Inflammation in acute kidney injury

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Inflammation is essential for body to eradicate microbial pathogens and also to mediate repair from tissue injury. However, maladaptive, excessive inflammation might contribute to further tissue injury, organ dysfunction and also fibrosis. Following diverse insults to kidney, sterile inflammation occurs and for the last several decades, much knowledge about the basic mechanisms of inflammation have been clarified mostly by using small animal models. Infiltrating leukocytes including neutrophils, M1 macrophages, CD4 T cells, B cells and invariant natural killer T cells as well as kidney resident dendritic cells play a critical role by releasing many proinflammatory mediators and contribute to the pathogenesis of AKI. In contrast, inflammation by M2 macrophages or regulatory T cells during the recovery phase has recently been demonstrated to be important in reparative mechanisms. In addition to leukocytes, kidney endothelial cells also play a role in inflammation by increasing adhesion molecule expression and vascular permeability. Tubular epithelial cells also participate in inflammation by upregulating toll like receptor expression, and also by increasing the production of proinflammatory cytokines and chemokines. However, despite major advances in understanding of precise molecular mechanisms of inflammation, little progress in developing clinically available therapies targeting this maladaptive inflammation has been made. This review summarizes the recent update in mechanisms of inflammation and also suggests future basic and clinical research directions toward better clinical translation.