Skin Vasculitis, Hypokalemia and Acute Renal Failure in Rhabdomyolysis Associated with Cocaine

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Dear Sir,

Cocaine use today is an important public health problem. Several medical complications have been related to this drug [1]. One of the most recently described is rhabdomyolysis (RB) [2–4]. Unknown mechanisms act in this association, a toxic effect and/or secondary ischemia to the indirect sympathomimetic action of cocaine have been implicated [2]. Nevertheless other authors think that cocaine by itself is not causing RB [5].

We report a patient with acute renal failure and systemic alterations both secondary to RB which was temporally associated with cocaine use. During the evolution period he developed a prolonged hypokalemia and leu-kocytoclastic vasculitis.

A 34-year-old previously healthy man was admitted to hospital with agitation, myalgias, but without seizures. He had sniffed cocaine 8 h before and refused alcohol, amphetamine and heroin use. Physical exam showed temperature 36.5 °C, heart rate 102 beats/min, blood pressure 100/60 mm Hg, generalized muscle swelling, herpetic lesions on lips and left forearm. The rest of the physical examination was normal. The following laboratory values were disclosed: BUN 3.5 mmol/l, serum creatinine 230 µmol/l, CK 8,930 U/l, potassium 4.9 mmol/l, sodium 140 mmol/l, chloride 101 mmol/l, pH 6.92, pO₂ 75 mm Hg, pCO₂ 28 mm Hg, bicarbonate 5.5 mmol/l, hematocrit 35.2%, leukocytes 16,000/µl, platelets 75,000/µl, fibrinogen 2.4 g/l, prothrombin time 18 s, fibrin degradation products 500–1,000 ng/ml, calcium 1.27 mmol/l, phosphorus 1.58 mmol/l, uric acid 1,410 µmol/l, total bilirubin 47.6 µmol/l, ALAT 330 u/l, ASAT 1,560 u/l, alkaline phosphatase 122 u/l, LDH 5,630 u/l, aldolase 46 u/l. Urinalysis: sodium 33 mmol/l, potassium 57 mmol/l, blood + + +, numerous granular casts without red cells. EKG: sinus tachycardia. Abdominal ultrasound: normal kidneys.

During the first 12 h he had 3 diarrheic depositions and CK rose to 100,000 u/l. Kalemia was 2–3 mmol/l in the following 3 days; intraerythrocytic potassium determined by osmotic hemolysis was 80 mmol/l. He presented anuria and deterioration of renal function. A diagnosis of acute tubular necrosis due to RB was considered and hemodialysis was started. After the 4th day he developed palpable purpuric lesions on ankles and feet. The muscle biopsy was compat-

ible with RB, and the skin biopsy revealed leukocytoclastic vasculitis with IgG and C3 deposits over the vascular walls (fig. 1). Tests for rheumatoid factor, ANA, C3, C4 and HBsAg were negative or normal. Urine toxicologic screening was positive only for cocaine. The patient was discharged after 29 days, with biochemistry controls normalized.

Fig. 1. Skin biopsy showing a small dermal vessel with acute and chronic inflammatory infiltration and nuclear debris. HE. × 400.

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Hypokalemia is a well-known cause for RB. On the other hand, in RB caused by other etiologies long hypokalemia is a rare event, even though it can exist in the initial phase [6]. In this patient the intraerythrocytic potassium and the severe and long hypokalemia that required supplements suggest a previous potassium cell depletion. This depletion, favored in general by physical work in hot weather, was aggravated by the acute diarrhea and could have contributed to the RB.

Herpes virus infection could be another factor in this case [7].

Drug-induced vasculitis has been described in heroin and amphetamine use [8]. Skin infarction due to blood vessel spasm [9] and cerebral vasculitis [10] have been related to cocaine. It has been thought that vasculitis is caused by cocaine itself or by some contamination product. We are unaware of any previously described cocaine vasculitis similar to this case. New case descriptions would contribute to establish cocaine as a drug causing vasculitis.

There are two different acute renal failure patterns related to cocaine RB: isolated renal failure with a benign evolution, and renal failure associated with systemic complications, which has frequently a fatal course [3,11]. Although in this patient systemic alteration was present, evolution was satisfactory.

As pointed out by others [5], it seems to us that cocaine RB requires additional factors for its full clinical expression. In its absence, RB can take a subclinical course, explaining its relative low frequency, in contrast to the widespread cocaine availability.

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