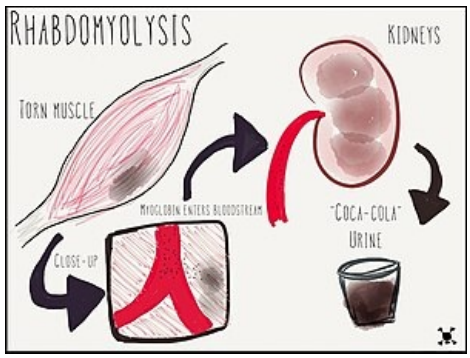
Rhabdomyolysis

Rhabdomyolysis is a condition in which **muscle cells are damaged** and myocyte content are released into the plasma. Rhabdomyolysis can be completely asymptomatic with elevation of muscle enzymes in the blood. However, extensive muscle damage can result in a severe increase in myoglobin and potassium ions - arrhythmias and acute renal failure.

If rhabdomyolysis is suspected, renal function, coagulation status, ionogram, circulatory parameters and lung function should be monitored.

Etiology

Rhabdomyolysis can be caused by congenital and acquired diseases.



Rhabdomyolysis

Congenital causes

- **Muscle glycogenosis disorders:** *myophosphorylase* deficiency (GSD V).
- **Fatty acid metabolism disorder:** primary carnitine deficiency (CUD), *carnitine acyl carnitine translocase* deficiency (CACT), *carnitine palmitoyl transferase II* deficiency (CPT II), long chain fatty acid *acyl-CoA dehydrogenase* deficiency (VLCAD) or deficiency Long chain fatty acid *dehydrogenase 3-hydroxyacyl-CoA* (LCHAD) and mitochondrial trifunctional protein (MTP) deficiency.
- **Mitochondrial disorders:** coenzyme Q10 deficiency, respiratory chain disorders.
- **Muscle diseases:** dystrophinopathy, myotonia congenita, myotonic dystrophy, etc.
- **Diseases at risk of malignant hyperthermia:** central core disease, familial malignant hyperthermia.
- **Other genetic syndromes:** autosomal dominant myoglobinuria, *myoadenylate deaminase deficiency*, *glucose-6-phosphate dehydrogenase deficiency*, idiopathic rhabdomyolysis.

Acquired causes

- **Direct muscle damage:** crush syndrome, burns, frostbite, lightning strike, long immobilization.
- **Extreme muscle activity:** excessive muscle effort in untrained individuals, status epilepticus, status asthmaticus, electric shock.
- **Extreme temperatures:** toxic shock syndrome, overheating, neuroleptic malignant syndrome.
- **Ischemia:** muscle compression, vascular occlusion, DIC.
- **Toxins:** snake venom, insect venom.
- **Drugs and drugs:** drugs (statins, fibrates, corticoids, phenytoin, succinylcholine), drugs (cocaine, toluene, ecstasy), alcohol.
- **Infections:** bacterial (legionella, salmonella, streptococci), viral (influenza A, B, adenoviruses, coxackie viruses), others (aspergillus, candida, mycoplasma).
- **Inflammatory myopathies:** polymyositis, dermatomyositis, vasculitis.
- **Endocrine disorder:** diabetic ketoacidosis, hypothyroidism, non-ketotic hyperosmolar coma.
- **Water and mineral disorders:** hyponatraemia (below 120 mmol/l) in SIADH, hypokalaemia, hypophosphataemia.

Consequences of rhabdomyolysis

With muscle cells necrosis, **potassium is flushed out** and hyperkalemia appears in the blood, which causes an arrhythmia in the patient. The released thromboplastin triggers disseminated intravascular coagulation (DIC). The patient is also threatened by a change in the permeability of the pulmonary capillaries, which can result in ARDS.

⚠With extensive rhabdomyolysis, hypovolemia and hypovolemic shock occur, which directly threatens the patient's life.

If the patient has developed rhabdomyolysis and serum creatine kinase levels are 80 times normal and, in addition, we detect metabolic acidosis with bicarbonate below 17 mmol/l, then rhabdomyolysis is highly likely to lead to **acute renal failure**, and forced diuresis must be started (delivery fluids and subsequent administration of loop diuretics).

Clinical Symptoms

Early rhabdomyolysis is manifested by nausea and vomiting, which are the consequences of ischemia or intestinal infarction.

A typical triad that occurs in 50% of patients is:

- **muscle pain** and weakness (pain may be diffuse or localized to a specific muscle group),
- **elevation of muscle enzymes** in serum,
- **myoglobinuria,** in which the urine is dark in color.

⚠ Myoglobinuria may be overlooked as it may develop and then cease before the examination. Myoglobin disappears from serum much faster than creatine kinase. Therefore, myoglobinuria may be absent at high creatine kinase levels.


Dark urine at rhabdomyolysis

Diagnosis

- Clinical signs and history.
- Serum levels of creatine kinase that do not originate in the myocardium.
- If rhabdomyolysis is suspected, the patient is examined for creatin kinase on admission and then 12 to 24 hours after admission.
- Urine myoglobin testing is not performed as standard. If the urine is the color of dark tea and no erythrocytes are present in the urine sediment, rhabdomyolysis is suspected.

Therapy

If possible, we will initiate rhabdomyolysis therapy by treating or eliminating the underlying cause. Symptomatic treatment includes adequate **hydration, alkalization of urine, and increased diuresis** in patients with low urine output. This way, we can alleviate acute renal failure caused by rhabdomyolysis. The patient is monitored in the ICU. We further supplement this treatment with nursing care, the prevention of pressure ulcers is important. If the patient reaches the stage of oliguric or anuric renal failure, we initiate intermittent **dialysis** or use continuous methods.

With severe rhabdomyolysis, we think of the danger of **compartment syndrome**; when the pressure rises above 35 mm Hg in a given osteofascial space, we approach the fasciotomy. Other therapeutic approaches depend on the underlying cause.

Links

Related Articles

- Muscle
- Acute renal failure
- Glycogenosis
- Mitochondrial diseases / Disorders of beta oxidation and ketogenesis
- Blast syndrome
- Crush syndrome
- Compartment syndrome

External links

- Acute renal failure in critically ill patients with rhabdomyolysis (article in the journal Internal Medicine for Practice) (http://www.internimedicina.cz/pdfs/int/2005/11/05.pdf)
- Rhabdomyolysis as a manifestation of a congenital disorder of energy metabolism in a two-year-old boy (article in the journal Pediatrics for Practice) (http://www.pediatriepropraxi.cz/pdfs/ped/2014/01/10.pdf)

Used literature

- JABOR, Antonín, et al. *Vnitřní prostředí.* 1. vydání. Praha : Grada, 2008. ISBN 978-80-247-1221-5.

References

1. RUCKI, Štěpán a Eva HRUBÁ. Rhabdomyolýza jako projev vrozené poruchy energetického metabolizmu u dvouletého chlapce. *Pediatrie pro praxi.* 2014, roč. 14, vol. 1, s. 38-41, ISSN 1803-5264.
2. ↑ SOUČEK, Martin, Sabina PÁLOVÁ a Jiří CHARVÁT. Akutní selhání ledvin u kriticky nemocných pacientů s rhabdomyolýzou. *Interní medicína pro praxi* [online]. 2005, roč. 11, s. 489-491, dostupné také z <http://www.internimedicina.cz/pdfs/int/2005/11/05.pdf>. ISSN 1803-5256.