Cardiorenal Syndrome

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Disclosures: None
Cardiorenal Syndrome (CRS)

• What is it?
• Why is it important?
• Is it relevant to primary care?

CRS - What Is It?

• Cardiorenal syndrome (CRS) – (ICD 10 - I13.10)
  • Increasingly used term for an old condition
    • Described by Robert Bright in 1836.
  • 2004 NHLBI - CRS is a condition in which therapy to relieve congestive symptoms of HF is limited by a decline in renal function as manifested by a reduction in GFR (Cardiocentric – acute HF scenario)
  • New studies show the interactions “go both ways”
CRS - Definition

- AHA Scientific Statement on CRS
  - Circulation. 2019;139:e840–e878.
- Cardiorenal syndrome encompasses a spectrum of disorders involving both the heart and kidneys in which acute or chronic dysfunction in one organ may induce acute or chronic dysfunction in the other organ.

Classification

“Cardiorenal”

- **Type 1 (acute)** – Acute HF results in acute kidney injury.
- **Type 2** – Chronic cardiac dysfunction (e.g., chronic HF) causes progressive chronic kidney disease (CKD).
- **Type 3** – Abrupt and primary worsening of kidney function due, for example, to renal ischemia or glomerulonephritis causes acute cardiac dysfunction, which may be manifested by HF.
- **Type 4** – Primary CKD contributes to cardiac dysfunction, which may be manifested by coronary disease, HF, or arrhythmia.
- **Type 5 (secondary)** – Acute or chronic systemic disorders (e.g., sepsis or diabetes mellitus) that cause both cardiac and renal dysfunction.

“Renocardiac”

Cardiorenal Syndrome (CRS)

• What is it?
• Why is it important?
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Do you recognize this patient?
– 60 yo with HTN, T2DM, worsening DOE and pedal edema.
– BP 150/90, Cr 1.6, K 4.7, BNP 700, EF 40%
– Increase BP meds and diuretics
– Two weeks later patient is breathing better
– BP 140/85, Cr 2.1, K 5.3

• What now?
  – Decrease or stop diuretic?
  – Change BP meds?
  – Consult?
  » Prescribe ourselves a “Chill pill.”
Cardiorenal Syndrome (CRS)

- Significant overlap in the diseases.
- The prevalence of moderate to severe reductions in GFR (less than 60 mL/min per 1.73 m$^2$) in patients with HF has ranged from 30 to 60 percent in large clinical studies.

Common Clinic or Urgent Care Scenario

- Chronic HF with mild exacerbation
  - Noncompliance with meds or diet, NSAIDS, steroids, stimulants, acute illness, Recent IV contrast, RAAS blockers
  - Relief of congestion is paramount
  - Diuretic prescribed or increased
  - May result in concomitant increase in Scr
Common ED Scenario

- Acute or Chronic HF with moderate to severe exacerbation
  - Noncompliance with meds or diet, NSAIDS, acute illness, severe medical emergency
  - Because of volume overload status, Scr may actually be better than baseline
  - Relief of congestion is paramount
  - Frequently results in concomitant increase in Scr

Beware the Edema

- Because edema is such a common presenting complaint and can be a component of cardiac and/or renal disease, watch for iatrogenic sources.
  - High dose calcium channel blockers
  - Steroids
  - TZDs
  - Dietary changes with increased sodium intake
Patients at risk for CRS Type 1 have a narrow window for management of both blood pressure and volume; extremes in either parameter can be associated with worsened renal function.

Crucial Outpatient Contributions

- Recognize the interdependency of the heart and kidneys.
- Control of risk factors can prevent/retard CRS.
- BP, weight, OSA, A1C.
- Always look for exacerbating factors:
  - NSAIDS.
  - Dietary sodium.
  - Medication compliance.
  - Contrast media.
**Crucial Outpatient Contributions**

- Monitor labs as needed. Keep other providers informed.
- Don’t overreact to changes in Scr.
  - Increasing diuretic and then increasing water/fluid intake.
  - Many changes in Scr will stabilize or return to baseline over several weeks.
    - ...up to a 30% increase in creatinine that stabilizes within 2 months was associated with long-term nephroprotection in a systematic review of 12 randomized controlled studies...
- If patient is clinically improving, then continue therapy and recheck lab work again

**Cardiorenal Syndrome (CRS)**

- What is it?
- Why is it important?
- Is it relevant to primary care?
Cardiorenal Syndrome (CRS)

• A highly prevalent overlap of heart and kidney disease that leads to significant morbidity and mortality and presents to the generalist and specialist alike on a daily basis.
• Because of these factors, our therapies and interventions may work at cross purposes within individual practices or between PCP and specialist.

CRS – Is There Any Hope

• Existing risk factors can be managed
• New biomarkers of kidney injury (beyond the Scr) are being evaluated.
• Existing cardiac biomarkers are readily available and new ones are under investigation.
• Better communication between providers can prevent “spinning/reinventing our wheels.”
• Greater vigilance and understanding of CRS should result in better outcomes.
Conclusions

• Significant overlap in risk factors for both heart disease and renal disease.
  – HTN, Diabetes, Obesity, etc
• Therapy aimed at the disease or the symptoms will likely induce measurable changes.
• Be vigilant but not overly reactionary.

Conclusions

• If the patient is stable or clinically improving:
  – Increase frequency of clinical assessment, lab assessment, or both.
  – Communicate to the patient and others that there is an overall plan.
  – Resist the temptation to alter the course based on minor changes in lab values.
  – Engage the patient in their care to avoid heart failure and dialysis.
Thank You

Selected References

CRS Type 1 Pathophysiological interactions between heart and kidney in type 1 cardiorenal syndrome (CRS) or “acute CRS” (abrupt worsening of cardiac function, e.g., acute cardiogenic shock or acute decompensation of chronic heart failure) leading to kidney injury. ACE = angiotensin-converting enzyme; ANP = atrial natriuretic peptide; BNP = B-type natriuretic peptide; CO = cardiac output; GFR = glomerular filtration rate; KIM = kidney injury molecule; N-GAL = neutrophil gelatinase-associated lipocalin; RAA = renin angiotensin aldosterone.

CRS Type 2 Pathophysiological interactions between heart and kidney in type 2 cardiorenal syndrome (CRS) or “chronic CRS” (chronic abnormalities in cardiac function, e.g., chronic heart failure) causing progressive chronic kidney disease (CKD). Figure illustration by Rob Flewell. LVH = left ventricular hypertrophy; RAA = renin angiotensin aldosterone.
CRS Type 3
Pathophysiological interactions between heart and kidney in type 3 CRS or “acute renocardiac syndrome” (abrupt worsening of renal function, e.g., acute kidney failure or glomerulonephritis) causing acute cardiac disorder (e.g., heart failure, arrhythmia, pulmonary edema). MPO = myeloperoxidase

CRS Type 4
Pathophysiological interactions between heart and kidney in type 4 cardiorenal syndrome (CRS) or “chronic renocardiac syndrome” (chronic kidney disease [CKD], e.g., chronic glomerular disease, contributing to decreased cardiac function, cardiac hypertrophy, or increased risk of adverse cardiovascular events).
CRS Type 5 Pathophysiological interactions between heart and kidney in type 5 cardiorenal syndrome (CRS) or “secondary CRS” (systemic condition, e.g., diabetes mellitus, sepsis, causing both cardiac and renal dysfunction). LPS = lipopolysaccharide (endotoxin); RVR = renal vascular resistance. Figure illustration by Rob Flewell.