Resolution of Acute Interstitial Nephritis without Steroids

Cince the writing of the article on fluroquinolone-Uinduced acute interstitial nephritis published elsewhere in this issue,1 2 additional cases of this condition have occurred at the same institution. Both cases involved the fluoroquinolone ciprofloxacin. Each patient previously had normal renal function. On day 5 and 7, respectively, of treatment with ciprofloxacin, serum creatinine levels were found to be significantly elevated, at 584 and 228 mmol/L, respectively (normal ranges 62 to 120 mmol/L for women and 55 to 115 mmol/L for men). The ciprofloxacin was discontinued. In the first case, prednisone was initiated, and the patient's serum creatinine returned to baseline in 3 weeks. In the second case, no steroid was used, and by day 16 this patient's creatinine level had also returned to normal.

The rapid and full recovery in the absence of corticosteroid use in the second case raises the question of the role of corticosteroids in drug-induced acute interstitial nephritis. There have been no prospective trials evaluating whether prednisone therapy is really beneficial or necessary in this condition. Most of the impetus to use corticosteroids comes from one small study,2 in which the number of days from peak serum creatinine to baseline was recorded. Eight patients with methicillin-induced acute interstitial nephritis received corticosteroids and 6 patients did not receive any drug therapy. In the prednisone-treated group, serum creatinine returned to baseline more rapidly than in the control group. Anecdotal case reports of corticosteroids hastening renal recovery in drug-induced acute interstitial nephritis make up the bulk of the additional evidence.3,4

In the treatment of drug-induced acute interstitial nephritis, discontinuation of the suspected drug is always recommended,³⁻⁷ but the role of corticosteroids is less clear.^{3,4,5,7} In the 2 cases presented here, resolution of the nephritis upon discontinuation of the suspected

offending agent, ciprofloxacin, was dramatic, whether or not steroids were used. The possibility that discontinuation of the drug is the most important factor in resolution of nephritis is supported by the fact that complete renal recovery appears to be inversely proportional to the duration of renal failure.³ Therefore, early intervention and discontinuation of the suspected agent are of the utmost importance.

In any case of drug-induced acute interstitial nephritis, the risk-benefit profile should be carefully evaluated before corticosteroids are initiated. These agents could contribute to the death of the patients, and their benefit is unclear.⁵

Séadna Ledger, BScPharm Pharmacist

Nephrology Program London Health Sciences Centre London, Ontario

Myura Rao, BSc, MD Nephrology Fellow University of Western Ontario London, Ontario

References

- MacAulay S, Bayliff C, Mehta S. Fluoroquinolone-induced acute interstitial nephritis: two case reports. Can J Hosp Pharm 2002;55:140-3.
- Galpin JE, Shinaberger JH, Stanley TM, Blumenkrantz MJ, Bayer AS, Friedman GS, et al. Acute interstitial nephritis due to methicillin. Am J Med 1978;65:756-65.
- Kelly CJ, Neilson EG. Chapter 32. Acute interstitial nephritis. In: Brenner BM, Levine SA, editors. Brenner & Rector's the kidney. 6th ed. Philadelphia (PA): WB Saunders; 2000. p. 1520-1.
- Buysen JG, Houthoff HJ, Krediet RT, Arisz L. Acute interstitial nephritis: a clinical and morphological study in 27 patients. *Nephrol Dial Transplant* 1990;5:94-9.
- Murray KM, Keane WR. Review of drug-induced acute interstitial nephritis. *Pharmacotherapy* 1992;12:462-7.
- Wolfson AB, Israel RS. Chapter 133. Interstitial disease. In: Rosen P, editor. *Emergency medicine: concepts and clinical practice*. 4th ed. St Louis (MO): Mosby-Year Book Inc; 1998. p. 2271.
- Schmitz PG. Chapter 148. Allergic interstitial nephritis. In: Noble J, editor. *Textbook of primary care medicine*. 3rd ed. St Louis (MO): Mosby-Year Book Inc; 2001. p. 1393.

