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Acute kidney injury causes the disrupt of lung cell cilia and release of them into alveolar space

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Objectives:

Acute kidney injury (AKI) induces the damage of multiorgan including lung. Recent studies have demonstrated that a disruption of cilium, a rod-like organelle, is associated with organ damage. In this study we investigated an association of cilia with the ischemia-reperfusion (I/R)-induced lung injury.

Methods: C57/BL6 male mice were subjected to 35 min of bilateral renal ischemia-reperfusion (I/R). Bronchoalveolar lavage fluid (BALF) was collected through the trachea after the occlusion of left lobes of lung using phosphate-buffered saline. After collection of BALF, blood, lung, and kidney were harvested. Kidney damage was evaluated by blood urea nitrogen (BUN) and PAS staining. In BALF, cell number and protein concentration were determined. Lung interstitium expansion, leukocyte infiltration, and alveolar damage were evaluated under microscope using lung tissue slide stained by H&E. In BALF, acetylated α tubulin (ac- α -tubulin, a marker protein of cilia), ADP-ribosylation factor-like protein 13B (ARL13B, a marker protein of cilia), and 4-hydroxy-2-nonenal (4-HNE) expressions were determined through western blot analysis. Lung tissue and BALF smear slides were immunostained using anti-ARL13B antibody.

Results: Kidney I/R caused the increase of BUN, reduction of alveolar size, expansion of lung interstitial tissue, and infiltration of leukocytes into the lung. In addition, kidney I/R induced increases of cell number, protein concentration, and 4-HNE, ac- α -tubulin, and ARL13B expressions in the BALF. DNA oxidation was greater in the lung of kidney I/R-induced mice than sham-operated mice. ARL13b-positive signal was lower in the lung of kidney I/R-induced mice than that sham-operated mice.

Conclusions:

These results indicate that AKI causes lung cell cilia fragmentation and lung tissue damage along with the increase of oxidative stress, suggesting that AKI-induced lung injury may be associated with lung cilia damage.