

Chapter 22

Serum Procalcitonin, Ischemia Modified Albumin Biomarkers in Tertiary Hospital Sepsis Patients

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ABSTRACT

Sepsis is described as a medical emergency situation due to the body's systemic immunological response to an infection that can lead to end-stage organ dysfunction and even death. Sepsis is one of the major causes of morbidity and mortality in critically morbid patients; however, there is a significant advancement in the understanding of the pathophysiology of this clinical syndrome, hemodynamic monitoring tools, and resuscitation measures. Due to the body's systemic immune reaction to an infection, which may result in end-stage organ malfunction and even death, sepsis is referred to as a medical emergency. It is one of the top ten leading causes of death globally. The epidemiological burden of sepsis is difficult to ascertain globally. Despite tremendous progress in the understanding of the pathogenesis of this clinical illness, hemodynamic monitoring systems, and resuscitation techniques, sepsis continues to be one of the leading causes of morbidity and mortality in critically ill patients.

1. INTRODUCTION

The Germ theory of disease in the 1800 century recognizes that sepsis is a debilitating and dangerous condition. Sepsis caused due to infection stimulates the host's defense to an extent where the host's own tissues are damaged (Cetinkaya, et al., 2014). Once the infectious agent invades the host's body, macrophages get activated and phagocytize them (Chuang, et al., 2006). This causes the secretion of pro-inflammatory cytokines activating the innate immunity of the body and recruitment of white blood cells (WBC / neutrophils) to the inflammatory site (Rad, et al., 2020). After phagocytosis, the neutrophils

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& macrophages kill bacteria through a combination of several methods like enzymatic degradation or respiratory burst producing free radicals and killing the bacteria by lipid peroxidation process, cross-link formation and DNA damage (Hu, et al., 2018). These mechanisms eradicate bacteria efficiently (Elewa, et al., 2015).

The inflammatory reaction could, however, be heightened, and bacteria could escape. Sepsis usually results from an overactive inflammatory response, but it can also be brought on by a serious bacterial infection that triggers a powerful inflammatory response and results in collateral damage (Kumar and Anand, 2016). In patients with severe sepsis, the main factor contributing to morbidity and mortality is the onset of widespread tissue hypoperfusion and oxidative damage. Cells and tissues are severely damaged by oxidative stress as a result of widespread reactive oxygen species (ROS) generation and tissue hypoperfusion and hypoxia (Boina, 2022). Ischemia and oxidative damage brought on by sepsis are thought to play a significant part in the pathophysiology of organ dysfunction and to be the first stage in the progression of multiple organ failure and mortality (Yin, et al., 2017).

An intelligent clinical response may benefit from prompt recognition of the aforementioned development (Pandya, et al., 2022). The oxidative alteration of cellular macromolecules, promotion of cell death by apoptosis, and structural damage to tissues are all consequences of the sepsis' hyper oxidative state, which comes from an imbalance between oxidants and antioxidants. Additionally, as a result of the counter-regulatory mechanisms activated by the onset of sepsis, inflammatory macromolecules such cytokines, chemokines, and vasoactive peptides are released (Vashishtha & Dhawan, 2023). These biomolecules have an immediate impact on metabolism in terms of cells, tissues, and organs. Mitochondrial dysfunction and oxygen utilization inhibition are two such effects that have been linked to sepsis. Therefore, it is crucial to analyze and interpret not just one test but a series of tests in order to understand and clarify the pathophysiologic mechanisms underlying sepsis (Jeganathan, et al., 2023).

Early classification and identification of patients who are more likely to die present severe clinical challenges for healthcare professionals, as prompt selection of the most effective therapeutic strategy and location for care are both essential in healthcare settings (Koksal, et al., 2007). In order to comprehend and clarify the pathophysiological mechanisms that take place in sepsis patients, it is crucial to analyze and interpret the findings of a variety of tests rather than just one. Numerous biomarkers have also been suggested concurrently. Procalcitonin is the most well-investigated biomarker in patients with sepsis or septic shock (PCT). Although PCT is correlated with organ failure and sepsis severity, specific outcomes can be predicted through dynamic changes in PCT levels during sepsis.

2. REVIEW OF LITERATURE

The relevance of lactate in sepsis patients as a biomarker of risk is of particular interest. As a sign of altered tissue perfusion, lactate has frequently been utilized as a marker. Greater lactate concentrations are associated with higher mortality rates, whereas hospital-associated lactate clearance is a marker for lower mortality rates. It is obvious that metabolic acidosis results in sepsis as a side effect of lactate buildup. Severe lactate buildup or decreased lactate clearance may be the causes of elevated serum lactate levels in sepsis. Given that the clearance of lactate is dependent on the hepatic and renal processes, this suggests that an increased serum lactate level is a sign of organ malfunction (Vijayan, et al., 2017).

The liver creates C-reactive protein (CRP), an acute phase reactant. Normal plasma concentrations of CRP are below ten mg/l, but they can rise significantly in response to trauma, inflammation, or other

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