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**Original research article**  
**Cardiorenal interactions**

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**ARTICLE INFO**

*Article history:*  
 Received 30 September 2017  
 Received in revised form  
 30 October 2017  
 Accepted 5 November 2017  
 Available online xxx

*Keywords:*  
 Cardiorenal syndrome  
 Chronic heart failure  
 Diabetic kidney disease  
 Renin–angiotensin system  
 Empagliflozin

**ABSTRACT**

Cardiorenal interactions are bidirectional. Renal hypoperfusion in patients with acute or chronic heart disease is associated with increased mortality and increased risk of end-stage renal disease. Heart damage and/or dysfunction in patients with acute and chronic kidney disease has significant negative impact on the patient survival. Awareness and early diagnosis and therapy may help to ameliorate negative consequences of the other organ damage, especially in acute setting. Search for therapeutic interventions aimed at concomitant cardio- and renoprotection is warranted.

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<https://doi.org/10.1016/j.crvasa.2017.12.006>  
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## Introduction

There are bilateral interactions between the heart and the kidney. On one hand, heart failure may result in renal hypoperfusion and decrease of glomerular filtration rate, on the other hand, chronic kidney disease (especially end-stage renal disease) is associated with increased cardiovascular morbidity and mortality.

The term cardiorenal syndrome (CRS) has been recently recommended to cover the ever increasing spectrum of cardiorenal interactions [1] (Table 1).

### Cardiorenal syndrome type 1

*Cardiorenal syndrome type 1* occurs in the setting of the acute worsening of heart function, most frequently in acute heart failure, or acute worsening of the chronic heart failure, in patients with acute coronary event, especially when resulting in cardiogenic shock and in patients after cardiac surgery. Acute kidney injury with decreased glomerular filtration rate is caused mainly by renal hypoperfusion and reduced oxygen delivery due to decreased cardiac output, other contributing factors may be the use of contrast media during coronary angiography and medication, especially inhibitors of renin-angiotensin system (both inhibitors of angiotensin converting enzyme and angiotensin antagonists) and diuretics, but also nephrotoxic antibiotics and nonsteroidal anti-inflammatory drugs. In CRS 1 it may be very difficult to define the optimal volume status as the patients are both at risk of overhydration (because of heart failure) and underfilling (because of excessive diuretic treatment with the impending risk of further worsening of kidney function, but also hypotension, hypoperfusion of other organs and tachycardia, or arrhythmias).

Cardiorenal syndrome type 1 develops more frequently in patients with diabetes, hypertension, obesity and metabolic syndrome, but also in patients with cachexia. The degree of kidney damage may be relatively mild and may remain undiagnosed without the use of specific biomarkers of acute kidney injury (as, e.g. NGAL, or KIM-1), but may be also severe and require (at least temporary) treatment with hemodialysis (intermittent or continuous).

Biomarkers may help to diagnose acute kidney injury as early as 2 h after the causal event (acute coronary event, cardiac surgery, administration of contrast media) when

serum creatinine still remains normal [2]. Recent reports on biomarkers more specific for acute cardiorenal syndrome, e.g. urinary angiotensinogen [3], or acute kidney injury after cardiac surgery, e.g. urinary netrin-1 [4], could further improve our potential to diagnose acute kidney injury early and also predict its outcome both in terms of overall and renal survival.

Patients with acute kidney injury were shown to be at increased risk of chronic kidney disease and end-stage renal disease [5]. Patients with acute kidney injury after coronary angiography were, however, also shown to have two times higher mortality (compared to patients without acute kidney injury, [6]) and similarly patients after acute myocardial infarction with acute kidney injury (defined as increase of serum creatinine by more than 30  $\mu\text{mol/l}$ ) had even after 4 years more than two times higher mortality compared to patients without acute kidney injury [7]. It is in keeping with the recent meta-analysis [5] which showed two times higher mortality for all patients with acute kidney injury (of different etiology) compared to patients with well preserved glomerular filtration rate. Interestingly enough, there is not only high in-hospital mortality, but also delayed mortality after 1 and 2 years after acute kidney injury [8].

Another recent meta-analysis demonstrated that patients with CRS-1 had two times higher mortality even 5 years after acute kidney injury and the mortality during the first month was almost five times higher [9]. The outcome is, not surprisingly, even much worse for patients who required renal replacement therapy (early mortality was ten times higher compared to patients without acute kidney injury). Patients with CRS-1 also required longer hospitalization and longer hospitalization in the intensive care unit (more than 3 times for all patients with CRS-1 and more than 20 times for patients who required renal replacement therapy – [9]).

Interestingly, the risk of acute kidney injury is much higher in patients with acute heart failure compared to cardiac surgery and namely acute coronary event (34%, 17%, 9%, respectively) and the risk of acute kidney injury requiring renal replacement therapy is also higher in acute heart failure and after cardiac surgery compared to acute coronary event (9% vs. 9% vs. 3% – [9]). On the other hand, mortality is higher in patients with acute kidney injury after cardiac surgery, compared to acute heart failure and acute coronary event (7.5% vs. 2.9% vs. 3.5%).

It is important to stress that early evaluation of urinary biomarkers may predict long-term outcome of the patients,

**Table 1 – Types of cardiorenal syndrome (according to [1]).**

Syndromes	Acute cardiorenal (type 1)	Chronic cardiorenal (type 2)	Acute renocardiac (type 3)	Chronic renocardiac (type 4)	Secondary cardiorenal (type 5)
Definition	Acute worsening of heart function resulting in kidney injury, or dysfunction	Chronic abnormalities in heart function leading to kidney injury, or dysfunction	Acute worsening of kidney function leading to heart injury or dysfunction	Chronic kidney disease leading to heart injury, disease or dysfunction	Systemic conditions leading to simultaneous dysfunction of heart and kidney
Primary events	Acute heart failure, or acute coronary event, or cardiogenic shock	Chronic heart disease	Acute kidney injury	Chronic kidney disease	Systemic disease (e.g. sepsis, amyloidosis)

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