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Potential biomarkers of vascular calcification in ferroptosis-related chronic kidney disease

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Objectives: Chronic kidney disease (CKD) is an increasingly serious health problem in the world. It is estimated that 9.1% patients suffer from CKD worldwide. Cardiovascular calcification is usually occurred in patients with CKD and significantly increases all-cause and cardiovascular mortality in uremic patients with long-term hemodialysis. KDIGO guidelines also emphasize the clinical importance of vascular calcification (VC). Therefore, it is of great significance to clarify the mechanism of VC in patients with CKD and to find potential therapeutic targets for providing effective clinical treatment. Our previous studies have shown that vascular smooth muscle cells (VSMCs) during vascular calcification associated with ferroptosis. In this study, we explored the mechanism of vascular calcification involved in ferroptosis-related CKD.

Methods: The calcification model was established by vascular smooth muscle cells and the inhibitor of ferroptosis (Ferrostatin-1) was used. On this basis, the proteins related to ferroptosis and calcification were screened by label free quantitative proteomics. On this basis, we selected the potential biomarkers to explore the further mechanism. The expression of proteins was detected by immunohistochemistry and western blot.

Results: Label free showed that 36 proteins involved in both vascular calcification and ferroptosis. Among them, Western blotting showed that Micu2 was significantly lower in calcification models both *in vivo* and *in vitro*, and the expression increased after inhibiting ferroptosis. The expression of Micu2 was significantly reduced in the aorta, abdominal aorta, thoracic artery and renal tissue in CKD animal model.

Conclusions: Micu2 may be a potential biomarker in vascular calcification of chronic kidney disease by regulating ferroptosis.