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# Persistent infiltration of M2 macrophages in human acute kidney injury predicts poor renal outcomes

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**Objectives:** Macrophages are important players in the development and recovery of ischemic AKI, but the persistent imbalance of M1/M2 macrophages may lead to progression to CKD. The role of macrophages has been elucidated in animal experiments, but not in human AKI. Therefore, we evaluated the role of macrophages in patients with histologic AKI.

**Methods:**

A total of 72 patients with histologic acute tubular necrosis from 2007 to 2017 were included. Kidneys were immunostained with anti-CD68 and anti-CD163 as macrophage and M2 markers, respectively.

**Results:**

Patients included 29 AKI of native kidneys and 43 AKI of deceased donor. They were followed up for 35.4±30.9 months after biopsy, and renal function was restored to eGFR≥60ml/min in 72.2%. High CD68+ cell infiltration was significantly associated with advanced stage of AKI, but only high CD163+ cell infiltration predicted low eGFR at 1month after biopsy. In subgroup analysis, high CD163+ cell infiltration in native AKI was significantly associated with poor renal function at 1month and non-recovery to eGFR≥60ml/min. However, in deceased donor AKI, CD163+ cell infiltration was only associated with delayed graft function. When the patients were divided into two groups according to biopsy time (early≤2days and late>2days after peak creatinine), in patients with late biopsy, high CD163+ cell infiltration predicted non-recovery, but not in patients with early biopsy. When analyzed according to the AKI stage, in 2 or 3 stage AKI patients, the high infiltration of CD163+ cells was not only associated with a poor renal function at 1 month, but also independently predict poor 3month eGFR after biopsy.

**Conclusions:** In conclusion, renal CD163+ cell was predictive of poor renal outcomes especially in native AKI, delayed biopsy or severe AKI patients. Although M2 macrophages are known to be indispensable for short-term recovery, their persistent infiltration could be associated with poor renal recovery and CKD progression in human AKI.