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The Kidney in Dialogue: Understanding AKI-induced Inter-organ Crosstalk and Systemic Consequences

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Acute kidney injury (AKI) is highly prevalent in critically ill patients with high in-hospital mortality and morbidity. However, a significant proportion of the AKI-associated mortality cannot be explained simply by loss of kidney function or by complications occurring during AKI treatment. Since the late 1990s, numerous experimental studies have been conducted to elucidate inter-organ crosstalk in AKI. Lung-kidney interaction was first demonstrated using ischemia–reperfusion injury models; subsequently, crosstalk between the kidney and other organs, including the heart, liver, brain, gut, and spleen was also identified. These mechanisms include inflammatory cytokines, oxidative stress, and immune cell responses. Patients with AKI frequently present with multisystem organ dysfunction rather than isolated renal injury. This observation supports the hypothesis that AKI triggers systemic inflammatory and immune responses, which in turn lead to remote organ damage. More recently, the application of organ network analysis has provided a novel framework for understanding these complex multi-organ interactions. Organ network analysis evaluates the interconnectivity between multiple organs, where highly connected organs are identified as functional clusters. In this framework, a higher number of clusters reflects a disrupted and fragmented network, whereas fewer clusters signify a robustly integrated system. A recent study applied this network analysis to data from critically ill adult patients, suggesting that organ-network disruption is associated with mortality; however, data supporting this concept have been limited to date. In this section, we will focus on the AKI-induced organ crosstalk and subsequent systemic consequences.

Keywords: acute kidney injury, interorgan crosstalk