Mod Rheumatol (2004) 14:422–423
DOI 10.1007/s10165-004-0335-0

Letter

Rhabdomyolysis and acute renal failure in a polymyositis patient

Chih-Neng Tsai · Ming-Fei Liu · Thy-Sheng Lin · Li-Hsin Lin · Chrong-Reen Wang

Key words Acute renal failure · Polymyositis · Rhabdomyolysis

To the Editor:

A 40-year-old woman was admitted to the National Cheng Kung University Hospital because of poor appetite and progressive weakness that had lasted 1 month. Her temperature was 37°C, pulse rate 84/min, and blood pressure 108/62 mmHg. On physical examination, there was pitting edema of the extremities. Muscle power was 3/5 over upper extremities and 2/5 over lower extremities. Other findings were unremarkable. The hemogram analysis showed leukocytosis of 14 400/μl and a normal hemoglobin level and platelet count. Blood chemistry tests showed urea nitrogen 31 mg/dl, creatinine 1.0 mg/dl, aspartate aminotransferase 1848 IU/l, alanine aminotransferase 1016 IU/l, sodium 123 mmol/l, potassium 4.7 mmol/l, calcium 6.7 mg/dl, phosphate 4.4 mg/dl, and albumin 1.9 g/dl. The urine dipstick test showed a positive reaction for blood, but no erythrocytes were found during the microscopic examination.

Intravenous albumin and furosemide were started, but the general weakness progressed and tea-colored urine was noted. Tracing her history, the patient noted that she had had pain over the extremities 2 months ago and had not been able to elevate her upper extremities for 1 month. Extremely elevated creatine kinase (31 427 IU/l) was found. Her serum myoglobin was 22 216 μg/l and the urine myoglobin 202 μg/l.

C.-N. Tsai · M.-F. Liu · L.-H. Lin · C.-R. Wang (✉)
Sections of Rheumatology and of Allergy and Immunology, Department of Internal Medicine, Medical College, National Cheng Kung University, No. 1 University Road, Tainan, Taiwan
Tel. +886-6-235-3535 (ext. 5366); Fax +886-6-276-6175
e-mail: wangcr@mail.ncku.edu.tw

T.-S. Lin
Department of Neurology, Medical College, National Cheng Kung University, Tainan, Taiwan

Oliguria developed, and the serum creatinine level became elevated. Rhabdomyolysis was noted, and an intravenous fluid supplement and alkalization were prescribed. Muscle power weakness was more obvious proximally than distally. Polymyositis was suspected, and 100 mg of intravenous hydrocortisone was administered every 8 h.

Muscle biopsy revealed degeneration and necrosis of muscle fibers, as shown in Fig. 1. The fibers had become pale and vacuolated and were undergoing phagocytosis by multinucleated giant cells. There were mononuclear cells infiltrating the perimysial and endomysial space. No evidence of vascular inflammation was found. Polymyositis was diagnosed. Examination of the autoantibody profile revealed positive anti-Jo-1 antibody. Her renal function deteriorated progressively, followed by acute pulmonary edema. The trachea was intubated with ventilator support, and she was transferred to the intensive care unit (ICU) for further treatment. Emergent hemodialysis was performed for acute renal failure and pulmonary edema. Hemodialysis was carried out regularly for 3 weeks and then discontinued owing to improved renal function and increased urine output. The muscle enzyme level decreased under high-dose corticosteroid treatment, but muscle power did not improve. After transfer to a general ward, she suffered from respiratory failure after an episode of choking. She was intubated and transferred to the ICU again. The treatment course that followed was complicated with repeated infection, and she succumbed to sepsis on the 52nd day of hospitalization.

Polymyositis belongs to the category of idiopathic inflammatory myopathy, with extramuscular manifestations such as fever, arthritis, Raynaud's phenomenon, gastrointestinal dysfunction, cardiac disturbance, and pulmonary involvement. The kidneys are spared in this disease. Acute renal failure occurs in rhabdomyolysis patients and is associated with high morbidity and mortality rates. Intravenous hydration should be initiated as early as possible to decrease the rate of oliguric or anuric renal failure. The etiology of rhabdomyolysis includes drugs, trauma, heat-related causes, ischemia, exertional causes, infection,

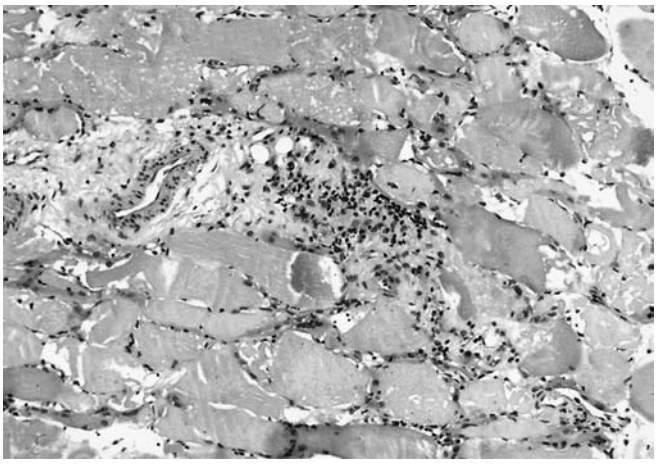

Fig. 1. Mononuclear inflammatory reaction is present in the perimysial and endomysial space. Muscle fibers show vacuolated degeneration and necrosis. (H&E, ×200)

metabolic causes, endocrinologic causes, and inflammatory causes including polymyositis.[1]

Rhabdomyolysis is rare in patients with polymyositis.[2–4] To the best of our knowledge, our patient is the first Chinese individual with this background reported in the literature. The manifestation of rhabdomyolysis is a life-threatening, uncommon condition in polymyositis patients. The current established approach to the recognition and treatment combined with refinement in dialysis can lead to complete, uncomplicated recovery of these patients. Although rhabdomyolysis is a rare manifestation of polymyositis, clinicians should be alert to the differential diagnosis of this complication.

References

1. Sauret JM, Marinides G, Wang GK. Rhabdomyolysis. Am Fam Physician 2002;65;907–12.
2. Thakur V, Desalvo J, Mcgrath H Jr, Weed S, Garcia C. Polymyositis-induced myoglobinuric acute renal failure. Am J Med Sci 1996;312;85–7.
3. Carleton H, Pitts W, Davidson W, Roth P. Muscle disease associated with renal failure. Arch Intern Med 1977;137:939–41.
4. Pirovino M, Neff MS, Sharon E. Myoglobinuria and acute renal failure with acute polymyositis. NY State J Med 1979;79:764–7.