RHABDOMYOLYSIS – DIAGNOSIS AND TREATMENT

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**Rhabdomyolysis: Diagnosis and Treatment**

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**Introduction**

Rhabdomyolysis results from the rapid breakdown of skeletal muscle fibers, which leads to leakage of potentially toxic cellular content into the systemic circulation (Hamel et al., 2015, p. 621). Rhabdomyolysis is a syndrome that is characterized by muscle necrosis and the release of intracellular muscle constituents into circulation leading to complications and disease. Rhabdomyolysis symptoms range from minor body aches to life-threatening disease and acute kidney injury. The clinical features of rhabdomyolysis include myalgia, muscle weakness, myoglobinuria and muscle swelling that develops over hours to days (Nance & Manten, 2015, p. 789).

Working in a prison unit I have witnessed several cases of patients that are to be treated for rhabdomyolysis. They have been captivated by these cases and I have learned to understand the mechanics of the syndrome. Given the number of cases that are witnessed in the unit and are eventually diagnosed with rhabdomyolysis it is pertinent for an advanced nurse practitioner to understand the pathophysiology of the disease. Understanding the mechanics of the disease will help in diagnosis thus timely treating the condition hence improving patient outcomes. Most of the cases encountered are non-traumatic exertional rhabdomyolysis as in patients. They do not have much to do with the prison camps and they do not engage in exercises such as squats or push-ups unknowingly causing rhabdomyolysis.

**Pathophysiological Processes**

- Clinical manifestations and complications of rhabdomyolysis result from muscle cell death, which may be triggered by a variety of initiating events (Torres, Helmstetter, Kaye, & Kaye, 2015).
- Traumatic or muscle compression (e.g. crush syndrome or prolonged immobilization)
- Nontraumatic exertional (e.g. marked strain in untrained individuals, hyperthermia, or metabolic myopathies)
- Nontraumatic nonexertional (e.g. drugs or toxins, infections, or electrolyte disorders)
- Common pathway for injury is an increase in intracellular free ionized cytoplasmic and mitochondrial calcium and sodium
- An increase in intracellular calcium and sodium draws water into the cell and disrupts the integrity of the intracellular space (Torres, Helmstetter, Kaye, & Kaye, 2015, p. 60)

This may be caused by depletion of adenosine triphosphate (ATP), the cellular source of energy, and/or by direct injury and rupture of the plasma membrane.

- Increased intracellular calcium leads to activation of proteases, increased skeletal muscle cell contractility, mitochondrial dysfunction, and the production of reactive oxygen species, resulting in skeletal muscle cell death.
- ATP depletion leads to myocyte injury and the release of intracellular muscle constituents, including creatine kinase (CK) and other cytoplasmic and mitochondrial enzymes.
- The end result of these alterations within the muscle cell milieu is an inflammatory, self-sustaining myotic cascade that causes necrosis of the muscle fibers and releases the muscle contents into the extracellular space and the bloodstream (Torres, Helmstetter, Kaye, & Kaye, 2015, p. 60).

**Signs & Symptoms**

- The classic triad of symptoms of rhabdomyolysis consists of myalgia, weakness, and tea-colored urine (Torres, Helmstetter, Kaye, & Kaye, 2015).
- Patients may also present with oliguria or even anuria.
- Drug-induced syndromes associated with rhabdomyolysis are characterized by muscle rigidity, hyperthermia, and metabolic acidosis (Chavez et al., 2015).

*Altered mental status may occur from the underlying etiology (e.g., toxins, drugs, trauma, or electrolyte abnormalities* (Miller, 2016).

**Significance of Pathophysiology**

Pathophysiology of rhabdomyolysis is significant to clinicians, knowledge of the pathways of the disease helps a clinician understand what to expect and how to properly manage a patient.

Pathophysiology of rhabdomyolysis allows the clinician to be proactive instead of reactive hence reducing complications from the syndrome. The clinician has to understand pathophysiology of rhabdomyolysis and the signs and symptoms of the disease to ensure timely diagnosis and treatment. Rhabdomyolysis can be life threatening and disabling, having a clinician who knows how the syndrome progresses is vital in ensuring patient outcomes.

While the patient benefits from a knowledgeable clinician, pathophysiology of rhabdomyolysis is also of critical significance to the patient. A patient who is knowledgeable about the disease process and pathophysiology is able to prevent the disease from occurring and has better outcomes when affected by such a disease. Patients who have an understanding of the pathophysiology of any disease are likely to be compliant to treatment regimen and are likely to engage in practices that prevent the disease. Understanding pathophysiology of rhabdomyolysis is important in the overall management of the disease and ensures successful reversal of symptoms.

**Implications for Nursing Care**

Treatment of Rhabdomyolysis requires early recognition of the syndrome and early intervention before systemic complications are set. In implications of nursing care involves providing ongoing nursing care. The treatment of rhabdomyolysis includes fluid replacement via intravenous therapy.

“Volume repletion with saline is essential to avoid hypovolemia; shock and acute kidney injury” (Gina & Pach, 2016, p. 2). Nursing care will include sequential monitoring of urine output or volume, color and specific gravity to guide continuous fluid and electrolyte replacement. The nurse should be vigilant to ensure no major complications occur.

**References**


