

Early Recognition, Biochemical Markers and Critical Care Strategies for Sepsis

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Introduction

Sepsis remains one of the most pressing challenges in modern critical care medicine, representing a complex syndrome that arises from a dysregulated host response to infection, leading to life-threatening organ dysfunction. Despite decades of research, sepsis continues to cause significant morbidity and mortality worldwide, accounting for millions of deaths annually and overwhelming healthcare systems, particularly in low- and middle-income countries. The heterogeneity of sepsis makes it especially difficult to diagnose and manage, as it presents across a spectrum of infections, organ failures, and host vulnerabilities. Early recognition is vital, as delays in diagnosis and intervention are directly correlated with poor outcomes. Biochemical markers have emerged as valuable tools to complement clinical suspicion, guiding diagnosis, prognostication, and treatment strategies. The convergence of early recognition, biomarker-driven diagnosis, and intensive care strategies forms the cornerstone of improving survival in septic patients, underscoring the need for timely, precise, and coordinated care [1].

Description

Sepsis is fundamentally a syndrome of host-pathogen interaction gone awry, characterized by an overwhelming inflammatory response coupled with immune dysregulation and metabolic dysfunction. The challenge in early recognition lies in its protean manifestations, which can range from subtle tachycardia and fever to fulminant shock and multi-organ failure. Traditional diagnostic criteria, such as the systemic inflammatory response syndrome (SIRS) definition, were criticized for being overly sensitive and non-specific, prompting the development of the Sepsis-3 definition. Under this framework, sepsis is defined as suspected infection plus acute organ dysfunction, measured through the Sequential Organ Failure Assessment (SOFA) score. For frontline settings, the quick SOFA (qSOFA) score—based on altered mentation, systolic blood pressure ≤ 100 mmHg, and respiratory rate ≥ 22 breaths per minute—provides a pragmatic screening tool. However, both clinical scoring systems and bedside suspicion require supplementation with biochemical markers to enhance diagnostic accuracy [2].

Biochemical markers play a pivotal role in the early recognition and stratification of sepsis. Among these, procalcitonin has emerged as a widely studied biomarker, rising rapidly in bacterial infections and correlating with disease severity. Its kinetics allows for both early diagnosis and therapeutic guidance, particularly in antibiotic stewardship programs. Novel biomarkers such as presepsin (sCD14-ST), interleukin-6 (IL-6), and Triggering Receptor Expressed On Myeloid cells-1 (TREM-1) are gaining attention for their diagnostic potential, though their widespread clinical use remains limited by cost and availability. Lactate, a marker of tissue hypoperfusion and metabolic stress, occupies a unique role as both a diagnostic and prognostic indicator. Persistent hyperlactatemia is strongly associated with increased mortality, guiding both resuscitation strategies and risk stratification in the ICU. The combination of clinical suspicion, organ dysfunction assessment, and biomarker analysis forms the backbone of early sepsis diagnosis [3].

Critical care strategies for sepsis revolve around timely and coordinated interventions. The Surviving Sepsis Campaign (SSC) guidelines have established structured bundles that emphasize immediate resuscitation within the “golden hours.” Central to these strategies is the prompt initiation of empiric broad-spectrum antibiotics, ideally within one hour of recognizing sepsis or septic shock. Delay in antimicrobial administration has been consistently linked to increased mortality. Source control—whether through abscess drainage, removal of infected catheters, or surgical intervention—is equally critical, as persistent infection perpetuates systemic dysfunction. Fluid resuscitation with crystalloids, guided by frequent hemodynamic reassessment, remains the first-line approach to restoring perfusion [4].

Hemodynamic management in septic shock typically begins with norepinephrine as the first-choice vasopressor, titrated to maintain Mean Arterial Pressure (MAP) ≥ 65 mmHg. Adjunctive agents such as vasopressin and epinephrine may be employed in refractory cases, while inotropes like dobutamine can augment cardiac output when myocardial dysfunction is evident. Biochemical markers not only aid in diagnosis but also guide therapeutic decisions in the ICU. Lactate clearance serves as both a resuscitation endpoint and a prognostic indicator, with declining levels reflecting improved tissue perfusion [5].

Conclusion

Sepsis epitomizes the challenges of critical care medicine, where rapid recognition, precise diagnostics, and coordinated interventions determine survival. The integration of early recognition strategies, clinical scoring systems, and biochemical markers such as procalcitonin, lactate, and emerging immunological mediators has advanced our ability to detect sepsis earlier and treat it more effectively. Future directions will likely harness precision medicine approaches, combining molecular diagnostics with targeted therapies to further improve outcomes. At the same time, global health efforts must ensure that early recognition and critical care strategies are implemented across all healthcare settings, bridging disparities in resources and outcomes. Ultimately, the synergy of early detection, biomarker guidance, and intensive care interventions offers the greatest promise for reducing the global burden of sepsis and improving both short-term survival and long-term recovery in affected patients.

Acknowledgement

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Conflict of Interest

None.

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