Acute Renal Failure Due to Hypersensitivity Interstitial Nephritis Induced by Warfarin Sodium

A.		Volpi
G.M.		Ferrario
F.		Giordano
G.		Antiga
G.		Battini
C.		Fabbri
M.		Meroni
A.		Sessa

Unità Operativa di Nefrologia e Dialisi. Servizio di Anatomia Patologica. Ospedale di Vimercate, Italia

Dr. A. Volpi, Unità Operativa di Nefrologia e Dialisi, Ospedale di Vimercate, Via Cereda, 2, I-20059 Vimercate (Italy)

Sir,

Adverse reaction to a growing number of drugs is being recognized as a cause of acute renal failure [1].

The clinician should suspect the possibility of acute drug-induced tubulointerstitial nephritis in all cases of acute renal failure of unknown etiology. Acute drug-induced hypersensitivity reaction is a common form of tubulointerstitial nephritis with sudden deterioration of renal function and oliguria. Renal function usually returns toward baseline after withdrawal of the drug.

Systemic signs and symptoms such as fever, skin rash, arthralgias, myalgias, eosinophilia and seldom lymphadenopathy suggest a hypersensitivity mechanism; certain observations suggest that immunologically mediated tissue damage is the consequence of cell-mediated injury [2].

We have observed a 65-year-old female who developed fever, arthralgias, gross hematuria, sterile pyuria and oliguric acute renal failure (serum creatinine rose from 1.2 to 6.8 mg/dl). She was treated for 25 days with diltiazem, nitroglycerine discs and warfarin sodium (20 mg/day) because of myocardial infarction.

Renal biopsy showed a typical picture of acute tubulointerstitial nephritis, with several interstitial eosinophils, mononuclear cells, patchy focal tubular necrosis and intratubular casts of eosinophil cells (fig. 1). The glomeruli showed mild mesangial hypercellularity with IgA and C3 mesangial deposits at immunofluorescence. Late assay of serum IgE was normal.

Withdrawal of warfarin sodium and treatment with corticosteroids (1 mg/kg/day) returned renal function to the normal level in 45 days (serum creatinine 1 mg/dl).

Acute renal failure due to acute drug-induced tubulointerstitial nephritis is seen more frequently in older patients and, usually, the mean latent period of treatment is 15 days (from 2 to 44 days) [2].

In the absence of other apparent causes we think that warfarin sodium hypersensitivity has likely been the

Fig. 1. Intratubular cellular debris with several eosinophilic leukocytes. HE. $\times 600$.

cause of acute renal failure in the patient; the associated Berger's disease could be a meaningless findings.

In conclusion, we report that among the oral anticoagulants, besides phenindione, warfarin sodium must be considered a drug capable of inducing tubulointerstitial nephritis [3].

References

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