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MUSCLE DESTRUCTION WITH MYOGLOBINURIA

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Abstract: Myoglobinuria is the presence of myoglobin in the urine, usually associated with rhabdomyolysis or muscle destruction. Myoglobin is present in muscle cells as a reserve of oxygen. Myoglobinuria is usually the result of rhabdomyolysis or muscle destruction. Any process that interferes with the storage or use of energy by muscle cells can lead to myoglobinuria.

Key words: Myoglobin, Rhabdomyolysis, Hyperthermia, Urine, Muscle destruction

Introduction:

Myoglobinuria is usually the result of rhabdomyolysis or muscle destruction. Any process that interferes with the storage or use of energy by muscle cells can lead to myoglobinuria. The release of myoglobin from muscle cells is often associated with an increase in levels of creatine kinase (CK), aldolase, lactate dehydrogenase (LDH), serum glutamic-pyruvic transaminase (SGPT), and other enzymes. When excreted into the urine, myoglobin, a monomer containing a heme molecule similar to hemoglobin, can precipitate, causing tubular obstruction and acute kidney injury.



A clinician caring for a patient with crush injuries or other causes of muscle destruction must recognize the presence and severity of myoglobinuria and initiate aggressive hydration to prevent acute kidney injury.

The most common causes of myoglobinuria in adults are trauma, alcohol and drug abuse, usually in relation to muscle necrosis from prolonged immobilization and pressure by the body weight. Prolonged ethanol consumption and seizure activity, similar to excessive physical activity, can produce an imbalance between muscle energy consumption and production, resulting in muscle destruction. In children and adolescents, the most common causes of rhabdomyolysis and myoglobinuria are viral myositis, trauma, exertion, drug overdose, seizures, metabolic disorders, and connective tissue disease.

Pathophysiology

Myoglobin is released from muscle tissue by cell destruction and alterations in the permeability of the skeletal muscle cell membrane. Under normal conditions, the sodium potassium ATPase pump maintains very low intracellular sodium content. A separate sodium-calcium channel then serves to pump additional sodium into the cell in exchange for calcium extrusion from the cell. In addition, most intracellular calcium is normally sequestered within organelles. Damage to muscle cells interferes with both mechanisms, leading to an increase in free ionized calcium in the cytoplasm. The high intracellular calcium activates numerous calcium-dependent enzymes that further break down the cell membrane, leading to the release of intracellular contents such as myoglobin and creatine kinase into the circulation. A model of the helical domains of myoglobin is shown in the image below.

Model of helical domains in myoglobin.

Myoglobin is a dark-red, monomeric heme protein that contains iron in its ferrous (Fe^{+2}) form. It is easily filtered by the glomerulus and is rapidly excreted into the urine. Plasma levels of myoglobin rapidly fall after its release. When large amounts of myoglobin enter the renal tubule lumen, it interacts with the Tamm-Horsfall protein and precipitates; this is a process favored by acidic urine. Tubule obstruction principally occurs at the level of the distal tubule. In addition, reactive oxygen species generated by damage to both muscle and kidney epithelial cells promote the oxidation of ferrous oxide to ferric oxide (Fe^{+3}), thus generating a hydroxyl radical.

Both the heme moieties and the free iron-driven hydroxyl radicals may be critical mediators of the direct tubule toxicity, which mainly occurs in the proximal tubule.

Thus, the precipitation of myoglobin in the renal tubules with secondary obstruction, tubular toxicity, or both constitutes the primary causes for acute kidney injury during myoglobinuria. A higher volume of urine flow and a higher urine alkalinity prevent myoglobin from precipitating as readily as it otherwise does.

Epidemiology

The frequency of myoglobinuria varies with the incidence of natural disasters and environmental trauma. Epidemics of viral myositis may temporarily increase the incidence in local areas. In urban areas with a high incidence of drug and alcohol abuse, many patients with myoglobinuria may present to emergency departments. Hot weather increases the incidence of stress induced rhabdomyolysis, especially in young athletes.

Mortality/Morbidity

Myoglobinuria causes little or no morbidity or mortality unless it is associated with the secondary complications of rhabdomyolysis, including hyperkalemia, hypocalcemia, and acute kidney injury. However, when it is associated with severe rhabdomyolysis, myoglobinuria-induced acute renal failure is a potentially lethal complication.

In adults, rhabdomyolysis can be complicated by acute kidney injury in approximately 30% of patients, with about 5% of those requiring hemodialytic support. In the pediatric age group, although previous small case series reported acute renal failure rates of 40-50%, a large retrospective review indicates that only about 5% of subjects with rhabdomyolysis develop acute kidney injury (defined as a serum creatinine level >97.5 percentile for age and gender). Rhabdomyolysis accounts for or contributes to about 7% of all causes of acute kidney injury in the United States. In both adults and children, the overall mortality rate of acute severe rhabdomyolysis is reported to be 7-8% and is primarily related to acute renal failure and multiorgan failure.

Race

Race is a factor only when natural disasters and economic shortfalls increase the rates of drug and alcohol abuse and the mortality rate among certain racial groups.

Sex

Myoglobinuria tends to affect males more than females because of the former group's predisposition to trauma and participation in strenuous physical exercise. Persons who exercise and have increased muscle mass have increased intracellular myoglobin content.

Age

In a recent large retrospective review, the median age was 11 years. The leading cause of rhabdomyolysis in the 0-9 year age range was viral myositis, whereas the leading diagnosis in the 9-18 year age range was trauma.

Causes

Trauma, vascular problems, malignant hyperthermia, certain drugs and other situations can destroy or damage the muscle, releasing myoglobin to the circulation and thus to the kidneys. Under ideal situations myoglobin will be filtered and excreted with the urine, but if too much myoglobin is released into the circulation or in case of renal problems, it can occlude the renal filtration system leading to acute tubular necrosis and acute renal insufficiency.

Other causes of myoglobinuria include:

- McArdle's disease
- Phosphofructokinase deficiency
- Carnitinepalmitoyltransferase II deficiency
- Malignant hyperthermia
- Polymyositis
- Lactate dehydrogenase deficiency
- Thermal or electrical burn

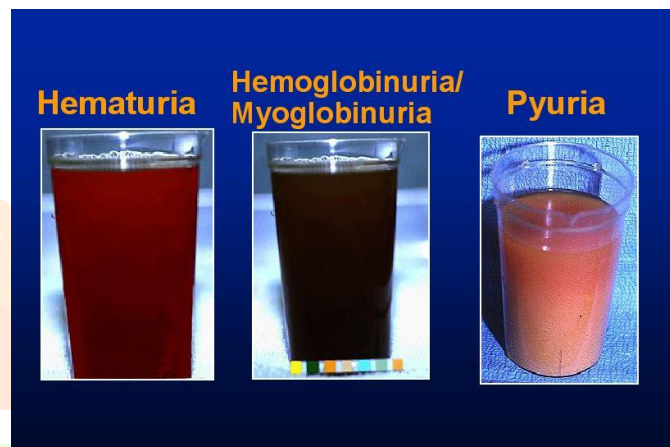
Pathophysiology

Myoglobinuria pathophysiology consist of a series of metabolic actions in which damage to muscle cells affect calcium mechanisms, thereby increasing free ionized calcium (cytoplasm). This in turn affects several enzymes that are calcium dependent, thereby compromising the cell membrane, which in turn causes the release of myoglobin.

Signs and symptoms

Signs and symptoms of myoglobinuria are usually nonspecific and needs some clinical prudence. Therefore, among the possible signs and symptoms to look for would be

- Swollen and painful muscles
- Fever, nausea
- Delirium (elderly individuals)
- Myalgia
- Dark urine
- Calcium ions (decrease)



Diagnosis

After centrifuging, the serum of myoglobinuria is clear, where the serum of hemoglobinuria after centrifuge is pink to red.



Treatment

Hospitalization and IV hydration should be the first step in any patient suspected of having myoglobinuria or rhabdomyolysis. The goal is to induce a brisk diuresis to prevent

myoglobin precipitation and deposition, which can cause acute kidney injury. Mannitol can be added to assist with diuresis. Adding sodium bicarbonate to the IV fluids will cause alkalization of the urine, believed to reduce the breakdown of myoglobin into its nephrotoxic metabolites, thus preventing renal damage. Often, IV normal saline is all that is needed to induce diuresis and alkalize the urine.

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