Original Research

Role of Hematological and Histopathological Parameters in the Diagnosis and Prognosis of Sepsis in Critical Care Units

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ABSTRACT

Aim: Sepsis remains a leading cause of morbidity and mortality in intensive care units (ICUs), necessitating early diagnosis and risk stratification. This study aims to evaluate the role of hematological and histopathological parameters in the diagnosis and prognosis of sepsis to improve clinical decision-making and patient outcomes.

Materials and Methods: A prospective observational study was conducted over 12 months in the ICU of a tertiary care hospital. A total of 120 patients diagnosed with sepsis based on Sepsis-3 criteria were included. Hematological investigations, including total leukocyte count (TLC), neutrophil-to-lymphocyte ratio (NLR), platelet count, and coagulation markers, were assessed at admission and at 24, 48, and 72 hours. Histopathological analyses were performed on tissue biopsies obtained from liver, kidney, bone marrow, and skin in indicated cases. Microbiological cultures were conducted to identify bacterial, fungal, and polymicrobial infections. Patients were categorized into mild sepsis, severe sepsis, and septic shock groups, and clinical outcomes, including 28-day mortality, ICU length of stay, and incidence of multiple organ dysfunction syndrome (MODS) and disseminated intravascular coagulation (DIC), were analyzed.

Results: The mean age of patients was 58.60 ± 12.30 years, with a male predominance (65.00%). Leukocyte counts and NLR increased significantly with sepsis severity ($p < 0.001$), while platelet counts decreased, correlating with the presence of DIC ($p < 0.001$). Coagulation markers, including PT, aPTT, and INR, were significantly prolonged in severe sepsis and septic shock ($p < 0.001$). Histopathological findings revealed liver necrosis (55.00%), renal tubular injury (50.00%), and bone marrow suppression (40.00%) in septic shock cases ($p < 0.001$). Microbiological analysis identified gram-negative bacteria (65.00%) as the predominant pathogens, with a notable increase in fungal infections (20.00%) in severe cases ($p < 0.050$). Mortality rates increased significantly with sepsis severity, reaching 65.00% in septic shock ($p < 0.001$), and ICU stay was significantly longer in these patients (18.70 ± 5.20 days, $p < 0.001$).

Conclusion: Hematological and histopathological parameters serve as crucial diagnostic and prognostic tools in sepsis. Increased leukocyte count, elevated NLR, thrombocytopenia, and coagulation abnormalities correlate with disease severity and poor outcomes. Histopathological findings provide valuable insights into organ dysfunction in sepsis, particularly in liver, kidney, and bone marrow involvement. The predominance of gram-negative infections and high mortality in septic shock emphasize the need for early identification and targeted therapeutic interventions.

Keywords: Sepsis, Hematological parameters, Histopathology, Prognosis, Critical care

Introduction

Sepsis is a life-threatening condition characterized by a dysregulated immune response to infection, leading to widespread inflammation, organ dysfunction, and, in severe cases, multi-organ failure. It remains one of the most significant causes of morbidity and mortality in intensive care units (ICUs) worldwide, with a growing burden on healthcare systems. The early and accurate diagnosis of sepsis is critical for timely intervention, as delays in treatment can significantly worsen patient outcomes. Despite advancements in critical care and antimicrobial therapy, the diagnosis of sepsis remains a challenge due to its complex and heterogeneous presentation. In recent years,

hematological and histopathological parameters have gained increasing attention as potential biomarkers for the early detection, severity assessment, and prognosis of sepsis.[1]The pathophysiology of sepsis involves a complex interplay between the host immune response and the invading pathogens. Upon infection, the immune system activates a cascade of inflammatory mediators, including cytokines, interleukins, and chemokines. This systemic inflammatory response often leads to endothelial dysfunction, coagulation abnormalities, and tissue damage, which contribute to the progression of sepsis. Hematological markers, such as leukocyte count, neutrophil-to-lymphocyte ratio (NLR), platelet count, and coagulation parameters, have been widely studied as indicators of the systemic response to sepsis. These markers provide valuable information on the immune status, inflammatory burden, and coagulation abnormalities that occur in septic patients.[2]Leukocyte count is one of the primary hematological parameters used in the diagnosis of sepsis. An elevated total leukocyte count, particularly with neutrophilia, suggests an ongoing infectious process and an active immune response. Conversely, leukopenia may be indicative of immune suppression or bone marrow exhaustion in advanced stages of sepsis. The neutrophil-to-lymphocyte ratio (NLR) has emerged as a reliable marker of systemic inflammation and has been associated with sepsis severity and patient prognosis. An increased NLR reflects heightened neutrophilic activity and lymphocyte depletion, both of which are hallmarks of the immune dysregulation observed in sepsis.[3]Platelet count and coagulation parameters are also crucial in assessing the severity and prognosis of sepsis. Thrombocytopenia, or a decreased platelet count, is frequently observed in septic patients and is often associated with disseminated intravascular coagulation (DIC), a severe complication of sepsis that leads to widespread microvascular thrombosis and bleeding tendencies. Elevated prothrombin time (PT), activated partial thromboplastin time (aPTT), and international normalized ratio (INR) suggest coagulation dysfunction, which can contribute to poor clinical outcomes. Furthermore, the presence of schistocytes on peripheral blood smear analysis may indicate microangiopathic hemolyticanemia, a condition often seen in septic patients with DIC.[4]Apart from hematological markers, histopathological examination of tissue samples provides valuable insights into the pathological changes that occur in sepsis. Tissue biopsies obtained from affected organs, such as the liver, kidney, lungs, and bone marrow, can reveal characteristic histopathological changes associated with sepsis-induced organ dysfunction. For instance, liver biopsies may show hepatocellular necrosis, congestion, and microvascular thrombosis, which are indicative of sepsis-related liver injury. Similarly, renal biopsies often demonstrate acute tubular necrosis, interstitial inflammation, and endothelial injury, all of which contribute to sepsis-associated acute kidney injury.[5]The bone marrow plays a critical role in the immune response to sepsis, as it is responsible for the production of leukocytes, platelets, and red blood cells. Histopathological examination of bone marrow aspirates and biopsies in septic patients may reveal myeloid hyperplasia, hypocellularity, or hemophagocytosis, depending on the stage of the disease. Myeloid hyperplasia suggests an increased demand for neutrophil production in response to infection, while hypocellularity indicates bone marrow suppression due to overwhelming sepsis. Hemophagocytosis, a process in which activated macrophages engulf hematopoietic cells, is frequently observed in patients with sepsis-associated hemophagocytic lymphohistiocytosis (HLH), a severe hyperinflammatory syndrome that carries a high mortality risk.[6]Histopathological findings in sepsis are not limited to solid organ damage but also extend to vascular and endothelial dysfunction. Septic shock, the most severe form of sepsis, is often associated with endothelial injury, capillary leakage, and microvascular thrombosis. These pathological changes contribute to the characteristic hypotension and tissue hypoxia observed in septic patients. Skin and soft tissue biopsies from patients with sepsis may exhibit leukocytoclastic vasculitis, microthrombi, and necrosis, particularly in cases of purpura fulminans or necrotizing fasciitis.[7]While hematological and histopathological parameters provide valuable diagnostic and prognostic information, their integration into clinical practice remains a challenge. The dynamic nature of sepsis necessitates serial monitoring of these parameters to assess disease progression and treatment response accurately. Automated hematologicalanalyzers have made it possible to rapidly assess blood counts and coagulation markers, thereby aiding in the early diagnosis of sepsis. However, histopathological examination requires tissue sampling, which may not always be feasible in critically ill patients. Advances in minimally invasive biopsy techniques and imaging-guided sampling have improved the feasibility of obtaining histopathological data in septic patients.The identification of reliable biomarkers for sepsis remains an area of active research. The combination of hematological markers, histopathological findings, and emerging molecular biomarkers, such as procalcitonin, C-reactive protein (CRP), and cell-free DNA, has the potential to enhance the accuracy of sepsis diagnosis and prognosis. Machine learning and artificial intelligence-based algorithms are also being explored to integrate these parameters into predictive models for early sepsis detection and risk stratification.[8]Hematological and histopathological parameters play a crucial role in the diagnosis and prognosis of sepsis. While hematological markers provide rapid and non-invasive insights into the immune response and coagulation status, histopathological examination offers a deeper

understanding of the tissue-level changes associated with sepsis-induced organ dysfunction. The integration of these parameters into clinical practice has the potential to improve sepsis recognition, facilitate early intervention, and ultimately enhance patient outcomes.

Materials and Methods

This prospective observational study was conducted over a period of 12 months in the Intensive Care Unit (ICU) at a tertiary care hospital. The study involved collaboration between the Department of Medicine and the Department of Pathology. Ethical approval was obtained from the Institutional Ethics Committee, and informed consent was secured from all participants or their legal representatives.A total of 120 patients diagnosed with sepsis were included in the study. The inclusion criteria comprised patients aged 18 years or older who were admitted to the ICU with a clinical diagnosis of sepsis based on the Sepsis-3 criteria, which includes a Sequential Organ Failure Assessment (SOFA) score of at least two points due to suspected infection. Patients with confirmed or suspected bacterial, viral, or fungal infections were considered for inclusion, provided complete hematological and histopathological data were available. Patients were excluded if they had pre-existing hematological malignancies, chronic liver disease, were receiving immunosuppressive therapy, chemotherapy, or radiotherapy, were pregnant, or had incomplete medical records or declined participation.

Methodology

Upon admission, all patients underwent a comprehensive clinical assessment and were categorized into three groups based on disease severity: mild sepsis (SOFA score 2-4), severe sepsis (SOFA score 5-9), and septic shock (SOFA score ≥10 requiring vasopressor support). Hematological and histopathological parameters were evaluated at admission and subsequently at 24, 48, and 72 hours to assess disease progression and prognosis.

Hematological Investigations

Blood samples were collected within six hours of ICU admission and repeated at 24, 48, and 72 hours. The hematological parameters assessed included complete blood count (CBC), total leukocyte count (TLC), neutrophil-lymphocyte ratio (NLR), platelet count, and hematocrit. The coagulation profile was analyzed through prothrombin time (PT), activated partial thromboplastin time (aPTT), and international normalized ratio (INR). Sepsis biomarkers such as C-reactive protein (CRP), procalcitonin (PCT), and ferritin were measured. Additionally, peripheral blood smear (PBS) evaluation was performed to assess morphological changes indicative of sepsis, including toxic granulation, band forms, and schistocytes in cases of disseminated intravascular coagulation (DIC).

Histopathological and Tissue Analysis

Histopathological evaluation was conducted on biopsy samples obtained from patients with suspected septic emboli, skin necrosis, or organ dysfunction. Liver and renal biopsies were performed in post-mortem cases or in patients with suspected sepsis-induced multiple organ failure. In selected cases of persistent thrombocytopenia or pancytopenia, bone marrow aspiration and biopsy were carried out to rule out marrow suppression or hemophagocytic lymphohistiocytosis (HLH).

Microbiological and Culture Analysis

Blood cultures were collected in aerobic and anaerobic culture bottles before the initiation of antibiotics. Additionally, body fluid cultures, including sputum, urine, cerebrospinal fluid (CSF), ascitic fluid, and pleural fluid, were analyzed for microbial identification and antibiotic susceptibility patterns.

Outcome Measures

Patients were monitored for 28-day mortality, ICU length of stay, and complications such as disseminated intravascular coagulation (DIC), acute respiratory distress syndrome (ARDS), and multiple organ dysfunction syndrome (MODS). The correlation between hematological and histopathological findings with clinical outcomes was analyzed to determine their prognostic significance.

Statistical Analysis

Data were analyzed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range) and compared using the Student's t-test or Mann-Whitney U test. Categorical variables were expressed as percentages and compared using the Chi-square test or Fisher's exact test. Survival analysis was conducted using the Kaplan-Meier method, and predictors of mortality were assessed through Cox proportional hazards regression. A p-value of less than 0.05 was considered statistically significant.

Results

Baseline Characteristics of Patients

The study included a total of 120 patients diagnosed with sepsis. The mean age of the patients was 58.60 ± 12.30 years, indicating that the majority of the affected individuals were middle-aged or elderly. Among the study population, 78 patients (65.00%) were male, suggesting a higher prevalence of sepsis among men. Comorbidities were present in 48 patients (40.00%), with 30 patients (25.00%) having diabetes and 36 patients (30.00%) having hypertension. Chronic kidney disease was observed in 18 patients (15.00%), reflecting its role as a predisposing factor for severe infections. These findings suggest that older age, male gender, and

underlying health conditions may contribute to the severity of sepsis and its associated complications.

Hematological Parameters at Admission

Hematological parameters were significantly altered across different severity groups. The total leukocyte count increased as sepsis severity progressed, with mild sepsis patients having a mean count of 11.20 ± 2.50 x10⁹/L, severe sepsis patients having 14.50 ± 3.20 x10⁹/L, and septic shock patients reaching 18.30 ± 4.80 x10⁹/L ($p < 0.001$). This trend reflects an exaggerated immune response in severe infections. The neutrophil-lymphocyte ratio (NLR), an established inflammatory marker, was significantly higher in septic shock (10.20 ± 3.50) compared to severe (7.60 ± 2.10) and mild sepsis (4.80 ± 1.20) ($p < 0.001$), indicating its potential as a prognostic marker. Platelet count was inversely related to sepsis severity, decreasing from 200.00 ± 50.00 x10⁹/L in mild cases to 110.00 ± 40.00 x10⁹/L in septic shock ($p < 0.001$), which suggests platelet consumption due to disseminated intravascular coagulation (DIC). Hematocrit levels were also reduced in severe sepsis and septic shock, likely due to fluid resuscitation and anemia associated with critical illness ($p < 0.050$).

Coagulation and Sepsis Biomarkers

Coagulation abnormalities were observed across all severity groups, with worsening dysfunction in septic shock. Prothrombin time (PT) increased significantly from 14.20 ± 2.10 seconds in mild sepsis to 22.10 ± 4.50 seconds in septic shock ($p < 0.001$), reflecting impaired coagulation and fibrinolysis. A similar trend was noted for activated partial thromboplastin time (aPTT), which increased from 34.50 ± 5.40 seconds in mild sepsis to 48.30 ± 8.70 seconds in septic shock ($p < 0.001$). The international normalized ratio (INR) also showed a progressive rise, indicating worsening coagulopathy ($p < 0.001$). Among sepsis biomarkers, C-reactive protein (CRP) levels increased from 50.00 ± 20.00 mg/L in mild cases to 140.00 ± 45.00 mg/L in septic shock ($p < 0.001$), demonstrating its role as an inflammatory marker. Procalcitonin levels also showed a significant increase, with values rising from 1.80 ± 0.90 ng/mL in mild sepsis to 8.90 ± 3.20 ng/mL in septic shock ($p < 0.001$), reinforcing its role as a key indicator of bacterial infection severity.

Histopathological Findings in Tissue Biopsies

Histopathological examinations revealed significant organ involvement in patients with severe sepsis and septic shock. Liver necrosis was observed in 22 patients (55.00%) with septic shock, compared to 12 (30.00%) in severe sepsis and 4 (10.00%) in mild sepsis ($p < 0.001$). Renal tubular injury was present in 50.00% of septic shock patients, which correlates with acute kidney injury due to sepsis-related hypoperfusion ($p < 0.001$). Bone marrow suppression was observed in 40.00% of septic shock patients, suggesting impaired hematopoiesis due to overwhelming infection or immune dysregulation ($p < 0.050$). Additionally, septic emboli were identified in 25.00% of septic shock cases, indicating microvascular thrombosis and perfusion abnormalities that may contribute to multi-organ dysfunction syndrome (MODS).

Microbiological Culture Results

Blood culture and microbiological analysis identified the predominant pathogens in sepsis patients. Gram-negative bacterial infections were more common, detected in 12 patients (30.00%) with mild sepsis, 20 (50.00%) with severe sepsis, and 26 (65.00%) with septic shock ($p < 0.001$). Gram-positive bacterial infections were also significant, with 20.00% in mild sepsis, 35.00% in severe sepsis, and 45.00% in septic shock ($p < 0.050$). Fungal infections were found in 20.00% of septic shock cases, suggesting an increased risk of secondary fungal infections in critically ill patients ($p < 0.050$). Polymicrobial infections were present in 25.00% of septic shock patients, which reflects the complexity of infections in ICU settings and the challenge of targeted therapy ($p < 0.050$).

Patient Outcomes Based on Sepsis Severity

The study revealed significant differences in outcomes based on sepsis severity. The 28-day mortality rate increased from 10.00% in mild sepsis to 65.00% in septic shock ($p < 0.001$), emphasizing the high fatality associated with advanced sepsis. The length of ICU stay was significantly longer in patients with septic shock (18.70 ± 5.20 days) compared to severe sepsis (12.30 ± 3.50 days) and mild sepsis (6.50 ± 2.10 days) ($p < 0.001$), reflecting prolonged recovery and complications in critically ill patients. MODS incidence was highest in septic shock (70.00%), followed by severe sepsis (40.00%) and mild sepsis (15.00%) ($p < 0.001$), which is consistent with worsening physiological derangements in severe infections. Similarly, DIC incidence was markedly higher in septic shock (45.00%), suggesting a strong correlation between coagulation disturbances and poor prognosis ($p < 0.001$).

Table 1: Baseline Characteristics of Patients

Characteristic	Number (n)/ Mean ± SD	Percentage (%)
Age (years)	58.60 ± 12.30	-
Male	78	65.00%
Comorbidities	48	40.00%
Diabetes	30	25.00%
Hypertension	36	30.00%

Chronic Kidney Disease	18	15.00%

Table 2: Hematological Parameters at Admission

Parameter	Mild Sepsis (Mean ± SD)	Severe Sepsis (Mean ± SD)	Septic Shock (Mean ± SD)	p-value
Total Leukocyte Count (x10⁹/L)	11.20 ± 2.50	14.50 ± 3.20	18.30 ± 4.80	<0.001
Neutrophil-Lymphocyte Ratio	4.80 ± 1.20	7.60 ± 2.10	10.20 ± 3.50	<0.001
Platelet Count (x10⁹/L)	200.00 ± 50.00	150.00 ± 45.00	110.00 ± 40.00	<0.001
Hematocrit (%)	40.00 ± 5.00	36.00 ± 4.00	32.00 ± 5.00	<0.050

Table 3: Coagulation and Sepsis Biomarkers

Parameter	Mild Sepsis (Mean ± SD)	Severe Sepsis (Mean ± SD)	Septic Shock (Mean ± SD)	p-value
Prothrombin Time (seconds)	14.20 ± 2.10	17.30 ± 3.20	22.10 ± 4.50	<0.001
aPTT (seconds)	34.50 ± 5.40	40.80 ± 6.50	48.30 ± 8.70	<0.001
INR	1.10 ± 0.20	1.30 ± 0.30	1.60 ± 0.40	<0.001
C-Reactive Protein (mg/L)	50.00 ± 20.00	90.00 ± 30.00	140.00 ± 45.00	<0.001
Procalcitonin (ng/mL)	1.80 ± 0.90	4.20 ± 1.50	8.90 ± 3.20	<0.001

Table 4: Histopathological Findings in Tissue Biopsies

Finding	Mild Sepsis (n=40)	Severe Sepsis (n=40)	Septic Shock (n=40)	p-value
Liver Necrosis (%)	4 (10.00%)	12 (30.00%)	22 (55.00%)	<0.001
Renal Tubular Injury (%)	2 (5.00%)	10 (25.00%)	20 (50.00%)	<0.001
Bone Marrow Suppression (%)	1 (2.00%)	6 (15.00%)	16 (40.00%)	<0.050
Septic Emboli (%)	1 (3.00%)	4 (10.00%)	10 (25.00%)	<0.050

Table 5: Microbiological Culture Results

Pathogen	Mild Sepsis (n=40)	Severe Sepsis (n=40)	Septic Shock (n=40)	p-value
Gram-Negative Bacteria (%)	12 (30.00%)	20 (50.00%)	26 (65.00%)	<0.001
Gram-Positive Bacteria (%)	8 (20.00%)	14 (35.00%)	18 (45.00%)	<0.050
Fungal Infections (%)	2 (5.00%)	4 (10.00%)	8 (20.00%)	<0.050
Polymicrobial (%)	2 (5.00%)	6 (15.00%)	10 (25.00%)	<0.050

Table 6: Patient Outcomes Based on Sepsis Severity

Outcome	Mild Sepsis (n=40)	Severe Sepsis (n=40)	Septic Shock (n=40)	p-value
28-day Mortality (%)	4 (10.00%)	14 (35.00%)	26 (65.00%)	<0.001
ICU Length of Stay (days)	6.50 ± 2.10	12.30 ± 3.50	18.70 ± 5.20	<0.001
MODS Incidence (%)	6 (15.00%)	16 (40.00%)	28 (70.00%)	<0.001
DIC Incidence (%)	2 (5.00%)	8 (20.00%)	18 (45.00%)	<0.001

Discussion

The findings of this study align with previous research indicating that older age and male gender are significant risk factors for sepsis. The mean age of 58.60 ± 12.30 years suggests that the immune response weakens with aging, leading to increased susceptibility to infections. Similarly, the male predominance (65.00%) observed in this study is

consistent with prior studies that suggest hormonal and genetic factors may contribute to gender-based differences in immune response and sepsis susceptibility. Comorbid conditions, including diabetes (25.00%), hypertension (30.00%), and chronic kidney disease (15.00%), were common among septic patients, reinforcing the role of underlying diseases in increasing sepsis risk and severity. A study by Martin et al. (2006) demonstrated similar demographic trends, reporting that sepsis incidence was higher in males and older adults, with a strong association between sepsis severity and comorbid conditions such as diabetes and chronic kidney disease.[9]

The observed increase in total leukocyte count (TLC) with sepsis severity ($p < 0.001$) aligns with findings from previous studies that show leukocytosis as a hallmark of sepsis due to immune system activation. Additionally, the neutrophil-lymphocyte ratio (NLR) was significantly higher in septic shock (10.20 ± 3.50) compared to mild sepsis (4.80 ± 1.20) ($p < 0.001$), reinforcing its role as a prognostic marker. Similar trends were reported by Zahorec (2001), who demonstrated that an elevated NLR is a reliable indicator of sepsis progression and systemic inflammatory response. The platelet count declined as sepsis severity increased ($p < 0.001$), likely due to platelet consumption in disseminated intravascular coagulation (DIC), a common complication in sepsis. Decreasing hematocrit levels in severe sepsis and septic shock indicate hemodilution due to aggressive fluid resuscitation and anemia associated with critical illness.[10]

Coagulation abnormalities observed in this study further confirm the link between sepsis severity and hemostatic dysfunction. The progressive increase in prothrombin time (PT), activated partial thromboplastin time (aPTT), and international normalized ratio (INR) ($p < 0.001$) reflects impaired coagulation control, which has been widely reported in septic patients. Moreover, elevated C-reactive protein (CRP) and procalcitonin (PCT) levels indicate persistent inflammation, with both markers increasing significantly as sepsis worsened ($p < 0.001$). A study by Matull et al. (2006) highlighted the role of CRP and PCT in differentiating sepsis severity, where PCT was particularly effective in predicting bacterial infections and sepsis progression.[11]

Histopathological evaluation revealed that liver necrosis was significantly more common in septic shock (55.00%) than in mild sepsis (10.00%) ($p < 0.001$), suggesting that hypoperfusion and oxidative stress play a role in sepsis-induced hepatic dysfunction. Additionally, renal tubular injury was identified in 50.00% of septic shock patients, correlating with acute kidney injury (AKI) secondary to sepsis-induced hypotension. These findings are in agreement with the work of Langenberg et al. (2006), who demonstrated that renal injury in sepsis is driven by altered perfusion dynamics rather than ischemia alone. Furthermore, bone marrow suppression (40.00%) in septic shock patients indicates the impact of systemic inflammation on hematopoiesis. Septic emboli (25.00%), indicative of microvascular thrombosis, were also significantly associated with severe cases, reinforcing the concept of sepsis-induced coagulopathy.[12]

Microbiological analysis identified gram-negative bacteria as the predominant causative pathogens, with a significant increase in incidence from mild sepsis (30.00%) to septic shock (65.00%) ($p < 0.001$). This aligns with the findings of Vincent et al. (2009), who reported that gram-negative organisms, particularly *Escherichia coli* and *Klebsiella pneumoniae*, are responsible for the majority of sepsis cases. Gram-positive bacterial infections also increased with sepsis severity ($p < 0.050$), reflecting their role in bloodstream infections. Notably, fungal infections were observed in 20.00% of septic shock patients, a finding consistent with increasing fungal sepsis incidence in critically ill patients. Additionally, polymicrobial infections (25.00%) in septic shock cases highlight the complexity of ICU-acquired infections and the challenge of targeted antimicrobial therapy.[13]

Patient outcomes varied significantly based on sepsis severity. The 28-day mortality rate increased from 10.00% in mild sepsis to 65.00% in septic shock ($p < 0.001$), reflecting the devastating consequences of sepsis-induced organ failure. Similar findings were reported by Kaukonen et al. (2014), who found that mortality rates in septic shock were markedly higher than in less severe forms of sepsis. Additionally, ICU length of stay was significantly longer in septic shock (18.70 ± 5.20 days) compared to mild sepsis (6.50 ± 2.10 days) ($p < 0.001$), emphasizing the burden of prolonged hospitalization. MODS incidence (70.00%) in septic shock patients was significantly higher than in mild cases (15.00%) ($p < 0.001$), confirming its role as a key predictor of mortality. Moreover, DIC incidence (45.00%) in septic shock indicates a strong association between coagulation abnormalities and poor prognosis. These findings highlight the importance of early sepsis recognition and aggressive management to improve patient outcomes.[14]

Conclusion

This study highlights the significant role of hematological and histopathological parameters in the diagnosis and prognosis of sepsis. Elevated total leukocyte count, neutrophil-to-lymphocyte ratio (NLR), and procalcitonin levels were strongly associated with disease severity, while thrombocytopenia and coagulation abnormalities correlated with poor outcomes. Histopathological findings, including liver necrosis, renal tubular injury, and bone marrow suppression, provided valuable insights into sepsis-induced organ dysfunction. The predominance of gram-negative infections and increased incidence of polymicrobial and fungal

infections in septic shock emphasize the complexity of sepsis management. Given the high mortality rate (65%) in septic shock patients, early detection using hematological markers and histopathological evaluations is crucial for timely interventions and improved survival outcomes.

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