The Management of Heart Failure in Kidney and Urinary Tract Syndromes

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Abstract

Kidney dysfunction and other related abnormalities are extremely common in all HF syndromes, both because of the similarity of risk factors and the similarity of demography of the two types of patients but also because of the common renal effects of agents used for the treatment of HF. Important renal syndromes for the HF patient include including chronic kidney disease, acute kidney injury, cardio-renal syndrome, and prostatic obstruction. In HF (all types including HFrEF, HFmrEF and especially HFpEF) chronic kidney disease (CKD) frequently co-exists and almost as frequently complicates the HF management. The two groups of syndromes share many risk factors (diabetes, hypertension, hyperlipidaemia) and often interact to worsen the prognosis of each other in a way that makes the patient with combined HF and renal disease at extremely high risk. This article reviews this common co-morbidity and how to manage it.

Keywords: Cardiology; Heart failure; Kidney disease; Guidelines

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Introduction

Kidney dysfunction and other related abnormalities are extremely common in all HF syndromes, both because of the similarity of risk factors and the similarity of demography of the two types of patients but also because of the common renal effects of agents used for the treatment of HF. Important renal syndromes for the HF patient include including chronic kidney disease, acute kidney injury, cardio-renal syndrome, and prostatic obstruction. In HF (all types including HFrEF, HFmrEF and especially HFpEF) chronic kidney disease (CKD) frequently co-exists and almost as frequently complicates the HF management. The two groups of syndromes share many risk factors (diabetes, hypertension, hyperlipidaemia) and often interact to worsen the prognosis of each other in a way that makes the patient with combined HF and renal disease at extremely high risk.[1,2] This common co-morbidity and how to manage it has been recently reviewed in the 2016 ESC/HFA heart failure guidelines.[3]

CKD is usually defined as an eGFR <60mL/min/1.73m² and/or the presence of albuminuria (high 30–300 or very high >300 mg albumin per 1 g of urine creatinine). There is a lack of an adequate evidence base for determining the best way to manage patients with combined HF and severe CKD for, almost universally, those with severe CKD (eGFR <30mL/min/1.73m²) were excluded from the large HF trials.

Another feature of concern is that even those who are started on appropriate HF medication a subsequent change in renal function (even if it were to later prove to be temporary) is used a reason to reduce dosage or cease mediation entirely. This so-called worsening renal function (WRF) is very commonly used clinically as a reason to reduce medication even if it does not satisfy the accepted diagnostic criteria for this term. WRF should be used to indicate an increase in serum creatinine by >26.5 µmol/L (0.3 mg/dL) and/or a 25% increase, or a 20% drop in glomerular filtration rate (GFR).[3] These small changes in renal function occur commonly but if repeated could progress underlying CKD.[4] Similar worsening of apparent renal function in the setting of an AHF hospitalization or an acute worsening of (decompensated) HF are not always clinically important if they prove to be short-lived. Effective treatments for AHF with appropriate decongestion, diuresis and relief of symptoms may satisfy such a WRF definition in the short-term but not prove detrimental long-term.[5] More significant and more prolonged worsening of renal function (acute kidney injury, AKI) is not as common in the setting of HF. When present there is an urgent need to review therapy as diuretic therapy combined with other nephrotoxic agents (the antibiotic gentamicin and trimethoprim for example, ACEIs, ARBs, NSAIDs, or high dose IV contrast media). It is important to differentiate short-term versus more
prolonged changes in apparent renal function. Short-term mild WRF is common in the clinical course of HF patients, especially during initiation and up-titration of RAAS inhibitor therapy or active decongestion with diuretics. If caused by RAAS inhibitor initiation or up-titration the change may be limited in time and extent and in many cases need not lead to permanent treatment discontinuation and unless the WRF is marked treatment benefit is largely maintained (497).[6] If repeated severe WRF should occur when RAAS inhibitors are used the clinician should look out for renal artery stenosis, fluid balance, adequate perfusion and all concomitant medication. Care is essential for electrolyte balance (see chapter on potassium balance).

Diuretic dosages may need to be increased in poor renal function, noting any resultant change in effective renal function. Renal diuretic resistance is more common with reduced renal reserve. Occasionally combination diuretics are needed, particularly a loop diuretic with a thiazide or with metolazone, but this should only be attempted by expert HF managers and only with very close observation on fluid balance, renal function and electrolyte balance.

Care is needed to avoid over-dosing renally excreted drugs such as digoxin, insulin, and low molecular weight heparin. Digoxin levels in particular should be closely monitored in patients with CKD and reduced doses utilized. Particular care is needed during angiography because of the risk of contrast-induced acute kidney injury (CI-AKI). Patients at risk need close monitoring. Other treatments are used in this setting, although the evidence for popular protective measures such as n-Acetyl cysteine remains weak.

In the setting of Acute HF, especially if severe, severe AKI may be common. In such cases renal replacement therapy may be needed as a short-term measure. The recommendations made in the recent ESC/HFA guidelines[3] state that ultrafiltration may be considered in the presence of AKI with refractory HF congestion, and if diuretic therapy has failed. Formal renal replacement therapy (including dialysis) should be considered in patients with refractory volume overload not responding to medical care.

In the presence of prostatic obstruction (common in older males with HF) care should be taken concerning drug interactions, for example, alpha-adrenoceptor blockers can cause hypotension, and sodium and water retention, and may not be safe in HFrEF. [3,7,8] If needed 5-alpha reductase inhibitors should be used in preference in all male HF patients.

Declaration of Interest
The author declares no conflicts of interest.

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References