Dear Sir,

Rhabdomyolysis (Rb) is a clinical and biochemical syndrome produced by the release of intracellular substances like CPK, LDH, GOT, aldolase, myoglobin and electrolytes (potassium, phosphorus). Its association with acute renal failure (ARF) [1] is well known and although its pathogenesis is unknown it is believed that it could be due to the toxic effects of the myoglobin or its derivatives like ferrihemoglobin [2] but in cases in which acidosis and dehydration [3] occur.

Among the ‘nontraumatic’ forms of Rb, the number of cases associated increases every day with drug abuse, so we want to inform people of a new case of Rb and ARF due to heroin poisoning but with special focus on the manner in which the drug is administered.

A 24-year-old male was admitted to the Emergency Ward having been in deep coma for approximately 12 h. At physical examination, the outstanding features were: BP: 140/70 mm Hg, HR: 130 beats/min, and breathing: 30 respirations/min. Deep coma (Glasgow scales score 4) and defective peripheral perfusion. The rest of the examination was normal. After orotracheal intubation, a cranial CAT was carried out in which a diffuse brain edema was found. The qualitative urine test for opiates was positive. Naloxone 0.8 mg i.v. were administered and he woke up in a state of agitation but without neurological focus. He remained in oliguria until the 9th day since his admission into hospital.

The most remarkable complementary features were: Hct: 54%, leukocytes: 30,000 with 87% of segments glucose: 101, uric acid: 16.3, creatinine: 16.7, urea: 430, Ca: 8, P: 8.4 mg/ dl, prothrombin activity: 68%, CPK: 28,000, GOT: 440 U/l, Na: 133, K: 4.6, CO3H: 16 mEq/l, myoglobin (at 5 days from admission into hospital): 776 ng/ml (n: 90 ng/ml), 1,25-dihydroxicholecalciferol: 15pg/ml(n: 18-78 pg/ml).

In the X-ray of the thorax, a right parahilar condensation was observed and abundant colonies of Staphylococcus aureus were isolated in the bronchial aspirate plating.

He needed extrarenal purification through conventional hemodialysis (3 sessions) and on the 9th day, he entered the polyuric phase. Subsequently, the patient revealed the kind of drug taken and the way of administration.

Even though the number of ‘nontraumatic’ Rb cases is high, the secondary processes due to heroin poisoning are rare and the drug is mainly inhaled [4,5]. Rb physiopathology in this kind

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of poisoning is multifac-torial but is mainly due to the direct toxic muscular effect of the drug [7] and/or myone-crosis caused by hypoxemia which results from the systematic effects of overdosage [2]. Two notable clinical facts about this patient are the excessive hyperuricemia probably as a consequence of the release of purines by the injured muscle [6] and the hypocalcemia due to the decrease in synthesis by 1,2-dihydroxy-cholecalciferol, hyperphosphatemia and a higher osseous resistance to PTH [7].

References

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