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Tubulointerstitial disease and ulcerative colitis

Sir,
The use of anti-inflammatory agents such as aminosalicylate is a major cause of renal failure (RF) in patients with inflammatory bowel diseases (IBD). Amyloidosi and, very rarely, glomerulopathies (IgA nephropathy and membranous glomerulopathy) may also accompany IBD. The description of tubulointerstitial disease unrelated to the consumption of nephrotoxic agents in such patients is really sparse in the literature. It has been shown that a minimal degree of renal tubular dysfunction (manifested as tubular proteinuria) exists even before the introduction of nephrotoxic drugs [1]. Here, we describe a patient with ulcerative colitis who developed new onset and unprovoked renal failure.

In spring 2004, a 19-year-old man, with a known history of ulcerative colitis (UC) diagnosed 1 year previously, was admitted to the nephrology ward for work-up of insidious onset renal failure. His serum creatinine levels were high (the maximum was 4.6 mg/dl) for the previous 6 months. He had a history of juvenile rheumatoid arthritis (JRA) in his early teens, was treated with prednisolone (5 mg/day), cyclosporin (100 mg/day) and low-dose tolmetin (from 1996 to 1998) with full recovery. The patient revealed a 3-year history of frequent and recurrent loose bowel movements that led to the diagnosis of UC last year. At the time of this diagnosis (of UC), the patient’s serum creatinine was found to be elevated at 3.7 mg/dl; which declined to a normal level by fluid therapy.

Physical examination revealed a teenager of short stature and mild obesity who was doing well and in no acute distress. On admission, serum creatinine and urea levels were 9.1 and 230 mg/dl, respectively. This unexplainable azotaemia led to a renal biopsy. Extensive work-up for extraintestinal manifestations of IBD was done. Renal biopsy demonstrated no amyloidosi on Congo red staining. However, intense interstitial mononuclear infiltration with occasional eosinophils, fibrosis and tubular atrophy were detected. Considering that the patient had not used any nephrotoxic agent for at least a year, we concluded that his kidney disorder might be part of a common immunological dysregulation that had begun with JRA and ended in UC and tubulointerstitialitis.

To our knowledge, overt renal failure due to interstitial nephritis (unrelated to nephrotoxic administration) has not been previously reported among UC patients. In a study of 43 UC patients, 23% of cases had pathologic enzymuria (a marker of early renal tubular injury) that almost normalized with anti-inflammatory therapies [2]. Furthermore, a strong correlation between disease activity and tubular proteinuria has also been found in IBD patients [3]. These findings may support the hypothesis that a tubulointerstitial disorder may be a natural part of IBD. We recommend that clinicians consider tubulointerstitial disease and renal failure as possible extraintestinal manifestations of UC.

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The search for a link between inflammation and hypertension—contribution from Bartter’s/Gitelman’s syndromes

Sir,
The involvement of inflammation and its mediators in cardiovascular pathophysiology and atherogenesis is increasingly recognized [1]. Plasma level of inflammatory molecules such as C-reactive protein (CRP); cytokines, such as tumour necrosis factor-α (TNF-α) and interleukin-6 (IL-6); chemokines, such as monocyte chemoattractant protein (MCP-1) and adhesion molecules, such as P-selectin and leucocyte adhesion molecules, intercellular adhesion molecules (ICAM-1), are