Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care
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The Role of Annexin A2 in Ischemia/Reperfusion-Induced Acute Kidney Injury and Fibrosis

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Objectives : Renal ischemia/reperfusion injury (IRI) is a major cause of acute kidney injury (AKI). Evidence suggests that ANXA2 is involved in inflammatory process and cell death. This study aims to define the role and molecular mechanisms of ANXA2 in renal IRI, clarify its regulatory function in IRI-induced AKI (IRI-AKI), and contribute new insights for therapeutic strategies against IRI-AKI.
Methods : ANXA2 expression in mouse kidneys after ischemia-reperfusion injury (IRI) was assessed by Western blot and immunohistochemistry (IHC). Kidney injury in Anxa2-KO and wild-type (WT) mice post-IRI was evaluated via Hematoxylin-eosin staining, periodic acid-schiff staining, KIM-1 immunofluorescence (IF), and fibrosis via Masson's trichrome and α-SMA IHC. Immune cell infiltration was quantified by flow cytometry and localized by neutrophil/macrophage IHC. Inflammatory cytokines in the medulla were measured within 24 hours. Phospho-MLKL (pMLKL) IF assessed necroptosis in tubular epithelial cells.
Results : Significant AKI was observed only after 30 minutes of ischemia, with concurrent upregulation of ANXA2 at both transcriptional and protein levels. IHC indicated that ANXA2 upregulation initiated in the medulla and extended to the cortex over time. Tubular injury scores in the medulla were significantly higher in Anxa2-KO mice compared to WT mice at day 1 post-IRI, while cortical differences emerged only at day 7. More severe injury to medullary tubular cells and fibrosis in Anxa2-KO mice. Inflammatory cytokines (TNF-α, IL-1β, IL-6) were elevated in Anxa2-KO kidneys at day 1, particularly in the outer medulla, alongside increased pMLKL.
Conclusions : ANXA2 exerts a protective role on medullary tubules early in renal IRI and negatively regulates innate immune cell infiltration into injured areas. Moreover, ANXA2 mitigates long-term renal fibrosis following IRI. Mechanistically, ANXA2 deficiency aggravates IRI-AKI by activating necroptosis and amplifying the inflammatory response.

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