【Outstanding Academic Research Meeting I-2】

Inflammation modulation as a novel therapeutic strategy for acute kidney injury

調節發炎作為急性腎損傷的新型治療策略

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Acute kidney injury (AKI) is defined as a rapid decline in kidney function caused by ischemia, intoxication, or infection. Tubular epithelial cells can synthesize and secrete a variety of inflammatory and fibrotic proteins in response to AKI injury.

On the other hand, the upregulation of inflammation increases the risk of AKI being converted into chronic kidney disease (CKD). Therefore, the management of inflammation may be crucial to delaying the development of kidney disease. Given that the mechanisms underlying the progression of AKI to CKD remain incompletely understood, and specific therapeutic interventions remain elusive. Here, we focus on anti-inflammatory strategies, such as hydralazine and inflammatory chemokines as treatments for AKI and AKI-to-CKD. Future studies may be worthy to validate the potential impacts and long-term safety of specific anti-inflammatory strategies for AKI related kidney diseases.