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Crosstalk: Heart and Kidney

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The heart and the kidney are two major organs with numerous interactions. The term cardio-renal syndrome (CRS) has been proposed to describe situations in which failure of one leads to failure of the other. CRS has five types: 1 acute heart failure leading to renal failure, 2 chronic heart failure leading to renal failure, 3 acute renal failure leading to heart failure, 4 chronic kidney disease leading to heart failure and 5 systemic disease leading to simultanenous kidney and heart failure. In the presentation, focus will be given on the two subtypes encountered in the acute setting: CRS-1 and CRS-3.

CRS-1 is common and associated with increased mortality and morbidity. Pathophysiological mechanisms leading to CRS-1 include low cardiac output flow (leading to decreased renal perfusion) and increased venous pressure. Optimization of cardiac output should therefore be the main therapeutic target. ACE-inhibitors should be withheld and re-introduced after the acute illness. Contrast associated procedures should not be withheld. Indeed, contrast-associated nephropathy is increasingly debated. However, iso-osmolar agents should be preferred and low dose administered. The second therapeutic target is decongestion. It is typically achieved with loop diuretics. However, diuretic resistance is common in CRS and might require dose increase or associated with other diuretics. In case of diuretic failure, fluid removal by renal replacement therapy should be considered. Other therapeutics (Tolvaptan, nesiritide, low dose dopamine) have failed to show clinical benefit in randomized controlled trials.

CRS-3 is less common but similarly associated with mortality and morbidity. Acute kidney injury might lead to acute heart failure through metabolic acidosis or uremia associated decrease in contractility, hyperkaliemia triggered arrhythmias and fluid retention. A prototype of CRS-3 is represented by reno-vascular hypertension, in which renal artery stenosis, via an activation of the renin-angiotensin aldosterone system leads to hypertension and heart failure. New biomarkers with possible causative mechanisms have been identified such as Galectin-3. However, to date, no specific therapy for AKI has been shown to be associated with improved outcomes. Early renal replacement therapy should be considered.