

ACUTE RHABDOMYOLYSIS FOLLOWING QUAIL CONSUMPTION

Ioannis S. Papanikolaou, MD; Spyros P. Dourakis, MD;
Vassilios S. Papadimitropoulos, MD; Stefanos J. Hadziyannis, PhD

The syndrome of acute rhabdomyolysis develops when damage to striated muscles occurs. Muscle damage is usually attributed to toxic, ischemic, infectious, inflammatory or metabolic insults, as well as to direct muscle injury.¹ A rarely reported cause of this syndrome is food poisoning due to the consumption of the European migratory quail (coturnism).^{2,3} We report the case of a 60-year-old male who was admitted to hospital due to acute rhabdomyolysis occurring a few hours after quail consumption.

Case Report

A 60-year-old man, having experienced muscular pain in the lumbar region and extremities for 6 hours, was admitted to hospital. The patient reported no exercise or trauma and was afebrile both on and before admission. He reported that 6 hours prior to the onset of symptoms, he had consumed quail, which had been shot in a rural area near Athens the previous autumn (5 months earlier) and stored in a deep freezer. The patient, a resident of the Athens district, had had an 80-pack per year history of tobacco smoking, but had abstained from smoking during the previous 8 months, and he denied any alcohol abuse. His family history was unremarkable. Except for tenderness of the proximal muscles of the extremities, physical examination was unrevealing. Laboratory values included an elevated white blood cell count at 19,230/mm³ (with a differential count of 91% neutrophils, 6% lymphocytes and 6% monocytes). Serum lactate dehydrogenase was 741 IU/L (normal 120-230), creatine kinase 35,650 IU/L (normal 25-190), creatine kinase-MB isoenzyme 514 IU/L (normal <12% of total creatine kinase), aspartate aminotransferase 770 IU/L (normal 10-40), alanine aminotransferase 209 IU/L (normal 10-40), fibrinogen 510 mg/dL (normal 200-400), serum aldolase 56 IU/L (normal 0-8) and serum and urine myoglobin 4500 µg/L (normal 0-70) and 35,100 µg/L (normal 0-70), respectively. Serum protein electrophoresis demonstrated a slight elevation of α₂-globulin. All other results, including complete blood count, blood chemistry,

coagulation studies, erythrocyte sedimentation rate, C-reactive protein, serum haptoglobin, serum immunoglobulin levels, serologic tests for HIV, *Toxoplasma gondii*, cytomegalovirus, herpes simplex virus type 1 and 2, Epstein-Barr virus and trichinosis, were negative or normal. Arterial blood gas values were normal. The urine was a reddish-brown color and urinalysis revealed proteinuria (>300 mg/dL). The benzidine test for hemoglobin was positive (++++) and the sediment was otherwise normal. Chest x-ray and electrocardiograph were normal and abdominal ultrasonography was unremarkable. Based on a review of the patient's history, clinical findings and laboratory abnormalities, a diagnosis of acute rhabdomyolysis, caused by the consumption of quail, was made. The patient was treated symptomatically with normal saline intravenous administration (3 liters daily) and urine alkalization. The latter was achieved with intravenous administration of sodium bicarbonate, as well as orally administered acetazolamide tablets and was documented with daily measurements of urine pH. These measures resulted in the disappearance of muscular pain and the normalization of urine color on the 2nd day of hospitalization; muscle enzymes gradually decreased to normal levels over a period of 21 days.

Discussion

Acute rhabdomyolysis is a syndrome resulting from damage to striated muscle, usually due to toxic, ischemic, infectious, inflammatory or metabolic insults, as well as to direct muscle injury.¹ A rarely reported cause of this syndrome is food poisoning due to the consumption of the European migratory quail (coturnism).² Coturnism has been known since ancient times and was first described in the Bible.¹⁻³ It has been observed in the Mediterranean region and the Middle East, especially Algeria, North Africa, southern France, the Sinai peninsula and Greece.²⁻⁴ The true extent of the syndrome is unknown, however, as many cases probably remain undiagnosed. Clinical manifestations include pain in muscles previously exerted during physical activity, muscular cramps and autonomic dysfunction occurring shortly (1.5-10 hours) after the consumption of quail and usually lasting 1-2 days.² Laboratory tests reveal increased levels in serum muscle enzymes, which usually return to normal values within 1-2 weeks, and

From the Academic Department of Medicine, Hippokraton General Hospital, University of Athens, Greece.

Address reprint requests and correspondence to Dr. Dourakis: 28 Achaia St., GR 115 23, Athens, Greece.

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myoglobinuria, which is responsible for the reddish-brown discoloration of urine.² Due to myoglobinuria, coturnism may be complicated by shock and acute renal failure; the latter may be extremely severe, necessitating dialysis.³

Our patient presented clinical and laboratory manifestations of acute rhabdomyolysis 6 hours following the ingestion of quail. The usual causes of rhabdomyolysis were excluded. It has been suggested that a toxic effect (perhaps the previous consumption of hemlock seeds by the quail) or a genetic sensitivity (a hereditary enzyme deficiency) may be the pathogenic basis of coturnism.² However, Kennedy and Grivetti⁴ have convincingly ruled out the previously widely held view concerning the toxic alkaloid conine, contained in hemlock seeds consumed by the quail, as the responsible pathogenic factor. On the other hand, our patient's own and his family's history were unremarkable with regard to this syndrome; also, his origin was completely unrelated to the island of Lesvos (the only place in Greece where coturnism has been known to occur).^{2,3} The genetic, epidemiological and biochemical

characteristics of coturnism have not yet been elucidated and firm experimental data remain to be documented.^{2,5}

Our case exemplifies that coturnism is a rare cause of acute rhabdomyolysis, but with a good prognosis, provided there is timely recognition and treatment, thus obviating severe complications.

References

1. Gabow P, Kaehny W, Kelleher S. The spectrum of rhabdomyolysis. *Medicine* 1982;61:141-52.
2. Papadimitriou A, Hadjigeorgiou GM, Tsairis P, Papadimitriou E, Ouzounelli C, Ouzounellis T. Myoglobinuria due to quail poisoning. *Eur Neurol* 1996;36:142-5.
3. Billis AG, Kastanakis S, Giamarellou H, Daikos GK. Acute renal failure after a meal of quail (letter). *Lancet* 1971;ii:702.
4. Kennedy WB, Grivetti EL. Toxic quail: a cultural-etiological investigation of coturnism. *Ecol Food Nutr* 1980;9:15-42.
5. Papapetropoulos T, Hadziyannis SJ, Ouzounellis T. On the pathogenic mechanism of quail myopathy. *JAMA* 1980;244:2263-4.