THE ROLE OF ENDOTOXICOSIS IN THE PATHOGENESIS OF LOBAR PNEUMONIA AND APPROACHES TO ITS CORRECTION: A NARRATIVE REVIEW

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Abstract. Lobar pneumonia is an acute inflammatory disease of the pulmonary parenchyma that can be accompanied by systemic inflammation and, in severe cases, sepsis and organ dysfunction. Endotoxicosis is used to describe disturbances associated with microbial products, inflammatory mediators, tissue injury, and altered metabolism. To review the role of endotoxicosis-associated mechanisms in lobar pneumonia and summarize diagnostic and evidence-based therapeutic approaches.

Keywords: lobar pneumonia, community-acquired pneumonia, endotoxicosis, endotoxemia, systemic inflammation, biomarkers, sepsis, supportive treatment.

Introduction. Pneumonia is an acute infection of the lung parenchyma and remains an important cause of infectious morbidity and mortality worldwide. Lobar pneumonia describes a pattern of inflammation affecting a substantial portion of a pulmonary lobe and is often associated with bacterial infection. The clinical course ranges from localized disease to respiratory failure, sepsis, and multiple-organ dysfunction [1]. Disease severity depends on the causative pathogen, host immune status, comorbidities, and the magnitude of the inflammatory response. Although inflammation is necessary for pathogen elimination, excessive or dysregulated

immune activation can damage pulmonary tissue and contribute to systemic complications [1,2]. Lipopolysaccharide (LPS), a component of the outer membrane of Gram-negative bacteria, is a major bacterial endotoxin. Pneumonia can produce systemic inflammation without demonstrable circulating endotoxin; therefore, endotoxicosis should not be assumed to be a universal or independently measurable feature of pneumonia [2,3].

Endotoxin and coagulation. LPS from Gram-negative bacteria can activate innate immune pathways through interactions involving LPS-binding protein, CD14, and Toll-like receptor 4. However, Gram-positive organisms, including Streptococcus pneumoniae, do not contain LPS but can still provoke strong inflammatory responses through other microbial structures [1,2].

Cangemi and colleagues studied 104 patients with community-acquired pneumonia and reported that low-grade endotoxemia was associated with coagulation activation during the early phase of illness [3]. This observational association supports further investigation but does not establish that endotoxin is the sole cause of coagulation abnormalities or that endotoxin removal improves outcomes.

Metabolic disturbances and organ dysfunction. Systemic inflammation may be accompanied by altered microcirculation, cellular energy metabolism, and elimination of metabolic products. In severe infection, these disturbances can contribute to renal dysfunction, hepatic abnormalities, altered mental status, and cardiovascular instability [4,5]. Elevated urea or creatinine is not specific for endotoxicosis and must be interpreted in relation to hydration, renal perfusion, pre-existing disease, and medication exposure.

Diagnostic Assessment. Clinical assessment should include respiratory rate, oxygen saturation, blood pressure, heart rate, temperature, mental status, and urine output. The Pneumonia Severity Index and CURB-65 can assist risk stratification in adults

with community-acquired pneumonia. When sepsis is suspected, clinicians should promptly assess organ dysfunction and initiate appropriate management [4-6].

A complete blood count may reveal leukocytosis, neutrophilia, leukopenia, or thrombocytopenia. C-reactive protein reflects acute-phase inflammation but is not specific for endotoxin. Procalcitonin may support antibiotic stewardship in selected settings, but it should not be used alone to decide whether to initiate antibiotics. Serum creatinine, urea, liver enzymes, bilirubin, platelet count, and coagulation tests may be indicated to assess organ dysfunction and complications.

Antimicrobial therapy and supportive care. The main treatment for bacterial lobar pneumonia is appropriate antimicrobial therapy. Initial selection should consider disease severity, likely pathogens, local resistance patterns, microbiological results, comorbidities, and allergies. The ATS/IDSA guideline provides evidence-based recommendations for adult community-acquired pneumonia, including empirical treatment and reassessment when microbiological results become available [4]. Antibiotics should be prescribed according to clinical indications, not solely because inflammatory markers are elevated.Oxygen should be provided when indicated, with escalation of respiratory support according to clinical status. Fluid management must be individualized: intravenous fluids may be necessary in dehydration or impaired perfusion, while excessive administration can worsen pulmonary edema and oxygenation. Patients with septic shock may require vasopressors and intensive care in accordance with sepsis guidance [5].

Anti-inflammatory and detoxification interventions. Inflammation is essential for host defense, and indiscriminate suppression may be harmful. Corticosteroids should not be given routinely to every patient solely to reduce inflammatory markers; their use depends on disease severity, specific indications, and current guidance. Severe community-acquired pneumonia requires

individualized decisions regarding adjunctive treatment and management of complications [7].

Enterosorption, hemoperfusion, and other extracorporeal techniques have been proposed for conditions described as endogenous intoxication. Such procedures must not replace antimicrobial therapy, oxygenation, hemodynamic support, or treatment of organ dysfunction. Kidney replacement therapy may be appropriate for established indications such as severe kidney failure, but pneumonia alone is not an indication for dialysis or hemoperfusion.

Discussion. The literature indicates that systemic inflammation in pneumonia results from complex interactions between microorganisms and host responses. Endotoxins may participate, particularly in Gram-negative infections, but their contribution varies and cannot explain all severe cases [1-3].

Similarly, renal and hepatic abnormalities indicate possible organ dysfunction without independently establishing endotoxicosis. International guidance emphasizes appropriate antimicrobial treatment and early recognition and management of complications [4,5,7].

Conclusion. Endotoxicosis-associated mechanisms may contribute to systemic manifestations of lobar pneumonia through microbial products, inflammatory mediators, endothelial dysfunction, coagulation activation, and metabolic disturbances. Treatment should prioritize appropriate antimicrobial therapy, oxygenation, individualized fluid management, and timely management of complications. Routine detoxification or extracorporeal blood purification solely for presumed endotoxicosis cannot be recommended without an established clinical indication.

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