

## Cocaine-induced acute renal failure without rhabdomyolysis

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### ABSTRACT

Several cases of acute renal failure (ARF) following cocaine abuse, all associated with rhabdomyolysis, have been reported. We report a case of ARF occurring secondary to cocaine abuse but without evidence of rhabdomyolysis. Literature on ARF without evidence of rhabdomyolysis following cocaine abuse is reviewed.

#### Key-Words:

Acute renal failure; cocaine; rhabdomyolysis.

### INTRODUCTION

Cocaine-induced acute renal failure (ARF) has been reported in association with rhabdomyolysis<sup>1-3</sup>. However, only a few cases of ARF without rhabdomyolysis has been described and those rarely<sup>4-6</sup>. We report a case of ARF which occurred after inhalation of cocaine without concomitant rhabdomyolysis.

### CASE REPORT

A 32 year-old man was referred to our hospital with deteriorating renal function. He admitted inhaling cocaine two days before admission and to a cocaine habit of three years' standing. However, he denied the intake of any other nephrotoxic drug. The last known serum creatinine level was 8mg/L, taken 12 months earlier. On admission his blood pressure was 180/100 mmHg, heart rate 72 beats per minute and he was afebrile. Blood analysis disclosed a serum creatinine

of 2mg/dl, creatinine-phosphokinase (CPK) 105 U/l, and potassium 3.8mmol/l. Urinalysis showed sodium of 30mmol/l. No protein or red blood cells were detected by dipstick. Haemoglobin and coagulation tests were normal. Myoglobulins were negative in blood and urine. Immunological studies showed normal levels of immunoglobulins, C<sub>3</sub> and C<sub>4</sub>. HIV, Hepatitis B, C and HCV were negative. Chest X-ray and electrocardiogram were unremarkable, ultrasound demonstrated normal-sized kidneys, and urine tested positive for cocaine. During the hospitalisation the patient's urine output remained at 1 to 1.5 L/d and his CPK within the normal range. Hypertension responded well to amlodipine. After a week, the patient had completely and spontaneously recovered from acute renal failure. His serum creatinine was 1.1mg/dl on discharge. Hypertension resolved as renal function improved.

### DISCUSSION

A wide spectrum of renal complications can occur with both acute and chronic use of cocaine<sup>1</sup>. ARF can occur as a result of rhabdomyolysis, and this is still the most common form of cocaine-induced renal pathology<sup>2,3</sup>. In contrast, cocaine-induced ARF occurred in the absence of rhabdomyolysis is rare, with only 3 previously reported cases (Table I).

In our patient, rhabdomyolysis could definitely be ruled out as the cause of ARF. The possibility that rhabdomyolysis developed before admission is extremely unlikely, because the half-life of serum creatinine kinase (CPK) is 17 hours. It is even longer in the presence of renal failure<sup>4,7</sup>. The patient was admitted 24 hours after inhalation of cocaine, and

**Table 1**

Report of the literature on cocaine induced ARF without rhabdomyolysis

Author, ref	Age	Sex	Time of ARF occurrence after cocaine use	Associated symptoms	Toxicology	Urine output	Histology	Time of resolution
Leblanc [4] 1994	16	F	3 days	None	–	1 – 1.5L/d	Normal appearance of kidney in biopsy	9 days
Amoedo [5] 1999	31	M	18 H	Hypertension	–	1 – 1.5L/d	–	10 days
Rivero [6] 2006	46	M	120 H	Elevated liver enzyme	Cocaine metabolites in urine	Normal	–	7 days
This case	32	M	48 H	Hypertension	–	1 – 1.5L/d	–	7 days

CPK was determined on admission and daily thereafter. Also laboratory tests were negative of myoglobulins in blood and urine.

The pathogenesis of ARF without evidence of rhabdomyolysis is unclear. Proposed mechanisms include intense intrarenal vasoconstriction and ischaemia<sup>4-6</sup>. Vasoconstriction caused by cocaine relates to the blockade of norepinephrine reuptake and to the release of adrenal catecholamines<sup>4,8</sup>. Ischaemia caused by intense intrarenal vasoconstriction result in medullary hypoxia and tubular dysfunction<sup>4</sup>. In our patient, the complete recovery of renal function argues that intense vasoconstriction may be the mechanism.

Cocaine has been associated with accelerated and malignant hypertension as well as implicated in hastening the progression of hypertensive nephrosclerosis to ESRD<sup>1,9,10</sup>. Cocaine blocks the presynaptic uptake of the neurotransmitters norepinephrine and dopamine. The resulting surplus of transmitters at the postsynaptic receptor sites activates sympathetic responses and thereby produces vasoconstriction and acute increases in heart rate and blood pressure<sup>1,11</sup>. We believe that in our patient hypertension was caused by an extension of the same effect.

In conclusion, this case adds to the previous case reports that ARF following cocaine abuse can supervene in the absence of concomitant rhabdomyolysis. Physicians should be aware of this feature of cocaine intoxication

**Conflict of interest statement.** None declared.

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