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Biphasic Protective Effects of Macrophage Migration Inhibitory Factor in Ischemia/Reperfusion-Induced Acute Kidney Injury

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Objectives : Ferroptosis is involved with occurrence and progress of renal ischemia/reperfusion (I/R) injury. Macrophage migration inhibitory factor (MIF) is a pleiotropic cytokine, which is closely associated with kidney diseases. However, the underlying mechanisms between MIF and ferroptosis in acute kidney injury (AKI) still remain unclear. In contrast to conventional strategies, we further investigated dynamic expression and role of MIF in both ischemic and reperfusion phase.
Methods : In the present study, we established hypoxia/reoxygenation (H/R) and I/R induced AKI models, verified ferroptosis by detecting pro-ferroptotic changes and associated indictors and used si-RNA or Adeno-Associated virus (AAV) to knock down the expression of MIF, observing subsequent changes of ferroptosis.
Results : The results showed that treatment of recombinant MIF reduced lipid ROS generation and MDA levels, upregulating glutathione (GSH) levels, with increasing AMP-activated protein kinase (AMPK) phosphorylation. While the deficiency of MIF blunted its protective effect on renal tubular epithelial cells, which demonstrated that MIF ameliorated ferroptosis injury via activating AMPK pathway in ischemia stage and increasing GSH content in reperfusion stage.
Conclusions : In conclusion, our results suggested a novel nephroprotective role of MIF on inhibiting ferroptosis and provide new insight into the prevention and treatment of AKI.

Figure1.png