

Immune Cells in Acute Kidney Injury

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Acute kidney injury (AKI) prolongs hospital stay and increases mortality in various clinical settings. Ischemia–reperfusion injury (IRI), nephrotoxic agents and infection leading to sepsis are among the major causes of AKI. Inflammatory responses substantially contribute to the overall renal damage in AKI. Both innate and adaptive immune systems are involved in the inflammatory process occurring in post–ischemic AKI. Proinflammatory damage–associated molecular patterns, hypoxia–inducible factors, adhesion molecules, dysfunction of the renal vascular endothelium, chemokines, cytokines and Toll–like receptors are involved in the activation and recruitment of immune cells into injured kidneys. Immune cells of both the innate and adaptive immune systems, such as neutrophils, dendritic cells, macrophages and lymphocytes contribute to the pathogenesis of renal injury after IRI, and some of their subpopulations also participate in the repair process. These immune cells are also involved in the pathogenesis of nephrotoxic AKI. Experimental studies of immune cells in AKI have resulted in improved understanding of the immune mechanisms underlying AKI and will be the foundation for development of novel diagnostic and therapeutic targets. This lecture will review what is currently known about the function of the immune system in the pathogenesis and repair of ischemic and nephrotoxic AKI. There will be focus on the role of lymphocytes, and an introduction into the discovery of the novel double negative T cell found in kidney.

Select references

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