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New coming bridge between AKI and CKD: Acute kidney disease

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Acute kidney injury (AKI) occurs in 5–10% of hospitalized patients and in more than 30% of critically ill patients. The increase in annual AKI incidence results in interrupted treatment for comorbidities, an increased risk of acute renal replacement therapy, longer hospital stays, an increased burden of medical costs, and increased mortality rates (50-80%). Furthermore, AKI patients show higher risks of progression to CKD or end-stage renal disease; the risk is greater than that of stage 3 CKD patients who did not undergo an AKI episode, and the risk is exacerbated when AKI patients experience more episodes and more severe injury. These issues are important, both in terms of medical and socioeconomic aspects.

The mechanisms by which AKI induces renal fibrosis are well known from several experimental studies; a loss of renal vasculature, tubular atrophy or dilatation, and alterations in glomeruli occur after approximately 40 weeks of ischemia-reperfusion injury. These changes lead to progressive renal fibrosis and permanent renal functional loss. Additionally, AKI causes endothelial dysfunction and increases the levels of various cytokines, including tumor necrosis factor-α, interleukin-1, and interleukin-6, which are reported to injure distant organs such as the heart, lungs, and liver. Therefore, AKI may be an independent risk factor for mortality and progression to CKD according to these hypotheses.

Figure 1. Clinical impacts of acute kidney injury

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