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Cardio-Renal Syndrome: A Case Report Highlighting Clinical Presentation, Management, and Outcomes

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Case Report

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ABSTRACT

Cardio-renal syndrome (CRS) represents a complex pathophysiological condition in which acute or chronic dysfunction in the heart or kidneys induces dysfunction in the other organ. It is associated with significant morbidity and mortality, posing challenges in diagnosis and management. This case report describes a patient presenting with acute decompensated heart failure complicated by worsening renal function, consistent with Type 1 cardio-renal syndrome. The report discusses clinical findings, diagnostic evaluation, therapeutic interventions, and outcomes, emphasizing the importance of early recognition and multidisciplinary management.

KEYWORDS

Cardio-renal syndrome (CRS), Type 1 cardio-renal syndrome, Acute decompensated heart failure,Acute kidney injury (AKI), Heart–kidney interaction.

INTRODUCTION

Cardio-renal syndrome is a bidirectional disorder involving the heart and kidneys, where dysfunction in one organ adversely affects the other. It is broadly classified into five subtypes based on the primary organ involved and the acuity of the condition. Type 1 CRS refers to acute worsening of cardiac function leading to acute kidney injury (AKI), while Type 2 involves chronic heart failure causing chronic kidney disease (CKD).

The interplay between cardiac and renal systems is mediated by hemodynamic, neurohormonal, and inflammatory mechanisms. Reduced cardiac output, venous congestion, activation of the renin-angiotensin-aldosterone system (RAAS), and sympathetic nervous system contribute to renal hypoperfusion and injury.

This case report presents a patient with acute decompensated heart failure who subsequently developed acute kidney injury, illustrating the clinical features and management challenges of cardio-renal syndrome.

Case Presentation

Patient Information

A 65-year-old male presented to the emergency department with complaints of progressive shortness of breath, swelling of the lower limbs, and decreased urine output over the past five days. He reported orthopnea and paroxysmal nocturnal dyspnea.

The patient had a known history of hypertension, type 2 diabetes mellitus, and ischemic heart disease. He was on irregular medication and had poor compliance with dietary restrictions.

Clinical Findings

On examination, the patient appeared dyspneic and fatigued. Vital signs revealed:

- **Blood pressure:** 160/95 mmHg
- **Heart rate:** 110 beats per minute

- **Respiratory rate:** 24 breaths per minute
- **Oxygen saturation:** 90% on room air

Physical examination showed bilateral pitting edema, elevated jugular venous pressure, and basal lung crackles. Cardiac auscultation revealed a third heart sound (S3 gallop), suggestive of heart failure.

Diagnostic Assessment

Laboratory Investigations

- Serum creatinine: 2.5 mg/dL (baseline: 1.2 mg/dL)
- Blood urea nitrogen (BUN): Elevated
- Serum electrolytes: Hyponatremia and mild hyperkalemia
- Brain natriuretic peptide (BNP): Elevated
- Hemoglobin: Mild anemia
- Urinalysis
- Proteinuria (+)
- No evidence of infection
- Imaging Studies
- Chest X-ray: Cardiomegaly with pulmonary congestion
- Echocardiography: Left ventricular ejection fraction (LVEF) of 30%, indicating systolic dysfunction
- Ultrasound abdomen: Normal kidney size with no obstruction

These findings supported the diagnosis of acute decompensated heart failure with acute kidney injury, consistent with Type 1 cardio-renal syndrome.

Diagnosis

Based on clinical presentation and investigations, the patient was diagnosed with:

Type 1 Cardio-Renal Syndrome (Acute Cardiac Failure Leading to Acute Kidney Injury)

Therapeutic Intervention

Initial Management

The patient was admitted to the intensive care unit and managed with:

- Oxygen therapy
- Intravenous loop diuretics (furosemide)
- Vasodilators to reduce preload and afterload
- Fluid restriction and sodium restriction
- Pharmacological Treatment
- Angiotensin-converting enzyme (ACE) inhibitors (initiated cautiously)
- Beta-blockers (introduced after stabilization)
- Aldosterone antagonists
- Insulin therapy for glycemic control

Monitoring

Close monitoring of renal function, urine output, electrolytes, and hemodynamic status was performed. Daily weight measurement and fluid balance assessment were maintained.

Clinical Course and Outcome

During the initial 48 hours, the patient showed partial improvement in respiratory symptoms; however, renal function worsened transiently due to aggressive diuresis. Adjustments in diuretic dosing and careful fluid management led to gradual stabilization.

By day 7, the patient's urine output improved, and serum creatinine levels began to decline. Edema reduced significantly, and respiratory status improved.

The patient was discharged after 10 days with stable renal function and improved cardiac status. He was advised strict medication adherence, dietary modifications, and regular follow-up.

DISCUSSION

Cardio-renal syndrome represents a complex interaction between cardiac and renal dysfunction. In this case, acute heart failure led to reduced renal perfusion and increased venous pressure, resulting in acute kidney injury.

Pathophysiology

The underlying mechanisms include:

- Reduced cardiac output leading to decreased renal blood flow
- Venous congestion impairing kidney function
- Activation of RAAS and sympathetic nervous system
- znflammatory and oxidative stress pathways

These factors contribute to a vicious cycle, where worsening renal function further exacerbates cardiac dysfunction.

Diagnostic Challenges

Diagnosing CRS requires distinguishing it from isolated cardiac or renal disease. Biomarkers such as BNP and imaging studies are useful in identifying cardiac involvement, while renal function tests help assess kidney injury.

Early recognition is crucial to prevent progression and complications.

Management Considerations

The management of CRS is challenging due to the need to balance fluid removal with preservation of renal function. Diuretics remain the mainstay of treatment but must be used cautiously.

ACE inhibitors and beta-blockers improve cardiac function but may initially worsen renal parameters. Therefore, close monitoring is essential.

In severe cases, renal replacement therapy or ultrafiltration may be required.

Prognosis

The prognosis of cardio-renal syndrome depends on the severity of organ dysfunction and timely intervention. Patients with CRS have higher rates of hospitalization and mortality compared to those with isolated heart or kidney disease.

Early diagnosis and multidisciplinary management can improve outcomes.

CONCLUSION

This case highlights the clinical complexity of cardio-renal syndrome and the challenges in its management. The interplay between heart and kidney dysfunction necessitates a comprehensive and individualized treatment approach.

Timely recognition, careful monitoring, and appropriate therapeutic interventions are essential to break the cycle of organ dysfunction and improve patient outcomes. Increased awareness and further research are needed to develop optimized treatment strategies for this condition.

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