



### Acute Rhabdomyolysis and Acute Cocaine Intoxication

Gargantilla Madera P<sup>1,2\*</sup>, Martín Cabrejas BM<sup>3</sup> and Pintor Holguín E<sup>2</sup>

<sup>1</sup>Department of Internal Medicine, Hospital El Escorial, Spain

<sup>2</sup>European University of Madrid, Spain

<sup>3</sup>Department of Gynecology and Obstetrics, Hospital Universitario Fundación de Alcorcón, Spain

\*Corresponding author: Gargantilla Madera P, Department of Internal Medicine, Hospital El Escorial, European University of Madrid, Madrid, Spain, E-mail: [pgargantilla@yahoo.es](mailto:pgargantilla@yahoo.es)

#### Keywords

Cocaine, Rhabdomyolysis, Creatinin-phosphokinase

Rhabdomyolysis is a condition resulting from the release of large quantities of myocyte breakdown products into the circulation, following injury to striated muscles. It is a clinical and biochemical syndrome, defined as an elevation of serum creatine kinase activity (sCK) to at least 10 times the normal upper limit followed by a rapid decrease of the sCK level back to normal values [1]. Other muscle enzymes in addition are elevated (aldolase, aminotransferases, lactate dehydrogenase), but it is not necessary to make the diagnosis. When massive amounts of myoglobin are released the binding capacity of the plasma protein is exceeded and it is filtered by the glomeruli, it may cause renal dysfunction.

The clinical presentation can vary widely: the classic triad (myalgia, weakness and pigmenturia) is seen in less than 10% of them. The aetiology is also quite diverse, including excessive muscular stress, direct toxic, physical damage and genetic defects. Many of the common abused drugs have been reported to cause rhabdomyolysis [1].

We present the case of a 36-year-old male patient, who did not have a previous medical history, and was admitted to the hospital because of myalgia and weakness. He admitted that he had inhaled 6 g of cocaine 12 h before admission denied the intake of any other drugs. The results of physical examination were normal. Blood analysis on admission disclosed a creatinine kinase (CK) 1,272 U/l, CK isoenzyme BM 54 U/L and myoglobin 1.4 mg/dL. The myoglobinuria was 0.6 mg/dL and his renal function was also normal. The patient improved with intravenous fluid plus sodium bicarbonate and his sCK was decreased to 230 U/l one day later, and then he was discharged.

The interest of this case is based on the fact that in this patient cocaine-induced acute rhabdomyolysis. The first case of rhabdomyolysis associated with acute cocaine intoxication in the medical literature was reported in 1987 [2]. About 24% of the cocaine users develop rhabdomyolysis [3], because this drug can cause a direct effect on the muscle tissue, inducing vasoconstriction and tissue ischemia. Cocaine-associated rhabdomyolysis may also contribute to hyperthermia and hyperactivity [4].

The goal of treatment of rhabdomyolysis is to cease muscle destruction (elimination of exposure), hydration and correction of electrolyte abnormalities [2]. An adequate volume of circulating blood is necessary to prevent acute renal kidneys. In the case of drug-induced rhabdomyolysis eliminating the exposure of the toxic may be the only treatment.

#### References

1. Curry SC, Chang D, Connor D (1989) Drug- and toxin-induced rhabdomyolysis. *Ann Emerg Med* 18: 1068-1084.
2. Herzlich BC, Arsura EL, Pagala M, Grob D (1988) Rhabdomyolysis related cocaine abuse. *Ann Intern Med* 109: 335-336.
3. Welch RD, Todd K, Krause GS (1991) Incidence of cocaine-associated rhabdomyolysis. *Ann Emerg Med* 20: 154-157.
4. Goldberg A (2015) Superimposed cocaine-induced rhabdomyolysis in a patient with aortic dissection rhabdomyolysis. *A A Case Rep* 4: 75-77.