Heart Failure COMPLICATED BY CARDIORENAL SYNDROME

NURSE TALKING TIPS SHEET

This nurse tipsheet was developed by AAHFN as resource in facilitating patient education. It provides additional information so that the nurse can supplement their patient teaching with the corresponding patient tipsheet. Because no one page could be exhaustive, a list of resources is provided on page two for additional information.

Patient teaching should focus on:

- Cardiorenal syndrome (CRS) is a complex disease process that requires delicate balance in the treatment of various types.
- Prevention of the syndrome provides superior outcomes when compared to treatment of the various types of CRS.
- Balanced aggressive treatment of HF will facilitate prevention of the syndrome.
- Key points of education: Medication use, prevention of hypo and hyperkalemia by diet and routine electrolyte monitoring, home blood pressure monitoring, fluid balance, daily weights, avoidance of further toxins such as contrast media, NSAIDs and thiazolidinediones (Actose, Avandia).
- Most supplements have not been tested in patients with HF and therefore may be inappropriate. COQ10 is under study as well as thiamine. Iron supplementation has found to be effective in patients with anemia and resultant HF.

Background:
The heart and kidneys are intimately related in function and work together to maintain delicate neurohormonal, vascular, electrolyte, and blood volume systems in the body. CRS is defined as the complex pathophysiological disorder of the heart and kidneys where by acute or chronic dysfunction in one organ may induce acute or chronic dysfunction in the other organ.

Five types of CRS exist:

Type 1 (acute): Acute heart failure (HF) results in acute kidney injury (AKI), previously called acute renal failure (ARF).

Type 2 (chronic): Chronic cardiac dysfunction (e.g., chronic HF) causes progressive chronic kidney disease (CKD), previously called chronic renal failure (CRF).

Type 3 (acute reno-cardiac syndrome): Abrupt and primary worsening of kidney function due to acute heart injury and/or dysfunction.

Type 4 (chronic reno-cardiac syndrome): Primary or chronic kidney disease contributes to cardiac dysfunction which may be manifested by coronary disease, HF or arrhythmias.

Type 5 (secondary): Acute or chronic systemic disorders that cause both cardiac and renal dysfunction.
Symptoms:

Patients are generally asymptomatic in regards to CRS, however the following risk factors increase the probability of developing CRS:

- Age
- Co-morbidities such as diabetes mellitus, uncontrolled hypertension, and anemia
- Drugs such as anti-inflammatory agents, diuretics, angiotensin converting enzyme inhibitors (ACEI) and angiotensin receptor blockers (ARB) and aldosterone receptor antagonists. Note that many of these are medicines specifically prescribed in HF.
- Impaired left ventricular function, prior myocardial infarction, poor functional class, elevated troponin.
- CKD with reduced glomerular filtration rate, elevated BUN and elevated Creatinine (Cr), elevated cystatin

Diagnosis:

Because CRS is a syndrome, the diagnosis is usually based on renal function studies, electrolytes, echocardiogram, troponins, glomerular filtration rate (GFR), C-reactive protein, uric acid, CK-MB, BNP, NT-proBNP. Serum cystatin C is under investigation as well as neutrophil gelatinase-associated lipocalin (NGAL) a biomarker for early kidney injury.

Treatment:

Successful treatment depends on the proper classification of the problem with prevention as the best strategy. Once the syndrome begins it is difficult to interrupt, is associated with serious adverse outcomes, and is not completely reversible in all cases. Aggressive management of heart failure symptoms is the key to prevention with usual self-management instruction: sodium restriction, exercise, daily weights, taking medications as prescribed, avoidance of harmful substances (alcohol, NSAIDs, Calcium Channel blockers), and regular follow-up.

If prevention is unsuccessful, several therapies can be tried. While there are no evidence-based guideline therapies in CRS, the following recommendations based on type of CRS have been suggested:

Type 1: Prompt resolution of causative factors such as infections, drugs, atrial fibrillation and hypertension. If the acute CRS is caused by shock, intra-aortic balloon pumping and inotropic agents may be needed.

Type 2: High dose diuretics, ACE-I, ARB, beta blockers, aldosterone receptor blockers, combination of nitrates and hydralazine and cardio re-synchronization therapy are important to reduce afterload and improve left ventricular functioning. Ultra filtration to decrease volume is currently being studied but has not been found to be superior to diuretics.

Type 3: In acute renal failure, hypovolemia and hypervolemia must be avoided. Monitoring of renal status is critical. Increase of the Cr more than .03mg/dl from baseline is clinically significant and should be reported. Electrolyte imbalance like hyperkalemia needs to be reported immediately. Avoid contrast media where possible.

Type 4: Management of CKD is imperative. Patients undergoing hemodialysis may develop acute ischemic symptoms such as chest pain, EKG changes and elevation of biomarkers. All cardiac medications should be prescribed and given based on the calculated GFR and not on Cr alone.

Type 5: Carries a negative prognosis. Systemic factors negatively impact function of the heart and kidneys. It is uncertain if reduction or elimination of the key components of the acute process will prevent both cardiac and renal decline.

For Further Reference:


