

Transition from AKI to CKD. The Modifiers and Prevention in Clinical and Experimental Studies.

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Acute kidney injury (AKI) complicates up to 15% of hospitalizations and can reach up to 50–60% in critically ill patients. AKI-related mortality is 23%, which increases to 49.4% in those requiring renal replacement therapy. The 16th Acute Disease Quality Initiative (ADQI) meeting defined acute kidney disease (AKD) as acute or subacute damage and/or loss of kidney function lasting 7–90 days following an AKI-initiating event. Many patients experience significant irreversible nephron loss and subsequently develop chronic kidney disease (CKD). Indeed, 20–50% of AKI patients develop progressive CKD.

Renal recovery after AKI is a complex process and it is highly dependent on baseline kidney function, severity, duration and etiology. The incidence of renal recovery can range from 0% to 90% considering all stages of AKI severity, but from 0% to 40% in cases of dialysis requiring AKI. The pro-fibrotic and pro-inflammatory pathways can result in maladaptive repair and transition to CKD after AKI. It is crucial to stop regarding AKI as a short-term reversible condition and to raise awareness on the long-term complications, such as progression to CKD, increased cardiovascular events and mortality. There is no effective or timely treatment to stop the progression. Therefore, it is essential to find a treatment that decreases inflammation and prevents fibrosis. Currently, regenerative medicine, is the most promising area.

We will present our preclinical study on the influence of human placental stem cells in the prevention of AKI progression to CKD in the experimental model.