

Acute Renal Failure Associated with Dermatomyositis and Colon Cancer

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Dear Sir,

Dermatomyositis is a collagen disease characterized by a nonpurulent polymyositis, often accompanied by dermatitis and localized edema. When skin lesions are absent, the condition is referred to as polymyositis. The kidneys are generally spared in dermatomyositis. A few cases of acute renal failure in dermatomyositis have been reported [1–4]. This report describes a case of acute renal failure associated with dermatomyositis and colon cancer in which the renal necropsy showed a typical acute tubular necrosis associated with nontraumatic rhabdomyolysis.

Case Report

A 49-year-old male patient was admitted with anuria, and proximal muscle weakness. He had had surgery for colon cancer 3 years earlier. The specimen obtained showed poorly differentiated adenocarcinoma.

On admission he appeared acutely ill. 'Heliotrope rash' and eyelid edema were noted on his face. There was no rash on any other part of the body. Neurologic examination showed marked weakness and hypotonia of proximal muscles, especially in the shoulders. Biceps, triceps and knee jerk were hypoactive. Sensation and superficial reflexes were normal. He was anuric. The laboratory findings revealed Hb 10.5 g/dl; WBC 6,900/mm³ with differential count showing neutrophils 75%, lymphocytes 15%. ESR was 116 mm/h. Serum Na was 140 mEq/l, K 7.0 mEq/l, P 4.0 mg/dl, uric acid 11.8 mg/dl, albumin 2.9 g/dl and globulin 3.0 g/dl. Serum creatine kinase was 144 U/l, lactic dehydrogenase 203 U/l, SGOT 265 U/l, SGPT 113 U/l. BUN was 84 mg/dl and creatinine was 13.9 mg/dl. A skin biopsy of the rash on the face revealed flattening of the rete ridge and liquefactive degeneration of basal cell layer.

A biopsy of the deltoid muscle showed edema between muscle fibers, variable diameter of muscle fibers, massive fiber necrosis, and perivascular infiltration of acute and chronic inflammatory cells. Electromyogram revealed myopathic findings compatible with dermatomyositis. Computed tomographic scanning of abdomen showed extensive obstructive uropathy in both midureters due to retroperitoneal lymphadenopathy but no evidence of liver metastasis. He had hemodialysis 3 times via subclavian catheter but refused further treatment. Two weeks later the patient died of uremia and sepsis with pneumonia. A renal necropsy performed just after death revealed focal, severe necrosis of the tubular epithelium with pigmented casts in the lumens. Interstitial tissue

showed focal fibrosis with mononuclear cell infiltration. But malignant cells were not found here. These findings were consistent with acute tubular necrosis from nontraumatic rhabdomyolysis.

Comments

Dermatomyositis is an inflammatory myopathy of unknown etiology characterized by progressive symmetric muscle weakness. The presence of characteristic cutaneous lesions in this condition distinguishes it from polymyositis. Five major criteria may be used to define polymyositis and dermatomyositis [5]. In our cases, all of the five abnormalities were found. Dermatomyositis and polymyositis have been known to be associated with an increased frequency of internal malignancies [6, 7]. Dermatomyositis is associated with malignancy more frequently than is polymyositis [8]. In fact, suggested classifications include a category of 'dermatomyositis and polymyositis with neoplasia' [5]. But a carefully controlled and prospective study is needed to support this notion unequivocally [9,10].

Although myoglobulinuria and myoglobulinemia are not uncommon in patients with dermatomyositis [11], acute renal failure in association with dermatomyositis has been reported only in a few patients [1–4]. The infrequent occurrence of renal failure may be related to a less severe degree of myoglobulinuria in these patients [11] or to lack of other known predisposing factors in the presence of which myoglobulinuric renal failure commonly gets precipitated [12]. The factors predictive of renal failure in this setting include the degree of serum creatine kinase, serum potassium, and the serum phosphorus

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level elevation; the degree of depression of serum albumin level; and the presence of dehydration or sepsis as the underlying cause [13]. Our patient had most of the factors. Though the exact mechanism of the development of acute renal failure in rhabdomyolysis is not clear, direct toxic effects of myoglobin or decomposition products such as ferrihemate, tubular obstruction by myoglobin or uric acid crystals, and renal ischemia due to release of vasoconstrictive mediators have all been proposed [14]. In animal experiments, importance of dehydration and acidic urine is emphasized [15].

The diagnosis of acute tubular necrosis in our patient was established on the basis of the clinical findings, the laboratory data, and the findings on renal necropsy. The relatively lower serum creatine kinase level in our case compared with other reports may be ascribed to coexistence of malignancy in which normal creatine kinase levels are described [16]. The confirmation of diagnosis of myoglobinuric acute renal failure may be made by identification of myoglobin in the urine. But we could not obtain a urine specimen because of total anuria. The noxious effect of myoglobin was probably enhanced by the obstructive nephropathy. The clinical findings of muscle weakness, pain and the increased values of creatine kinase, lactic dehydrogenase, and transaminases support the diagnosis of rhabdomyolysis. In our patient, a combination of nontraumatic rhabdomyolysis and obstructive uropathy by metastatic colon cancer seemed to have caused the acute renal failure. Nontraumatic rhabdomyolysis is not a rare cause of acute renal failure and should be considered when the etiology of acute renal failure is uncertain.

References