D.	Dragan	Ljutić ^a
M.	Meri	Glavina ^b

Dragan Ljutić, MD, PhD, Department of Internal Medicine, Clinical Hospital Split, 58 000 Split (Croatia)

Dear Sir,

A 49-year-old woman was referred to the Renal Unit because of azotemia and fatigue. Two months earlier, she had had herpes zoster located on the right T11 dermatome. Twenty days after skin eruption, iridocyclitis of the left, and a week later of the right eye were diagnosed and successfully treated. During that time she had general malaise, fatigue, anorexia, and mild fever. The erythrocyte sedimentation rate (ESR) was 106 mm/h, serum creatinine 154 μmol/l, total protein 85.5 g/l, IgG 20.8 g/l, IgA 6.8 g/l, and circulating immune complexes 0.180 g/l. Serology revealed previous cytomegalo-, Epstein-Barr, herpes simplex, and varicella zoster virus infections. Because of an increment of creatinine over the following 3 weeks, she was referred to our unit.

On admission, the physical examination was unremarkable. Laboratory tests revealed ESR of 110 mm/h, normocytic normo-chromic anemia (hemoglobin 104 g/l), urea of 17.5 mmol/l, creatinine of 664 µmol/l (later, the highest was 671 µmol/l), IgG of 19.8 g/l, and IgA of 7.1 g/l. Other biochemical

 $\frac{5}{8} \frac{5}{8}$

• **□** • **□** • **□** , **□** **□** **●**

Æ

■ ■ ■ ■ ■, $\frac{1}{8}$, $\dots$, $\bullet V$

Fig. 1. Infiltration of mononuclear cells in the renal interstitium. PAS. $\times 40$.

malities. Immunofluorescence studies were negative. Prednisone was given (80 mg, with subsequent slowly tapering). Serum creatinine decreased to $156 \mu\text{mol/l}$ within 2 weeks. The follow-up of 4 months revealed stable renal function and no uveitis relapse.

In the present case, typical clinical and laboratory findings of TINU syndrome followed herpes zoster. At the time of hospitalization there was no serological evidence of acute viral infection, but there was evidence of previous herpes simplex, varicella zoster, cytomegalo- and Epstein-Barr virus infec-

tions. It is known that herpes simplex virus can produce tubulointerstitial nephritis [2]. The same is true for cytomegalo- and Epstein-Barr viruses [3, 4]. Varicella zoster virus reactivation has also been associated with a deterioration in renal failure, but there were no histological or microbiological data showing that herpes zoster causes tubulointerstitial nephritis [5]. The question is whether preceding viral reactivation (in the present case of varicella zoster) could be a trigger, the cause, or a consequence of the immunological disorder underlying TINU.

References

Cameron JS: Allergic interstitial nephritis: Clinical features and pathogenesis. *Q J Med* 1988;250:97-115.

Silbert PL, Matz LR, Christiansen K, Saker BM, Richardson M: Herpes simplex virus interstitial nephritis in a renal allograft. *Clin Nephrol* 1990;33:264-268.

Cameron J, Rigby RJ, Van Deth AG, Petrie JJB: Severe tubulointerstitial diseases in a renal allograft due to cytomegalovirus infection. *Clin Nephrol* 1982;18:321-325.

Kopolovac J, Pinkus G, Rosen S: Interstitial nephritis in infectious mononucleosis. *Am J Kidney Dis* 1988;12:565-567.

Bourbigot B, Quillien MC, Aïriau J, Hardey E, Guiserix J, Leroy JP, Clodes J, Chasel C: Renal failure in the course of reactivation of varicella zoster virus infection in a renal transplant recipient. *J Infect* 1986;13:159-162.

486 Ljutić/Glavina Tubulointerstitial Nephritis with Uveitis
Syndrome following Varicella Zoster Reactivation