Abstract Submission No. : 9069
May 28(Sat), 10:40-12:40 KSN-TSN-JSDT Joint Symposium

Prevention and treatment of acute kidney injury from the viewpoint of renal congestion

Masanori Abe
Nihon University School of Medicine, Japan

Acute kidney injury (AKI) is traditionally and almost exclusively attributed to renal hypoperfusion, and it is commonly accepted that causative factors are pre-renal, such as a reduced ejection fraction or absolute or relative hypovolemia acting directly on oxygen transport mechanisms and the renal autoregulation systems, leading to a decrease in glomerular filtration rate (GFR). Indeed, kidney blood flow rate is associated with GFR. When mean arterial pressure (MAP) is >65 mmHg, GFR is maintained normally. However, when MAP falls to <65 mmHg, kidney blood flow rate and GFR rapidly decrease and then urine volume progressively decreases. This leads to oliguria or anuria. Therefore, the kidneys need MAP of at least 65 mmHg. Yet, accumulating clinical and experimental evidence is emerging that paints a more complex clinical picture where AKI is strongly linked to the hemodynamic changes in the renal venous micro- and macrocirculation. Accordingly, the transmission of the increased venous pressure to the renal venous compartment and the consequent increase in renal afterload has a pivotal role in inducing and sustaining kidney damage.

We created a rat model of renal congestion by completely clamping the inferior vena cava (IVC). After 5 min of IVC clamp, the conventional fixation method showed that the tubular lumina was obstructed by swollen cells and ischemia-associated cell debris. However, we could not find the peritubular capillary with this conventional method. Next, an in vivo cryo technique was used to observe the renal congestion. It also showed that the lumina was obstructed by the swollen cells and ischemia-associated cell debris, but the peritubular capillary could be confirmed. After removing the IVC clamp, renal congestion resolved. The swollen cells returned to their cuboidal form, and the cell debris disappeared from the tubular lumina. Finally, the lumina was open. This result indicates that if renal congestion can be promptly and effectively treated, urine volume can be increased and recovery from AKI is possible.

The appropriate combined use of diuretics is important. Tolvaptan, a selective vasopressin type 2 receptor antagonist that increases free water clearance, may be useful for treating renal congestion. Furthermore, removal of blood to the extracorporeal circuit in continuous renal replacement therapy may relieve renal congestion directly. In this talk, I will discuss the prevention and treatment of AKI from the viewpoint of renal congestion.