Second Attack of Acute Tubulointerstitial Nephritis Induced by Cefataxim and Pregnancy

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Dear Sir,

Acute interstitial nephritis (AIN), also called tubulointerstitial nephritis, is frequently associated with hypersensitivity reactions to medications such as antibiotics or nonsteroidal anti-inflammatory drugs and systemic infections [1-6]. While over 40 drugs have been reported to cause acute renal failure due to AIN, agents of the penicillin class or other antibiotics remain the foremost offenders both in frequency of reactions and their significance [1-6]. Pregnancy is a risk factor in patients with renal diseases, but it is possible, depending on the clinical signs of the disease [7-9].

The patient, a 20-year-old single female, was admitted to the Department of Nephrology because of general fatigue, anorexia, vomiting, fever, myalgia and macrohematuria. Therapy with cefataxim, 1 g/daily during 15 days was performed previously because of cystitic problems. On admission the patient was febrile (39°C). Erythrocyte sedimentation rate was 89 mm/h, red blood cell (RBC) count $3.5 \times 10^4/\text{mm}^3$ and white blood cell (WBC) count 11,000/mm³ with 6% eosinophils. The serum urea level was 35.6 mmol/l during the first day and creatinine 998 $\mu\text{mol/l}$ during the second. Diuresis was 200 ml/daily during these 2 days. Fever stopped on the 3rd day and diuresis started with development of polyuria on the 10th day (2,500-4,400 ml). Serum levels of urea and creatinine decreased to normal ranges on about the 15th day. Urinalysis showed glycosuria and moderate proteinuria (2.15 g/l) and

Fig. 1. Electron micrograph of glomerular capillary loop: regular structure of glomerular basement membrane with slight focal fusion of foot processes. $\times 8,580$.

renal tubules was accompanied by severe edema and accumulation of lymphocytes, rarely polymorphonuclears, eosinophils and plasma cells. Some tubules contained casts, in others the tubular cells seemed to be degenerating, particularly in relationship to the most dense cellular infiltrates. Immuno-fluorescent studies presented focal granular deposits of IgG and C3(+) in the mesangial area. Electron microscopy showed focal fusion of foot processes with inflammatory cells in the interstitium. Tubular basement

urinary sediment 15-20 RBC and 10-15 WBC. Plasma proteins and serum complement studies were normal. Serum levels of IgA (5.15 g/l) and IgM (4.02 g/l) were elevated. Urine culture, coproculture, hemoculture and testing for brucellosis were negative. Chest radiography was normal. Ultrasonographic examination of the kidneys revealed their enlargement with parenchymal edema. No histopathologic changes of glomeruli and extraglomerular vessels were observed on optical microscopy. Diffuse separation of the

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Fig. 2. Electron micrograph of interstitium: increased number of fibroblasts, lymphocytes, monocytes and neutrophils with intensive deposition of collagen. $\times 6,292$. membrane presented irregularity and defects.

The patient was admitted again after 6 months because of fever, anorexia, vomiting and macrohematuria. During the preceding month she had been treated with cefataxim for 7 days, again because of cystitic problems. Oliguria was present only during the first day of admission. SR was elevated again (147/h), RBC and WBC counts were normal. Because of the early start of diuresis, elevation of serum urea was only to 16.8 mmol/l and creatinine to 347 $\mu\text{mol/l}$; proteinuria was 2.5 g/l. Urinary sediments presented 20-30 RBC and 30-40 WBC with 8% eosinophils. Ultrasonographic examination revealed edematous kidneys and an unexpected finding: a pregnancy of 10 weeks of gestation. Normal fetal growth was noted during the follow-up, the body weight of the newborn was 3.2 kg. Cephalosporins were not given anymore to our patient and she has been without attacks of AIN for the past 2 years. The list of drugs inducing AIN is still growing; over 40 drugs have been implicated as the etiological agent associated with this pattern of renal injury [1-6, 10-12]. Antibodies known as etiological factors for AIN are phenoxymethylpenicillin potassium, doxycycline, trimetoprim, sulfamethoxazole, oxytetracycline, rolitetracycline, rifampin, ampicillin, amoxycillin, erythromycin, vancomycin, propicillin potassium, ciprofloxacin, gentamycin sulfate, chloramphenicol, carbamezapine and other drugs, but cefataxim has not been reported [1-6, 10-12]. Pregnancy is a risk factor in renal disease [7-9] and vice versa, but the second attack of the disease in our patient was not severe and the pregnancy was with a good outcome.

References

- Appel GB: A decade of penicillin related acute interstitial nephritis – More questions than answers. *Clin Nephrol* 1980;13:151-154. Laberke HG, Bohle A: Acute interstitial nephritis: Correlations between clinical and morphological findings. *Clin Nephrol* 1980; 14: 263-273.
- Baldwin DS, Levine BB, McCluskey RT, Gallo GR: Renal failure and interstitial nephritis due to penicillin and methicillin. *N Engl J Med* 1968;279:1245-1252.
- Neilson EG: Pathogenesis and therapy of interstitial nephritis. *Kidney Int* 1989;35:1257-1270.
- Cryst C, Hammar SP: Acute granulomatous interstitial nephritis due to co-trimoxazole. *Am J Nephrol* 1988;8:483-488.
- Nortier J, Depierreux M, Bourgeois V, Dupont JP: Acute interstitial nephritis with nephrotic syndrome after intake of naproxen and amoxycillin. *Nephrol Dial Transplant* 1990;5:1055-1057. 11
- Jungers P, Forget D, Houiller P, et al: Pregnancy in IgA nephropathy, reflux nephropathy and focal glomerular sclerosis. *Am J Kidney Dis* 1987;9:334-338.
- Packham DK, North RA, Fairly KF, et al: IgA glomerulonephritis and pregnancy. *Clin Nephrol* 1988;30:15-21.
- Packham DK, North RA, Fairly KF, et al: Membranous glomerulonephritis and pregnancy. *Clin Nephrol* 1987;28:56-64. Korzets Z, Elis A, Bernheim J: Acute granulomatous interstitial nephritis due to nitrofurantoin. *Nephrol Dial Transplant* 1994;9:713-715.
- Bailey JR: Ciprofloxacin-induced acute interstitial nephritis. *Am J Nephrol* 1992;12:271-273.

Garcia-Martín F, Mampaso F, de Arriba G, Moldenhauer F, Martin-Escobar E, Saiz F: Acute interstitial nephritis induced by etambutol. Nephron 1991;59:679-680.

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