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Molecular Mechanisms in Inter-Organ Crosstalk (Kidney-Lung Axis)

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Acute kidney injury (AKI) occurs frequently in critically ill patients as a part of multiorgan dysfunction/failure, and when coexisting with respiratory failure, it substantially increases mortality. Despite knowledge of lung lesions in patients with uremia as early as the 1930s through autopsy, this kidney-lung interaction has been overlooked in clinical settings due to the complex pathophysiology of multiorgan failure. The recent COVID-19 pandemic has presented an example of the kidney-lung interaction.

Volume overload in kidney failure has been thought to inhibit effective gas exchange in alveoli, causing a deleterious effect on the lung. However, recent studies have revealed that the kidney-to-lung interaction is also caused by remote inflammatory response via immune cells, cytokines, chemokines after AKI; neutrophils and monocytes/macrophages increase in the lung after AKI, and some soluble molecules (e.g., IL-6, IL-8, IL-10, osteopontin, TNF, HMGB1) reportedly play a role in the lung inflammation phenotype after AKI. Additionally, neuro-immune interactions, such as those involving the vagus nerve and spleen, could induce or modify lung injury after AKI. Most of the underlying mechanisms of the kidney-lung interaction remain mostly unknown.

In this session, I will review the observations and research findings regarding the kidney-to-lung interaction in both humans and rodent models, highlighting what is currently known and unknown. I would also like to discuss what should be further investigated to translate current knowledge into improved patient care for those with multiorgan failure.