Rhabdomyolysis may be related to many causes, including alcoholism, use of toxic substances, crush injury, seizures, and infections. Bacterial and viral infections, even without direct muscle invasion, account for only 5% of rhabdomyolysis [1]. Of the bacterial pathogens associated with rhabdomyolysis, Legionella pneumophila is the most common while Streptococcus pneumoniae has rarely been reported as causative. We report the case of a patient with rhabdomyolysis associated with S. pneumoniae bacteremia who had undergone splenectomy but had not received pneumococcal vaccination afterwards.

**Case Report**

A 31-year-old male taxi driver was admitted because of flu-like symptoms beginning 1 week earlier with watery diarrhea, chills, and shortness of breath in the night prior to admission. Several bouts of watery diarrhea, chills, and non-projectile vomiting with dark and bily vomitus developed after eating some barbecued foods at dinner 1 day before hospitalization. Progressive shortness of breath and oliguria ensued.

He had been well except for a splenectomy performed after a traffic accident 3 years before this admission. The post-splenectomy course had been smooth. Pneumococcal vaccination was not administered when he was followed as an outpatient. He smoked half a pack of cigarettes per day and drank one bottle of beer every other day for several years. He denied recent travel and a history of drug or food allergy.

At the emergency room, his temperature was 38.8°C, respiratory rate 24, oxygen saturation 90% while breathing ambient air, heart rate 140 beats per minute, and blood pressure 105/70 mmHg. His conjunctiva were not pale, sclera were not icteric, and pupils were isocoric with prompt light reflex. There was no ulcer in the oral cavity and the neck was supple without lymphadenopathy or jugular vein engorgement. Chest wall expansion was symmetric and breath sounds were clear without rales or crackles. Heart sounds were regular without murmurs. The abdomen was soft and mildly distended without tenderness or rebound tenderness. A median surgical scar was noted. The liver was not palpable. The bowel sound was hypoactive. His extremities were cyanotic but freely movable without edema. White blood cell count (WBC) was 38,540/µL with band form of 21% and segment 73%. Blood urea nitrogen was 21 mg/dL, creatinine 6.0 mg/dL, creatine phosphokinase (CPK) 5,331 U/L, lactate dehydrogenase 1,733 U/L, and aspartate transaminase 149 IU/L. Arterial blood gas analysis showed pH 7.37, paO2 143 mmHg, pCO2 21 mmHg, HCO3⁻ 12.5 mmol/L, and a base excess of –9.7 mmol/L. Urinalysis showed pH 6.0, protein 1+, occult blood 3+, and WBC 4 to 5, red blood cell count (RBC)

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5 to 8, and epithelial cells 2 to 3 per high power field in the sediment. There was no pneumonic infiltrate on chest roentgenogram.

After hospitalization, antibacterial therapy with ceftriaxone, oxacillin, and metronidazole was initiated. He was intubated because of impending respiratory failure on the second hospital day. Antibiotics were switched to ampicillin-sulbactam and ciprofloxacin on the third hospital day when preliminary blood cultures yielded gram-positive cocci and fever subsided thereafter. Blood culture results showed S. pneumoniae, which was resistant to penicillin, erythromycin, and tetracycline by the disk diffusion method. The isolate was identified as serotype 23F by the capsular swelling method. Antisera were obtained from the Statens Serum Institut in Copenhagen, Denmark [2]. Abdominal sonogram was suggestive of parenchymal liver disease without ascites or a distended gallbladder. Because of the lack of evident infection foci, computerized tomography of the head was performed and revealed fluid collection in the left maxillary and sphenoid sinuses with mucosal thickening in the left ethmoid sinuses. Rhabdomyolysis with acute renal failure was diagnosed and hemodialysis was started because renal function did not improve after hydration and alkalization of urine. Peak CPK concentration was 28,635 U/L on the fifth hospital day, which declined rapidly after initiation of hemodialysis. The frequency of hemodialysis decreased gradually and no dialysis was needed on the tenth hospital day. The patient was discharged on the twenty-seventh hospital day. There was no further fluid accumulation or mucosal thickening on Water’s view of skull roentgenograms just before discharge. The serum creatinine concentration declined to 2.3 mg/dL on discharge and 1.3 mg/dL during follow-up on an outpatient basis.

**Discussion**

This case demonstrates the rare association of pneumococcal bacteremia and paranasal sinusitis with rhabdomyolysis and myoglobinuric renal failure. The only other possible cause of the rhabdomyolysis in this case was ethanol use, since the patient had a history of consuming one bottle of beer every other day. The spectrum of alcohol-induced myopathy has been well characterized [3]. Acute alcoholic rhabdomyolysis usually occurs after binge drinking and is characterized by the abrupt onset of intense myalgia and swelling, most prominent in lower extremities. The clinical presentations in this case were not consistent with those of acute alcoholic myopathy, in that there was no history of binge drinking, edema, or myalgia. Rather, the clinical presentations were predominately those of a paranasal sinusitis infection and pneumococcal bacteremia. Therefore, the pneumococcal infection might have played a role in the development of the skeletal muscle injury in this case.

The exact mechanism of rhabdomyolysis associated with invasive pneumococcal infections is unclear. A number of other bacterial infections have occasionally been reported in association with rhabdomyolysis, although their role is not clear [4]. Animal studies have shown that streptolysin S and extracellular products of streptococci are toxic to skeletal muscle [5], but a direct role of pneumococci in the causation of rhabdomyolysis has not been demonstrated. There is evidence that skeletal muscle metabolism is altered in the presence of pneumococcal infections in rats [6]. Therefore, it is possible that products of the pneumococci and disruptions of the energy production pathways in skeletal muscle may be important in the development of muscle injury.

Although only about 0.5% of sinusitis is complicated with clinically evident acute bacterial sinusitis, S. pneumoniae is the most common pathogen in adults [7]. The persistence of flu-like symptoms for more than 7 to 10 days in this case is compatible with clinical features of bacterial sinusitis. Splenectomy may put patients at risk for overwhelming infections especially by encapsulated organisms, such as S. pneumoniae, in about 60 to 70% of cases [8]. Our patient had this typical picture, as reported by Hroncich and Rudinger [9]. About 2.5% of splenectomized patients die later from fulminant bacterial infections because of deficient clearance of bacteria that may result from reduced phagocytosis, decreased immunoglobulin M (IgM) production, disturbances of the complement system, and lack of tuftsin. The Advisory Committee on Immunization Practices (ACIP) recommends that pneumococcal vaccination should be administered at least 2 weeks before elective splenectomy or as soon as this condition is identified, repeated every 3 to 5 years, depending on age and medical condition. Amoxicillin-clavulanic acid, trimethoprim-sulfamethoxazole, or cefuroxime axetil prophylaxis should be taken at the first sign of infection [10]. However, others argue that these measurements do not eliminate the risk for invasive pneumococcal infections [11, 12]. Our patient had neither early vaccination after splenectomy nor antibiotic prophylaxis at the first sign of minor illness. This was probably why he developed such a fulminating course.

This report demonstrated that infection with S. pneumoniae may precipitate rhabdomyolysis and acute renal failure. As Spataro and Marone reported [13], rhabdomyolysis associated with pneumococcal infection may be underreported because it is usually asymptomatic and self-limited, but it may be fatal in immunocompromised patients. While early diagnosis and aggressive treatment may decrease infectious morbidity and improve survival in splenectomized patients, the need for prevention against invasive pneu-
mococcal infections with vaccination should be emphasized.

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