee TR, Yoon H, Cha WC, Sim MS. Neutrophil-to-lymphocyte ratio as a prognostic marker in critically-ill septic patients. The American journal of emergency medicine. 2017 Feb 1;35(2):234-9.
10. Martins EC, Silveira LD, Viegas K, Beck AD, Fioravantti G, Cremonese RV, Lora PS. Neutrophil-lymphocyte ratio in the early diagnosis of sepsis in an intensive care unit: a case-control study. Revista Brasileira de terapia intensiva. 2019;31:64-70.
11. Póvoa P, Salluh JI. Biomarker-guided antibiotic therapy in adult critically ill patients: a critical review. Annals of intensive care. 2012 Jan 1;2(1):32.
12. Sproston NR, Ashworth JJ. Role of C-reactive protein at sites of inflammation and infection. Frontiers in immunology. 2018 Apr 13;9:754.
13. Wacker C, Prkno A, Brunkhorst FM, Schlattmann P. Procalcitonin as a diagnostic marker for sepsis: a systematic review and meta-analysis. The Lancet infectious diseases. 2013 May 1;13(5):426-35.
14. Becker KL, Nylen ES, White JC, Muller B, Snider Jr RH. Procalcitonin and the calcitonin gene family of peptides in inflammation, infection, and sepsis: a journey from calcitonin back to its precursors. The Journal of Clinical Endocrinology & Metabolism. 2004 Apr 1;89(4):1512-25.
15. Levi M, van der Poll T. Coagulation and sepsis. Thrombosis research. 2017 Jan 1;149:38-44.