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SIMILARITIES AND DIVERGENCES IN SEPSIS INDUCED BY GRAM-POSITIVE AND GRAM-NEGATIVE BACTEREMIA: A COMPARATIVE ANALYSIS

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Introduction: Sepsis represents a life-threatening organ dysfunction precipitated by a dysregulated host immune response to an infectious insult. The etiologic agents encompass both Gram-positive (G^+) and Gram-negative (G^-) bacteria. Although syndromes induced by G^+ and G^- bacteremia are frequently conceptualized as identical nosological entities in clinical practice—warranting a uniform empirical therapeutic regimen during the hyperacute phase—contemporary molecular and epidemiological investigations underscore their fundamental structural and immunopathological heterogeneity. Grounded in recent evidence-based clinical data, this study aims to systemize the pathophysiological similarities and distinct divergences between these two pathogenic cohorts.

Methodology: A retrospective analysis was conducted utilizing datasets from multicenter clinical trials and large-scale meta-analyses. The evaluation focused on the inflammatory mediator profiles, hemodynamic instability, the severity of organ dysfunction, and clinical outcomes characteristic of both Gram-positive and Gram-negative sepsis.

Results (Divergences): Initiation of the pathophysiological cascade demonstrates strict receptor specificity. The cell wall component of G^- pathogens, lipopolysaccharide (LPS/endotoxin), interacts directly with Toll-like Receptor 4 (TLR4), provoking markedly elevated serum concentrations of acute-phase reactants—specifically procalcitonin (PCT) and C-reactive protein (CRP)—compared to G^+ encounters. Regarding clinical severity, G^- bacteremia is independently associated with a significantly higher risk of developing severe sepsis and septic shock (OR: 1.73). Anatomically, the primary infectious foci differ: G^- strains are predominantly implicated in pneumonia, intra-abdominal, and urogenital infections, whereas G^+ pathogens correlate heavily with cutaneous, soft tissue, and intravascular catheter-related infections (mediated via TLR2/TLR6 activation or superantigens).

Results (Similarities): Despite the divergence in the initial magnitude of the immunological cascade, large-scale clinical validation confirms that patient-centered definitive outcomes demonstrate no statistically significant difference ($p > 0.05$). The composite 28-day mortality rate

is identical between the two groups (OR: 0.95). Furthermore, the overall hospital and Intensive Care Unit (ICU) length of stay, leukocyte profile dynamics, and the relative risks of developing systemic coagulopathy (characterized by D-dimer elevations, APTT prolongation, and thrombocytopenia) remain uniform across both cohorts.

Conclusion: Although Gram-negative sepsis is characterized by a more aggressive initial clinico-laboratory presentation and profound endotoxemia, the long-term prognosis and lethal outcomes are proportional to Gram-positive sepsis. These findings highlight the critical imperative for early multiplex molecular diagnostics and the timely optimization of etiopathogen-targeted antimicrobial therapy.

Keywords: Bacteremia, Infectious Focus, Lipopolysaccharide, Procalcitonin, Sepsis, Clinical Outcome