Controversy regarding paracetamol- and codeine phosphate-induced acute tubulointerstitial nephritis-uveitis (TINU) syndrome

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To the Editor,

I read with great interest the case report by Alaygut et al.[1] about two cases with tubulointerstitial nephritis-uveitis (TINU) syndrome due to paracetamol and codeine phosphate use. However, some equivocal conditions need to be clarified.

Firstly, it is controversial to define the second case as tubulointerstitial nephritis (TIN) or TINU syndrome due to lack of either histopathological analysis, or renal failure or uveitis. Despite spontaneous regression of renal findings, it is invalid to diagnose TIN without a biopsy. A similar clinical course may be seen in various other renal disorders related to tubules. It has been reported that β_2 microglobulin is markedly elevated in almost every case tested in TINU syndrome, and looking for evidence of such elevation may be useful in cases where renal biopsy is not indicated[2]. Krebs von den Lunge-6 (KL-6) glycoprotein in serum or renal biopsy may also be a valuable tool in the diagnosis and follow-up of TINU syndrome[3]. The authors may confirm the diagnosis with these methods. Additionally, patients may be regarded as TINU cases when an actual attack occurs, rather than in instances where there is only a potential risk for development of uveitis in the future.

Secondly, the time between usage of the drugs and emergence of TINU seems too long in both cases to hold the drugs responsible (500 mg paracetamol/10 mg codeine phosphate, three times/day for two days three weeks previously in Case 1 and three times/day for four days one month previously in Case 2). Also, in Case 2, the complaints at admission had been ongoing for four months, and various medical interventions had taken place. For that reason, TIN/TINU may not be related to paracetamol and codeine phosphate in these cases.

Key words: Tubulointerstitial nephritis-uveitis syndrome (TINU), paracetamol, codeine phosphate.

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