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# CORTICOSTEROIDS IN SEPSIS-ASSOCIATED ACUTE RESPIRATORY DISTRESS SYNDROME

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Sepsis-associated acute respiratory distress syndrome (SA-ARDS) is a severe pulmonary complication of sepsis characterized by dysregulated systemic and pulmonary inflammation, disruption of the endothelial and alveolar epithelial barriers, increased pulmonary capillary permeability, and diffuse alveolar damage. These pathophysiological processes contribute to non-cardiogenic pulmonary edema, severe impairment of gas exchange, and hypoxemia.

Given their potent anti-inflammatory and immunomodulatory effects, corticosteroids have a strong pathophysiological rationale in the management of SA-ARDS. Through binding to the glucocorticoid receptor, corticosteroids regulate gene transcription, suppress NF-κB- and AP-1-mediated pro-inflammatory signaling pathways, and reduce the production of inflammatory mediators. These effects may attenuate alveolar–capillary barrier injury, improve oxygenation and lung compliance, and shorten the duration of mechanical ventilation. However, physiological improvement does not necessarily translate into a survival benefit.

Corticosteroids differ substantially in their glucocorticoid and mineralocorticoid activity, duration of action, and clinical context of use. Hydrocortisone is commonly administered in septic shock to support hemodynamic stabilization; methylprednisolone has been extensively investigated in early and persistent ARDS because of its potent glucocorticoid and anti-inflammatory activity; and dexamethasone, with its high glucocorticoid potency, prolonged biological activity, and minimal mineralocorticoid effect, has demonstrated favorable outcomes in selected patients with moderate-to-severe ARDS. These pharmacological and clinical differences suggest that corticosteroids should not necessarily be considered interchangeable, and assessment of corticosteroid therapy should account for the specific agent, dose, timing, duration, and clinical context.

Despite a clear biological rationale, clinical evidence specific to SA-ARDS remains limited. Heterogeneity among existing studies precludes definitive conclusions regarding the optimal corticosteroid agent, dose, timing of initiation, and duration of treatment. Future research should focus on well-designed studies specifically targeting SA-ARDS and on developing individualized corticosteroid strategies guided by patients' clinical and biological characteristics.

**Keywords:** sepsis-associated ARDS; corticosteroids; hydrocortisone; methylprednisolone; dexamethasone; alveolar–capillary barrier