AKI

급성 신손상의 병인기전

전남대학교 의과대학

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Pathogenesis of Acute Kidney Injury

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The pathogenesis of acute kidney injury (AKI) is complex. whereas initiating events may be dissimilar such as vasoconstriction, leukostasis, vascular congestion, cell death, and abnormal immune modulators and growth factors, subsequent injury responses may involve similar pathways. Pathogenic factors have formed the basis of rational therapeutic interventions. However, many targeted clinical therapies have failed, are inconclusive, or have yet to be tested. Given the complexity of the pathogenesis of AKI, it may be naive to expect that one therapeutic intervention would have success. Some examples of detrimental processes that can be blocked in preclinical models to improve kidney function and survival are apoptotic cell death in tubular epithelial cells, complement-mediated immune system activation, and impairment of cellular homeostasis and metabolism. Modalities with the potential to decrease morbidity and mortality in patients with AKI include vasodilators, growth factors, anti-inflammatory agents, and cell-based therapies. Therapies may need to simultaneously target multiple pathways to achieve success. Pharmacologic agents that target these diverse pathways are being used clinically for other indications. Using combinatorial approaches in future clinical trials may improve our ability to prevent and treat AKI.

Key Words: Acute kidney injury, Apoptosis, Inflammation, Acute tubular necrosis