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**Volume management in patients with AKI and heart failure:
Cardiorenal syndrome**

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To maintain homeostasis of the cardiovascular system, the heart and kidney are bidirectionally influenced, so acute or chronic dysfunction of one organ can cause dysfunction of the other. This is defined as cardiorenal syndrome (CRS). Concurrent dysfunction of heart and kidney adversely affect one another and eventually worsen the patient outcomes through vicious cycle.

Recently, the CRS classification system (type1-5) has been proposed but the underlying pathophysiology is multifactorial, and clinical access is still difficult. In patients with CRS, decreased cardiac output lowers renal perfusion pressure, induces renal ischemia, and increases renin-angiotensin-aldosterone system, sympathetic hypersensitivity and arginine-vasopressin secretion, further worsening cardiac function with systemic fluid retention. Although these hemodynamic changes due to decreased cardiac output are known to be a major mechanism of CRS, given that acute kidney injury is common in heart failure patients with preserved EF, "renal venous congestion" has been suggested as an important pathway to exacerbate CRS. Therefore, diagnosing, monitoring, and controlling venous congestion is important to improve outcomes of CRS. Volume control with diuretics or ultrafiltration is a clinically effective in reducing venous congestion, but there is still insufficient evidence to reduce complications and mortality. Recently, new drugs such as ARNI and SGLT2 inhibitors are emerging as effective drugs for CRS treatment, but more efforts are needed to individualize diagnosis and treatment strategies considering various clinical aspects and complications of CRS.