THE KOREAN SOCIETY OF NEPHROLOGY
APCN & KSN 2024
June 13(Thu) - 16(Sun), 2024 Coex, Seoul, Korea
Promoting Sustainable Kidney Health: The Asia-Pacific and Beyond

Submission No. : KRFP-0011
Session Title : KSN Research Fund Project
Session Topic : -
Date & Time, Place : June 14 (Fri) / 08:30-10:00 / Room 4 (201)

Role of B cell metabolism in ischemic acute kidney injury repair

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Acute kidney injury (AKI) is a critical medical problem in both native kidneys and kidney transplants, affecting a number of patients globally without definitive treatment. B cells play an important role in the pathogenesis of AKI and repair but are relatively less understood than T cells. Metabolic reprogramming of lymphocytes regulates their function, is a rapidly emerging field, and has not been studied in detail in the context of AKI. Thus, we aimed to elucidate the dynamics of B cell metabolism in ischemic AKI. We induced ischemia-reperfusion injury in C57/B6 mice. B cells were isolated from postischemic kidneys at different time points and analyzed by a flow cytometry-based immune-metabolic assay with interrogating metabolic programs. B cells from postischemic kidneys showed reduced expression of glycolysis and oxidative phosphorylation related machineries early after injury, whereas B cells during late recovery period showed comparable expression of those pathways to steady-state kidneys. B cells from postischemic kidneys during the late recovery period exhibited upregulation of fatty acid oxidation and mTOR activities. Splenic B cells after ischemic AKI showed increased mTOR activity as well. In conclusion, B cells undergo distinct metabolic reprogramming during ischemic AKI. Reconstitution of B cell function by targeting the B cell metabolic pathway could be a promising therapeutic approach for AKI.

Keywords: acute kidney injury, lymphocyte, B cell, repair, metabolism