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An Update on Management of Cardiorenal Syndrome

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Cardiorenal syndrome (CRS) refers to a combination heart and kidney dysfunction. It can be in the form of acute and chronic dysfunction that will affect each other. This condition adversely impacts both organs and contributes to significant clinical outcomes. There is a need to understand the pathophysiology, diagnosis and ultimately therapeutic options for the management of CRS.

The pathophysiology of CRS is complex and believed to be multifactorial. The immune system, the sympathetic nervous system, the renin-angiotensin-aldosterone system and oxidative stress actively play a role in the development of CRS. CRS has been divided into five types (Type I-V) depending on onset and the primary organ involvement. In the setting of pre-existing chronic kidney disease, uraemic molecules have been shown to be key mediators of the syndrome.

Type I : Acute Cardiorenal Syndrome
Type II : Chronic Cardiorenal Syndrome
Type III : Acute Renocardiac Syndrome
Type IV : Chronic Renocardiac Syndrome
Type V : Secondary Cardiorenal Syndrome

Common risk factors for CRS include diabetes mellitus, hypertension, atherosclerotic, anemia and certain medications. There are also many other factors that may not have clear pattern of relationship with CRS. In general, all types of CRS exhibit systemic congestion, changes in blood pressure and increased serum creatinine. Hyperkalemia and diuretic resistance are other associated manifestations.

In the clinical setting, CRS is usually observed in the context of acute decompensated heart failure (ADHF). Determining kidney dysfunction and understanding the significance of elevations and variability of serum creatinine are important in diagnosing kidney injury. Diagnosing CRS in the setting of ADHF, represents a significant clinical challenge for clinicians as decongestive therapy itself may complicate the assessment of renal function. Therefore, certain diagnostic tools may be required to reach at the diagnosis. Imaging modalities such as echocardiography and ultrasound should be used to evaluate the functions of both organs. When available, cardiac and kidney biomarkers will provide additional value to existing clinical assessments and standard laboratory parameters.

CRS management depend on the type and associated risk factors. Understanding of the pathophysiology is essential in making treatment decisions. Diuretic therapy, especially loop diuretics remains the mainstay of the treatment strategy. There are many other therapies that can be effectively used such as vasodilators, inotropic supports, and ultrafiltration therapy in the setting of diuretic resistance. Blockade of the renin-angiotensin-aldosterone system is the cornerstone in the management of chronic CRS while sodium-glucose co-transporter-2 inhibitors (SGLT2i) are the newest anti-diabetic drugs that also exert cardiorenal protection.

The complexity of CRS pathogenesis and the therapeutic challenges in managing CRS require us to continue to search for optimal strategies that can benefit both organs and the most important patient outcomes.