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Abnormal lipid metabolism in kidney fibrosis models

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Objectives: Under the hypothesis that chronic kidney disease accompanies abnormal lipid metabolism accumulation and metabolic pathway abnormalities in kidney tissues, we investigated the changes in lipid metabolites and metabolic pathways following in kidney fibrosis models.

Methods: Renal lipid metabolomics was assessed by MALDI-imaging mass spectrometry(MALDI-IMS) and LC-MS/MS and the change of renal transcriptomics was RNAseq at 6 weeks of adenin-induced nephropathy and 7 and 14 days of unilateral ureteral obstruction(UUO) mice models.

Results: At 6 weeks in the adenine model, inflammatory cell infiltration and renal fibrosis were characteristic findings.
UUO model also showed characteristic tubular dilatation and kidney fibrosis, especially at 14 days.
In the MALDI-IMS analysis, decreased membrane phospholipids such as phosphatidylcholine were the most important feature of both adenin and UUO models.
LC-MS/MS analysis indicated that typical intrarenal lipid change in kidney fibrosis was an increase of cholesterol ester(CE) in both adenin and UUO models. Interestingly, the main fatty acids of the increased CE were polyunsaturated fatty acids (PUFAs) including arachidonic acid.
Intrarenal transcript RNAseq analysis showed that activation of the inflammatory and immune cell pathways was the main feature of both renal fibrosis models.
The increased expression of cPLA2 was confirmed by immunohistochemistry and Western blot analysis in both models.

Conclusions: Renal fibrosis is accompanied by the accumulation of CE in the kidney. In particular, the accumulation of PUFA following membrane phospholipid degradation is thought to be a cause of the increased inflammatory response in the kidney disease.