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Rhabdomyolysis with acute renal failure secondary to taking methadone

Nefrologia 2012;32(2):262-3
doi: 10.3265/Nefrologia.pre2011.Dec.11256

To the Editor,

Rhabdomyolysis is a disorder caused when damaged muscle releases toxic substances such as creatine kinase (CK) and myoglobin into the bloodstream. The main associated risk factors are alcoholism, lesions caused by compression, overexertion, heat intolerance, sunstroke, low phosphate levels, convulsions and drug use or overdose. Drugs commonly involved are cocaine, amphetamines, statins and heroin.

We present the case of a male patient aged 41 years who was transported to the emergency department due to reduced consciousness after falling at home. His medical history included hepatitis C, paranoid schizophrenia and habitual use of heroin. In the 72 hours prior to admission, he began to experience muscle weakness and widespread myalgia, which coincided with starting a methadone rehabilitation programme. In the 24 hours before admission, he also experienced headache, nausea, vomiting and a fever of 39.5ºC.

Physical examination showed drowsiness, lack of awareness of surroundings, hypotension (70/30mm Hg), 72% baseline oxygen saturation and signs of mucocutaneous dehydration. The examination revealed no other significant abnormalities. The blood count and biochemical test results were as follows: pH 7.10; PCO_2 23mm Hg; HCO_3 16mEq/l; haemoglobin 15.5mg/dl; leukocytes $23x10^3$/uL (88% neutrophils); C-reactive protein (CRP) 10mg/dl; creatinine 2.88mg/dl; potassium 6.6mEq/l; alanine aminotransferase 160IU/l; aspartate aminotransferase 523IU/l; phosphorus 7.0mg/dl; CPK 86 000IU/l. Neurological disorders were ruled out by a cranial CT and lumbar puncture. In subsequent hours, renal function deteriorated until serum creatinine reached 11.4mg/dl. Volume replacement therapy was initiated, which resulted in significant positive balances and urine production; renal function improved continuously over the following days. Haemodialysis was not required. Upon discharge, the patient's creatinine level was 2.1mg/dl, but he was lost to follow-up.

Acute renal failure is the most severe complication of rhabdomyolysis, whose prognosis depends on the degree to which renal function is compromised. It often presents with high CPK levels, hypercalcaemia, hyperphosphataemia and high anion gap metabolic acidosis. Our patient experienced all of these disorders. "Hard" drugs such as heroin and cocaine are classically associated with rhabdomyolysis. It is widely demonstrated that narcotics lead to rhabdomyolysis, which causes acute renal failure due to tubular obstruction arising when myoglobin leaks into the kidney.[1]

Mechanisms associated with the development of rhabdomyolysis in cases of drug abuse are decreased level of consciousness, coma or prolonged immobilisation being the main cause. Prolonged compression of muscles leads to ischaemia, which in turn triggers rhabdomyolysis. On the other hand, the direct manner in which methadone leads to rhabdomyolysis seems to arise from an increase in the muscle's demand for oxygen, which augments the state of muscle ischaemia.[1,2]

Most of the cases described are associated with abuse of multiple substances, such as heroin, cocaine, benzodiazepines or alcohol.[3,4] However, our case is an interesting one, since few articles describe rhabdomyolysis and acute renal failure caused by methadone abuse.[1,5]

Conflicts of interest

The authors affirm that they have no conflicts of interest related to the content of this article.

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Tubulo-interstitial nephritis and uveitis with Fanconi syndrome

Nefrologia 2012;32(2):263-4
doi:10.3265/Nefrologia.pre2011.Dec.11238

To the Editor,

Tubulointerstitial nephritis (TIN) with uveitis (TINU) is a rare disease whose pathogenesis is unknown.

We present the case of a boy 9 years of age examined for glycosuria. The patient had asthenia, anorexia and had lost more than 5kg body weight in the course of 1 month. No prior infections. The patient experienced nocturnal enuresis which was treated with desmopressin; that treatment was suspended 2 months before onset of the listed symptoms.

Examination revealed high blood pressure (maximum 135/97mm Hg), with other values being normal.

Blood analysis revealed renal dysfunction: creatinine (Cr) 1.2mg/dl; creatinine clearance 54ml/min/1.73 m^2; urea 51mg/dl; uric acid 2.1mg/dl; phosphorus 4.4mg/dl; mild metabolic acidosis with C-reactive protein 29mg/l and globular sedimentation velocity 62mm (Table 1).

Table 1. Laboratory blood and urine analyses at onset of symptoms and during syndrome progression

Patient evaluation	On admission	Day 10	Day 20	Day 60	Day 120	Day 210	Reference values
Biochemical tests							
Cr (mg/dl)	1.2	1.1	1.1	1	0.8	0.7	0.2-0.7
CrCl (ml/min/1.73m^2)	54	51	66	67	78	116	89-165
Urea (mg/dl)	51	36	62	44	38	33	21-50
Uric acid (mg/dl)	2.1	1.9	2	2.4	3.2	3.4	3.4-7
Glucose (mg/dl)	71	90	82	84	82	93	60-100
Phosphorus (mg/dl)	4.4	3.3	3.9	4.3	4.5	4.5	4.5-5.5
Calcium (mg/dl)	9.1	9.7	10	10.3	9.9	10.4	8.8-10.8
Proteins (g/dl)	7.5	8.5	8.3	7.8	7.2	7.2	6-8
Sodium (mEq/l)	136	141	139	139	142	142	135-145
Potassium (mEq/l)	4.3	4.6	4.4	4.8	4.8	4.8	3.5-5
Cl (mEq/l)	102	115	111	107	107	108	101-111
pH	7.3	7.26	7.3			7.33	7.36-7.4
pCO_2	36	43	39			40.3	40-45
Bicarbonate (mmol/l)	17.5	19	19			21.1	22-26
EB	-6.1	-7.2	-6			-3,9	-2-3
Urine							
pH	6	6.5	7		6.5		5-7.5
Proteins (g/l)	1.2	0.7	0.3	0.12	0.06	0.05	0.01-0.14
Prot/Cr	0.94	0.7	0.63	0.27	0.12	0.08	<0.2
µalb/Cr (mg/g Cr)	148	107	99	42	<6	<6	0-30
Glucose (g/l)	0.8	1	1	0.3	0	0	0-0.5
TPR (%)	78	85	87	88	89	92	90-95
FEUA(%)	48	31	33	17			<10%
UrEl (mg/100 GF)	0.63	0.60	0.67	0.56	0.42	0.41	0.2-0.42
Proteinuria (mg/kg/day)	13	9.7	11	4.4	2.3	1.1	<4

µalb: microalbuminuria; CrCl: creatinine clearance; Cr: creatinine; EB: excess of bases; FEUA: fractional excretion of uric acid; UrEl: Uric acid excretion index; Prot/Cr: protein/creatinine index; TPR: tubular phosphate reabsorption.

The urine analysis showed glycosuria (0.8g/l); non-nephrotic range proteinuria (13mg/kg/day, microalbuminuria 148mg/g Cr); hyperphosphaturia with low tubular phosphate reabsorption (78%); hyperuricosuria with a high uric acid index (0.78/100ml glomerular filtration); normal glomerular filtration and calcium levels (1.1mg/kg/day); fractional excretion of sodium 0.3%, and fractional excretion of potassium 19%. Urinary sediment showed granular and hyaline casts, leukocyturia without eosinophils and a negative urine culture.

These findings are compatible with multiple dysfunctions of the proximal tubule (PT), or Fanconi syndrome.

Liver, thyroid and parathyroid functions were normal. The immunological study revealed low-level positivity for antinuclear antibodies (1/80). The antistreptolysin O (ASLO) titre was high (2175IU/ml) with a negative oropharyngeal culture. Immunoglobulin levels were normal, but IgG was high 1 week later (1820mg/dl). An IgM test was run for Epstein-Barr, cytomegalovirus, parvovirus B19, toxoplasmosis and hepatitis B and C. All were negative. Renal ultrasound was normal.

Given the PT dysfunction and mild renal failure, we suspected a case of TIN. We adopted a watchful waiting approach and kidney function and blood