中文題目：生物標記可溶性調節鐵調素在急性腎損傷期間促進鐵沉積

英文題目：Soluble hemojuvelin, a novel biomarker promotes iron deposition during acute kidney injury

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Backgrounds：Free iron plays an important role in the pathogenesis of acute kidney injury (AKI) via the formation of hydroxyl radicals. Systemic iron homeostasis is controlled by the hemojuvelin/hepcidin axis ratio in the liver, but less is known about this process in AKI.

Material and results：By proteomics, we have identifed a 42 kDa soluble hemojuvelin (sHJV), a proteolytic form of the 50 kDa processed by furin protease from membrane-bound hemojuvelin (mHJV), was detected in the urine during post cardiac surgery AKI. Urinary HJV levels could be used to specifically predict AKI. Furthermore, the biopsies from human and mouse specimens with AKI confirm that extensive HJV is expressed in the renal tubules. In the human renal proximal tubule cells (HK-2), iron overload induces the expression of HJV and results in iron deposition. Recombinant HJV and furin inhibitor up-regulate the expression of kidney-protective heme oxygenase-1. The furin inhibitor decrease the generation of sHJV under iron overload and prevent iron deposition in HK-2 cells. Furin inhibitor could also reduce renal tubule apoptosis, prevent the accumulation of iron in the kidney and further ameliorate ischemic-reperfusion injury during AKI.

Conclusion：Our findings link HJV inextricably with renal iron homeostasis for the first time, add new significance to early predict AKI, and identify novel therapeutic targets to reduce the severity of AKI by furin inhibitor.

Key words: Acute on chronic kidney injury, hemojuvelin, biomarker, iron