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Neurohormonal Activation in Cardiorenal Syndrome

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Introduction
Cardiac and renal dysfunction are highly related as each system is interdependent on the other; and subsequently, dysfunction of one system can potentiating the development of the other. A condition referred to as Cardiorenal Syndrome, or CRS (Scully & Goldsmith, 2013). In CRS, heart failure and renal failure exist together: Heart failure is the result of decreased heart efficiency and aquaporin channels, renal dysfunction can result in increased fluid retention and the maintenance of fluid and sodium from the kidneys. The net effects of the RAA results in increased blood volume and systemic vascular resistance to increase renal perfusion. A negative feedback system in turn inhibits the RAA system once homeostasis is returned (Klabunde, n.d.).

Signs and Symptoms
The signs and symptoms associated with CRF are similar to those experienced in heart failure. The signs and symptoms are the result of cardiac and renal dysfunction combined together. The New York Heart Association classification scale can be used to describe symptoms.

Nursing Implications
Nearly five million people in the United States are diagnosed with heart failure, and another half a million each year. There is a significant economic burden as well, with over $33 billion associated costs in 2010 (Coons, McGraw, & Murali, 2011). Lifestyle modifications should include the elimination of behaviors that can further decrease cardiac function. Modifiable lifestyle factors include:

- Smoking
  - Diet
  - Exercise
  - Alcohol use

Cardiac output is essential to reduce the signs associated with medications used in CRF management, especially hypotension. Patients should be educated to:

- Eliminate the use of NSAIDs, which can decrease renal function and should be taught to
- Limit salt substitutes (often low sodium diets are implemented), as the replacements can be high potassium (O'Donnovan, 2014).
- Serum potassium and creatinine levels should be monitored by practitioners for the assessment of renal function. At the initiation of an ACE-I, renal function should be monitored, and dosing is adjusted according to O'Donnovan (2014).

Underlying Pathophysiology
The common pathological mechanism of CRS is the chronic activation of the RAA resulting in oxidative stress leading to impaired cardiac and renal dysfunction. When cardiac output is decreased, hyperperfusion of the kidneys and a decrease in glomerular filtration rate (GFR) can result. This decrease in perfusion results in the activation of the RAA, a normal mechanism of compensation. Activation of the RAA system results in several changes. First, conversion of angiotensin to angiotensin II results in vasoconstriction, in turn, increased systemic vascular resistance and blood pressure. Second, Aldosterone is stimulated to be released from the adrenal cortex. Aldosterone results in the retention of fluid and sodium from the kidneys. The net effects of the RAA system is increased blood volume and systemic vascular resistance to increase renal perfusion. A negative feedback system inhibits the RAA system once homeostasis is returned (Klabunde, n.d.).

Significance
Cardiorenal Syndrome is a complex condition. Normal compensatory mechanisms used to maintain homeostasis actually become deleterious for both cardiac and renal function. The significance of the pathophysiology lies in the management of CRS.

- Treatment of CRS involves choices that are contradictory.
- Cornerstone of management is ACE-I but use can worsen renal function as indicated by elevated serum creatinine and BUN levels (House, et al., 2010).
- Diuretics are used to reduce preload, have been shown to be beneficial in heart failure.
- Similar to ACE-I, diuretics can have negative effects and further complicate CRS.

Conclusion
Cardiorenal Syndrome is a highly complex pathophysiologic processes that involves several mechanisms. The RAA acts normally as a compensatory neurohormonal mechanism to maintain GFR and renal perfusion. In heart failure however, chronic activation of the RAA system due to decreased renal perfusion can lead the development of functional and cardiac dysfunction, deemed Cardiorenal Syndrome (Giam, et al, 2016). The associated renal pathophysiologic process of CRS guides therapies, which can be directly contradictory, meaning that the management of heart failure can worsen renal function and vice versa (Konari, et al., 2010). Nurses have several implications in the management of CRS, specifically related to education. As evidence continues to emerge related to education, the care team must continue to advance, but at the current time, CRS remains a complex process that can lead to poor outcomes.

Table 1. Classifications of CRS

<table>
<thead>
<tr>
<th>Class</th>
<th>Description</th>
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<tbody>
<tr>
<td>Class I</td>
<td>acute cardiac injury results in renal injury</td>
</tr>
<tr>
<td>Class II</td>
<td>heart failure leading to chronic renal failure</td>
</tr>
<tr>
<td>Class III</td>
<td>acute kidney injury resulting in cardiac injury</td>
</tr>
<tr>
<td>Class IV</td>
<td>chronic renal failure leading to heart failure</td>
</tr>
<tr>
<td>Class V</td>
<td>cardiac and renal dysfunction secondary to a systemic disease</td>
</tr>
</tbody>
</table>

Figure 1: Cardiorenal Syndrome (House, et al., 2010).

In Cardiorenal syndrome heart failure results in renal injury. Chronic activation of the RAA system becomes chronically activated, resulting in deleterious effects that can potentiate the failure of both cardiac and renal dysfunction. Chronic activation of the RAA system can result in inflammation, apoptosis, and oxidative stress. Angiotensin II appears to be a significant contributor in CRS. A chronic elevation of angiotensin II has been shown to induce apoptosis in renal and cardiac cells as well as activation of NADPH oxidase. NADPH leads to the release of reactive oxygen species (ROS), which in turn reduces the availability of nitric oxide (vasodilating effects in normal circumstances) and increased vascular constriction. The increased levels of ROS also stimulate the production of pro-inflammatory mediators. The best understood mediators are interleukin-6 and transforming growth factor-beta, which contribute to fibrosis in the heart and kidneys. Fibrotic tissue results in a change of structure, producing a change of function. Angiotensin II is also responsible for the stimulation and release of aldosterone, which when chronically activated, can cause increased collagen formation and further increasing fibrosis (Giam, et al, 2016). According to Konari, et al., (2010), the cytokines produced also have a negative inotropic effect, which causes a further reduction in cardiac output and renal perfusion. When combined together, all components lead to decreased functioning of cardiac and renal systems. Other deleterious effects of chronic activation of RAA system includes an increased preload and afterload, which can complicate heart failure further.

Figure 2: RAA System (Klabunde, 2014).

References