NDT Plus (2011) 4: 173–174
doi: 10.1093/ndtplus/sfr045
Advance Access publication 4 April 2011

Case Report

NDT PLUS
Nephrology Dialysis Transplantation

An unusual case of rhabdomyolysis

Gianni Bellomo[1], Giorgio Gentili[2], Claudio Verdura[1], Giuseppe Calabrò[2] and Maria Luisa Miele[2]

[1]Department of Nephrology, San Giovanni Battista Hospital, Foligno (Pg), Italy and [2]Department of Emergency Medicine, San Giovanni Battista Hospital, Foligno (Pg), Italy

Correspondence and offprint requests to: Gianni Bellomo; E-mail: bellomopg@yahoo.it

Abstract
We report an unusual case of rhabdomyolysis due to coturnism (food poisoning caused by eating quails). The patient's clinical course is described, and possible pathogenetic mechanisms of this syndrome are briefly discussed.

Keywords: coturnism; quail; rhabdomyolysis

Background

Rhabdomyolysis is a syndrome in which skeletal muscle destruction results in the release of large quantities of toxic muscle cell components into the plasma, which can lead to acute renal injury. The etiology of skeletal muscle injury is quite diverse, including excessive muscular stress and ischaemia, genetic defects, hypo/hyperthermia, electrolyte disturbances and direct toxic or physical damage [1]. We report here a case of acute rhabdomyolysis due to an unusual cause.

Case report

A previously healthy 54-year-old white man, with no family history of kidney disease, came to our attention with a 4-h history of excruciating chest pain, irradiated to both upper limbs and, later, to the thighs, associated with tremor; pain had begun 2 h after consuming a meal, shared with other family members, which included roasted quails which had been shot in a rural area the end of September, (2 months earlier) and stored in a deep freezer; he was not taking any medications and denied having had fever, traumas or having done strenuous exercise, but admitted being a heavy smoker (~25 cigarettes/day). His past medical history included only a diagnosis of borderline hypertension, made 1 year earlier, treated with a low-sodium diet alone. On admission, physical exam was unrevealing, except for proximal muscle tenderness on palpation; his blood pressure was 210/130 mmHg, oxygen saturation 97% in ambient air and his body temperature was 36.6°C. An electrocardiogram showed sinus tachycardia with no other abnormalities; in order to rule out aortic dissection, a computed tomography scan of the thorax and abdomen was performed, which showed mild chronic lung disease, cysts of the liver and both kidneys and no evidence of aortic dissection. Lab testing on admission demonstrated signs of severe rhabdomyolysis, with preserved renal function. Table 1 displays the time course of muscle enzyme levels, serum creatinine and other relevant parameters. A toxicology screen for substances of abuse was negative on admission. HIV, as well as B and C hepatitis markers were negative. The patient's urine (pH 6.5) was reddish brown and tested strongly positive (+++) at the benzidine test; mild proteinuria (2+ dipstick) was also present, whereas the urine sediment was unremarkable. The patient was immediately hydrated intravenously (3500 mL normal saline/24 h); his symptoms subsided within 24 h, the urine cleared after 48 h and he was dismissed on the fourth day, after an uneventful hospital course. The patient was again seen 1 week later, and all lab tests (including renal function and muscle enzymes) had returned to normal.

Discussion

As other causes of rhabdomyolysis were excluded, we concluded that our patients' symptoms were caused by food poisoning due to the consumption of quails, in other words, coturnism.

The syndrome of food poisoning by the European migratory quail (*coturnix coturnix*, hence the name coturnism) has been known for centuries and was first described in the Bible [2]. It has been observed in the Mediterranean region, the Middle East, North Africa, southern France, the Sinai Peninsula and Greece [3, 4]. The true incidence of coturnism is unknown, however, as many cases probably remain undiagnosed. Clinical manifestations include pain in the muscles previously exerted during physical activity, muscular cramps and autonomic dysfunction, occurring shortly (1.5–10 h) after the consumption of quail and usually lasting for 24–48 h. Laboratory tests generally show increased levels of serum muscle enzymes, which usually

Table 1. Time course of lab parameters

Parameter (units, reference values)	On admission	6 h	24 h	48 h	96 h
Creatine kinase (U/L, 55–170)	18 600	22 143	11 701	4723	438
Aspartate aminotransferase (U/L, <41)	551	586	438	295	89
Alanine aminotransferase (U/L, <64)	244	202	195	191	85
Myoglobin (ng/mL, 17–105)	3898	2851	615	188	74
Lactate dehydrogenase (U/L, 240–480)	1298	1772	1916	1663	1338
Creatinine (mg/dL, 0.7–1.3)	0.81	1.21	1.28	0.95	0.79
Potassium (mmol/L, 3.6–5.0)	3.9	4.4	4.1	4.1	4.0

return to normal within 1–2 weeks, and myoglobinuria, which is responsible for the reddish-brown discoloration of the urine. Coturnism, following the precipitation of myoglobin in the renal tubules, may be complicated by shock and acute renal failure requiring dialysis, which in our case was prevented by the prompt initiation of therapy (only a transient, mild rise of serum creatinine was observed).

Regarding the pathogenesis of coturnism, it has been suggested that quail toxicity be caused by their feeding on poisonous plants, such as hemlock (conium maculatum) that contains coniine, a toxic alkaloid; coniine can be lethal in a dose of 150 mg; but in smaller doses, it produces neurotoxic effects, acute rhabdomyolysis and acute renal failure [3, 4]. However, coniine triggers clinical manifestations only in susceptible individuals; in fact, our patient was the only one affected, though having shared his meal with family members who did not report any disturbances. He may have been rendered more susceptible to quail toxicity by his heavy smoking habit or by a muscle genetic abnormality: in this respect an association between coturnism and calpain 3 (a striated muscle protease) deficiency has been recently reported [5]. Nonetheless, the genetic, epidemiological and biochemical features of coturnism have not yet been elucidated in full, and firm experimental data remain to be documented.

In conclusion, we believe this case report teaches us that when confronted with unexplained rhabdomyolysis, coturnism must also be taken into consideration.

Conflict of interest statement: None declared.

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Received for publication: 4.2.11; Accepted in revised form: 7.3.11