Mediators of Inflammation

Hindawi

Special Issue on

Progression from Acute Kidney Injury to Chronic Kidney Disease: Role of Inflammatory Mediators

CALL FOR PAPERS

Acute kidney injury (AKI) causes at least 2 million deaths worldwide each year, and its incidence rate is increasing. Evidence suggests that patients who have recovered from AKI have a 25% increased risk for developing progressive chronic kidney disease (CKD) and even end-stage renal disease (ESRD). In addition, these patients demonstrate a 50% increase in mortality after 10 years. These clinical data demonstrate that AKI closely correlates with poor long-term patient outcomes and later CKD development. However, to date, few mechanisms and individualized molecules have been identified, which affect the progression from AKI to CKD; therefore, this is an important research field that may lead to the development of therapies which prevent the progression to ESRD.

Recent studies have described novel mechanisms underlying AKI, indicating that additional cell death pathways of regulated necrosis, such as pyroptosis and ferroptosis, can lead to inflammation. Moreover, increasing data suggests that inflammation plays an important role in the acute and recovery phase following the initial insult. Among the possible factors implicated in this process associated with AKI damage, the role of inflammatory mediators and their relation to monocyte/macrophage and T cells phenotypic balance have emerged as factors of potential interest.

Although clinical observations and experimental studies describe a clear association between AKI and CKD, they do not establish a causal link among them. This special issues therefore aims to collect original research and review articles concerning how inflammation may affect the progression from AKI to CKD, an important research area that may lead to therapies to prevent further progression to ESRD.

Potential topics include but are not limited to the following:

- Role of inflammatory mediators of intrinsic kidney cells, macrophages, and T cell subsets in the inflammatory process associated with AKI and AKI-to-CKD transition
- Inflammatory mediators inducing signaling pathways and immune cells interactions engaged in AKI-associated inflammation as potential therapeutic targets
- Importance of inflammatory mediators in cell death subroutines contributing to AKI and AKI-to-CKD progression
- Experimental models to study the inflammatory mediators implicated in AKI and AKI-to-CKD progression
- Breakthroughs in the current understanding of the role of inflammatory mediators in progression from AKI to CKD

Authors can submit their manuscripts through the Manuscript Tracking System at https://mts.hindawi.com/submit/journals/mi/pfac/.

Papers are published upon acceptance, regardless of the Special Issue publication date.

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