Salmonella Group C Enteritis Complicated by Rhabdomyolysis

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Rhabdomyolysis, a potentially lethal clinical syndrome [1], is characterized both by elevated serum concentration of muscle enzymes (creatine kinase, aminotransferases) and symptoms of myalgia and weakness. Sometimes myoglobinuria is also present [2].

In addition to crush injuries, prolonged immobility, exposure hypothermia, generalized seizures and drug intoxication, infectious agents, most commonly viral or bacterial, are also associated with rhabdomyolysis. In 1996, Singh et al. [3] reviewed the viral and bacterial causes of rhabdomyolysis. Influenza virus and *Streptococcus pneumoniae* were the leading etiologic agents for rhabdomyolysis among viral and bacterial causes, respectively. *Salmonella* infection with *Salmonella enteritidis*, *S. bonariensis*, *S. thyphi*, *S. typhimurium* and 09 serotype was also described in patients with rhabdomyolysis [4]. Only one case report described rhabdomyolysis in *Salmonella* group C infection [5]. Here we report the second case of this rare complication.

PATIENT DESCRIPTION

A 24 year old man presented to our center with a chief complaint of watery diarrhea. Two days before admission he had suffered from abdominal pain and high fever. Gradually he also noticed liquid stool, anorexia, myalgia and weakness. The stool was without blood, mucus or pus. The patient denied receiving medications, having contact with a sick person, travelling to an exotic place, trauma or intramuscular injections. He had eaten in a restaurant 3 days previously and was otherwise healthy without self or family history of any myopathies.

On admission to the emergency department the patient presented with fever (39.4°C) and tachycardia (heart rate 120/min). His blood pressure was 140/70 and clinical examination revealed dehydration and mild diffuse tenderness in the abdomen. The rest of the physical examination was unremarkable.

Laboratory studies disclosed the following significant data: serum sodium 136 mEq/L (136–145), potassium 3.7 mEq/L (3.5–5), chloride 94 mEq/L (98–106), bicarbonate 21.1 mEq/L (21–30), blood urea nitrogen 47.8 mg/dl (2.5–8), creatinine 1.06 mg/dl (< 1.5), white blood cells 5920/ml (4500–11,000/ml), hematocrit 45% (41–53%), platelets 133,000/ml (150,000–350,000/ml), CK 24,073 U/L (60–400), lactate dehydrogenase 900 U/L (100–190), aspartate aminotransferase 341 U/L (0–35), alanine aminotransferase 85 U/L (0–35), alkaline phosphatase 79 U/L (30–120), prothrombin time 19 seconds (11–13 sec), and partial thromboplastin time 44 sec (25–38 sec). Myoglobin was absent in the urine and chest X-ray was normal. Blood serology studies for hepatitis A, B, and C, Epstein Barr virus and cytomegalovirus, performed because of elevated liver enzymes, were negative.

CK = creatine kinase

Stool cultures were drawn, and empiric antibiotic treatment with ofloxacin at a dose of 200 mg twice daily was initiated. The patient was also treated with intravenous saline and bicarbonate. Two days after his hospitalization stool cultures grew *Salmonella* group C, which was sensitive to ceftriaxone, ciprofloxacin, ofloxacin and sulfamethoxazole/trimethoprim. Ofloxacin was continued for a total of 14 days. During his hospitalization the patient's symptoms subsided and 12 days after admission CK levels and other blood studies were normal.

COMMENT

Salmonella constitutes a large genus within the family Enterobacteriaceae. The growth of *S. typhi* and *S. paratyphi* is restricted to human hosts, where these organisms cause typhoid fever. The remaining serotypes, named non-typhoidal *Salmonella*, can colonize the gastrointestinal tracts of a broad range of animals including mammals. In humans they often cause gastroenteritis. Sometimes also localized infection and/or bacteremia can be observed. Clinical laboratories initially divide *Salmonella* into serogroups (A, B, C, D, and E) based on reactivity to somatic O-antigen antisera (lipopolysaccharide cell-wall components). Our patient was infected with *Salmonella* group C, which is non-typhoidal *Salmonella*.

Infections account for 5% of the causes of rhabdomyolysis. *Salmonella* infection is an infrequently reported cause of rhabdomyolysis. Nevertheless, approximately 30 cases of rhabdomyolysis associated with *Salmonella* have been reported to

date. In adults, 20 cases were reported: 10 were caused by non-typhoidal *Salmonella*, and only one by *Salmonella* group C [5]. The remaining 10 cases were caused by typhoid fever. In children six cases have been reported, all from Japan. Among 60 bacteria-related cases in the English literature, *Salmonella* infections accounted for six cases [3]. The age range was 32–84 years and the range of CK levels was 1870–24,360 U/L. Four of six patients had acute renal failure, and there were no deaths [3]. Blanco and co-authors [1] analyzed 52 cases of rhabdomyolysis and found that infections were the cause of rhabdomyolysis in 31%. The main microorganism implicated was Gram-negative. They also found that infectious rhabdomyolysis was associated with a higher morbidity but not with a higher risk of death [1].

Although the exact mechanism of the rhabdomyolysis that occurs during the course of salmonellosis awaits complete elucidation, some pathogenic considerations have been suggested. A decrease in the activities of oxidative and glycolytic enzymes of skeletal muscle together with simultaneous activations of several lysosomal enzymes in rats infected by *Salmonella typhimurium* have been described. An endotoxin effect on muscle enzymes may be present in *Salmonella* infection. Other mechanisms that relate to muscle cell injury were suggested, including direct bacterial invasion, hypoxia, dehydration, acidosis, electrolyte disturbances, and hypophosphatemia. Some rhadomyolysis cases are secondary to a muscle genetic defect. In theses cases *Salmonella* infection could be the trigger.

Acute renal failure complicates cases of severe rhabdomyolysis, and sometimes dialysis is needed; when volume depletion from severe gastroenteritis is present, the risk of acute renal failure is even higher. On the other hand, even without the presence of rhabdomyolysis the incidence of renal dysfunction accompanying *Salmonella* infection is fivefold higher than in patients with gastroenteritis caused by other pathogens. Therefore, it is important to remember that rhabdomyolysis can be an extra-intestinal manifestation of salmonellosis. The serum concentration of muscular enzymes should be measured and, if elevated, appropriate treatment should be given, thereby reducing the chance of acute renal failure.

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