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PROCEEDINGS ABSTRACT

Pathophysiological mechanisms and emerging pharmacological strategies in sepsis-associated multiple organ dysfunction: a review

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Sepsis is a life-threatening syndrome that arises when the host response to an infection becomes dysregulated and injurious rather than protective. Instead of containing the infection, the immune, vascular, and metabolic responses begin to damage host tissues, leading to progressive dysfunction in multiple organs. This process remains a major cause of death in critically ill patients despite advances in intensive care practice, antimicrobial therapy, and organ support. The burden of sepsis is particularly high in patients with a delayed diagnosis, severe comorbidities, advanced age, or a persistent shock, and mortality rises markedly once failure develops in more than one organ system. Its clinical course is often heterogeneous, which partly explains why many promising interventions have not translated successfully from experimental models into routine clinical care across diverse patient populations. The biological basis of the sepsis-associated multiple organ dysfunction is highly complex. It involves an early surge of inflammatory mediators triggered by both pathogen- and damage-associated molecular patterns, that is followed by immune exhaustion or immunosuppression. Endothelial injury, microcirculatory failure, disturbed coagulation, mitochondrial dysfunction, and an altered cellular metabolism contribute to the impaired tissue oxygen use and the progressive cellular damage. These changes affect the liver, the lungs, the heart, the kidneys, and other organs through overlapping mechanisms rather than through isolated pathways. Hepatic dysfunction may reflect cytokine-driven injury, cholestasis, and impaired perfusion; cardiac dysfunction is linked to inflammatory signalling, mitochondrial injury, and reduced contractile performance; acute lung injury develops through an alveolar–capillary barrier disruption and neutrophil recruitment; acute kidney injury involves inflammation, microvascular disturbance, oxidative stress, and tubular dysfunction. Due to the fact that the aforementioned organ-specific manifestations share common pathophysiological networks, sepsis should be viewed as a systemic disorder rather than a series of distinct complications. Current management still largely depends on the rapid recognition, the timely (broad-spectrum) antimicrobial therapy, source control, haemodynamic stabilization, and intense organ-supportive care. Although these measures are essential, they do not directly reverse any of the molecular disturbances that drive organ injury. For this reason, increasing attention has turned toward mechanism-based therapies that can interrupt disease progression. In fact, recent preclinical studies have explored several pharmacological agents that exhibit antioxidant, anti-inflammatory, immunomodulatory, and even mitochondria-protective

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properties, including melatonin, metformin, and palmitoylethanolamide. Significant interest has recently also grown in therapies aiming at restoring the metabolic balance, improving microcirculatory flow, modulating key inflammatory mediators, and protecting cellular bioenergetics during severe infection. This review summarizes the main pathophysiological mechanisms involved in the sepsis-associated multiple organ dysfunction and discusses current and emerging pharmacological strategies in the field. By bringing these interconnected processes together, this review attempts to provide a clearer framework for our understanding of the disease progression and for the identification of more rational therapeutic targets. Such a framework can be of high importance, given that a stronger mechanistic understanding may help us improve treatment selection, optimize the timing of intervention(s), and support better clinical outcomes.

Keywords

mitochondrial dysfunction; multiple organ failure; oxidative stress; pharmacotherapy; sepsis

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Conflicts of interest statement

None to declare.

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