Acute interstitial nephritis in association with polymyositis

Sir,

Inflammatory myopathies are diseases in which muscles are damaged by endomysial and occasionally perivascular infiltration of inflammatory cells. The clinical syndrome with these pathological changes is termed polymyositis. Renal involvement in polymyositis is distinctly uncommon.[1]

A 39-year-old male presented with progressive weakness of upper and lower limbs for six months, worsening two months prior to admission. Proximal muscle weakness with sparing of extraocular and facial muscles was present. There was no history of skin rash, exposure to myotoxic drugs or toxins. Proximal muscle weakness was present on examination. No lymphadenopathy or uveitis was present.

Investigation showed elevated muscle enzymes with creatine kinase (CK) 12,980U/L, lactate dehydrogenase (LDH) 1600U/L and aspartate amino transferase (AST) 198U/L. Renal and liver functions were normal. Muscle biopsy was suggestive of inflammatory myopathy and a diagnosis of acute polymyositis was made. He was initiated on oral prednisolone at 1 mg/kg/day and discharged. Over the next three months an improvement in the levels of muscle enzymes was observed. Prednisolone was tapered to 0.5 mg/kg/day and oral azathioprine 150 mg was added once daily. Prednisolone was stopped after six months.

Seven months after discharge he was readmitted with fever and acute parotitis. Investigations showed leucocytosis and neutrophilia. His blood urea was 76 mg/dl, serum creatinine 1.7 mg/dl, serum calcium 7.8 mg/dl and serum phosphorus 2.7 mg/dl with normal potassium levels. Muscle enzymes were elevated (CK 575U/L, LDH 548U/L, AST 47U/L). Urinalysis showed no eosinophiluria. The blood and urine cultures were sterile. There were no features to suggest sicca syndrome and the parotitis resolved with antibiotic. It was hence not investigated any further. No features of sarcoidosis were present.

The serum creatinine increased to 6.4 mg/dl over the next three days and hemodialysis was initiated. He improved clinically but renal function failed to improve and a renal biopsy was performed.

Light microscopy was suggestive of acute tubulo-interstitial nephritis with tubular injury [Figure 1]. Immunofluorescence was negative for immunoglobulins and C3. Oral prednisolone was restarted at 1 mg/kg/day and he was discharged with a serum creatinine of 2.7 mg/dl. Renal function normalized on follow-up.

Acute tubular necrosis related to myoglobinemia and myoglobinuria is a well-recognized feature of acute rhabdomyolysis in polymyositis.[2] Mesangial proliferation is the commonest glomerular lesion in polymyositis and suggests a possible association between arthritis and glomerulonephritis.[3] Chronic glomerulonephritis has also been infrequently reported.[2] Association of acute interstitial nephritis (AIN) has not been described with polymyositis.

A study of 64 patients with neuromuscular disease on azathioprine reported toxicity with reversible leucopenia (22%), hepatotoxicity (9%) and a systemic reaction (12%).[4] Azathioprine hypersensitivity manifests with fever and gastrointestinal symptoms initially. Maculopapular rash, urticaria, vasculitis, erythema multiforme or erythema nodosum may occur. Hepatotoxicity and nephritis have also been reported.[5] This reaction is observed in patients early during treatment with azathioprine. This patient

Figure 1: Photomicrograph showing renal tissue with moderately dense acute inflammatory cells in the interstitium, within basement membrane and lumen of tubules (H and E, ×400)
received azathioprine for more than six months and did not have any features to suggest a hypersensitivity reaction. Other medications taken by him were reviewed but no drug causing AIN was consumed. An association of AIN with polymyositis was thus made. The resolution of renal failure and improvement of myositis with corticosteroid treatment indicates the possibility of an immune pathophysiology of this association.

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References