Acute Kidney Injury

Pathophysiology of AKI

Kitae Bang

Eulji University school of medicine

Acute kidney injury (AKI) is the leading cause of nephrology consultation and is associated with high mortality rates. The primary causes of AKI include ischemia, hypoxia or nephrotoxicity. An underlying feature is a rapid decline in GFR usually associated with decreases in renal blood flow. Inflammation represents an important additional component of AKI leading to the extension phase of injury, which may be associated with insensitivity to vasodilator therapy. It is suggested that targeting the extension phase represents an area potential of treatment with the greatest possible impact. The underlying basis of renal injury appears to be impaired energetics of the highly metabolically active nephron segments (i.e., proximal tubules and thick ascending limb) in the renal outer medulla, which can trigger conversion from transient hypoxia to intrinsic renal failure. Injury to kidney cells can be lethal or sublethal. Sublethal injury represents an important component in AKI, as it may profoundly influence GFR and renal blood flow. The nature of the recovery response is mediated by the degree to which sublethal cells can restore normal function and promote regeneration. The successful recovery from AKI depends on the degree to which these repair processes ensue and these may be compromised in elderly or CKD patients. Recent data suggest that AKI represents a potential link to CKD in surviving patients.