

Nephrotic Syndrome after Norfloxacin

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Dear Sir,

Pure nephrotic syndrome after administration of norfloxacin has not been previously reported. We describe 1 case.

A 65-year-old woman with a history of hysterectomy and oophorectomy, late-onset diabetes, and rheumatoid arthritis had been taking indomethacin daily for 5 years. One week before admission, she was treated with norfloxacin, 800 mg daily, for a lower urinary tract infection for 3 days. On the 4th day, limb swelling and progressive breath-lessness were observed. Her general practitioner prescribed furosemide, amlodipine, and isosorbide dinitrate. The patient was seen on January 24, 1994, for the first time. Her physical examination was normal except obesity and bilateral lower limb pitting oedema. The BP was 18/10. Blood eosino-phils, Waaler-Rose test, hepatitis B surface antigen and antibody, ANCA, FAN, C3, C4, and antibasement antibody were normal. Proteinuria was 3.6 g/day on admission, but later reached 9.6 g/day. A renal biopsy specimen showed mild focal-segmental glomerulosclerosis. The treatment consisted of clonidine, amlodipine, lisinopril, and diet. After the start of lisinopril treatment, proteinuria abruptly decreased to 3 g/day.

Follow-up showed progressive disappearance of the proteinuria. On May 2, 1994, proteinuria was equal to zero. At the patient's request, indomethacin was reinsti-tuted at the end of November, 1994. Protein-

uria did not recur. The serum creatinine level remained normal throughout this period. The patient was not rechallenged with norfloxacin.

Renal side effects of quinolones are infrequent. Acute renal failure has been reported and was attributed to either drug-induced tubulointerstitial allergic nephritis or crystal-luria [1, 2].

Nephrotic syndrome and acute renal failure have also been reported with ciprofloxacin in 1 patient [3]. Acute renal failure without proteinuria has been described in association with norfloxacin [4]. The histological appearance of the kidney could be linked with obesity only [5].

Calcium antagonists seem to protect the kidney, and converting enzyme inhibitors are able to decrease proteinuria [6]. The initial improvement of the nephrotic syndrome observed in our patient could have been related to the combined effect of two renal-protective drugs and the withdrawal of the causative agent.

In conclusion, a patient who had been treated with indomethacin for years without any renal toxicity developed a nephrotic syndrome after 3 days of norfloxacin treatment. Discontinuation of both drugs was followed by remission. Rechallenge with indomethacin did not trigger a relapse. This strongly suggests a causative role of norfloxacin in the nephrotic syndrome.

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