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AKI to CKD: pathophysiology and management

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Acute kidney injury (AKI) and chronic kidney disease (CKD) have been regarded as two distinct syndromes. However, the close relationship of AKI and CKD as interconnected syndromes is now widely accepted. Although AKI is reversible in many cases, a substantial portion of AKI progresses to CKD. Increasing incidence and lack of early diagnostic tools as well as effective therapeutic options for AKI are still unresolved issues in nephrology. The main pathologic features of AKI to CKD progression are tubular atrophy and interstitial fibrosis caused by maladaptive repair process of AKI. The balance between repair and regeneration pathways (e.g. apoptosis, dedifferentiation, and proinflammatory and anti-inflammatory, epigenetic, and profibrotic mediators) is the cornerstone of pathophysiologic mechanisms which determine the direction of the repair process: adaptive repair inducing recovery or maladaptive repair leading to CKD.

Attenuating the progression of AKI to CKD can be summarized depending on the degree and stage of AKI. Proper initiation and discontinuation of renal replacement therapy (RRT) are crucial for severe AKI. Conservative management strategies to reduce metabolic demand and avoiding potential nephrotoxic agents are important for facilitating adaptive repair during the recovery phase of AKI. Appropriate management of intravascular volume, hypertension, anemia, metabolic acidosis, and electrolyte disturbances are required to delay the progression of AKI to CKD.

In this session, we will review 1) the pathophysiology of AKI to CKD transition and 2) important management points to minimize the risk of AKI to CKD transition.