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Chronic urate nephropathy with a disproportionated elevation in serum uric acid

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A 38-year-old man visited our institution for evaluation of proteinuria. Prior to this, the patient was diagnosed with hypertension. One year ago, the patient was admitted to our hospital because of gout on his right knee and proteinuria. At that time, aspiration of synovial fluid revealed positive intracellular and extracellular birefringent crystals by compensated polarized light microscopy. He has been taking medications for gout and hypertension since. The urinalysis revealed 2+ proteinuria and 10–15 red blood cells per high-power field. The laboratory tests showed haemoglobin 14.7 g/dl, uric acid 11.5 mg/dl, blood urea nitrogen 29 mg/dl, creatinine 1.96 mg/dl, cholesterol 260 mg/dl and triglyceride 477 mg/dl. Ultrasonography demonstrated normal-sized kidneys with mildly increased parenchymal echogenicity in both kidneys. Microscopic study of renal biopsy showed uric acid crystals in amorphous (asterisks) or spindle shapes (arrows) deposited within the renal medulla surrounded by mononuclear inflammatory cells (Figure 1).

For the pathophysiology of chronic urate nephropathy, a series of reactions initiated by hyperuricaemia or uricosuria was suggested: intra-tubular deposit of uric acid crystals, consequent local obstruction and rupture of crystals into the interstitium and, finally, an inflammatory response and interstitial fibrosis around the crystals [1]. In addition, recent studies have suggested that hyperuricaemia may induce renal injury independent of crystal formation by several mechanisms: activation of the renin–angiotensin system, stimulation of cyclooxygenase-2 and inhibition of local nitric oxide synthase with a reduction in endothelial nitric oxide [2]. Patients may present with hypertension, hyperuricaemia, mild azotaemia and mild proteinuria. We can also consider chronic urate nephropathy if there is an elevation in the serum uric acid concentration out of proportion to the degree of renal insufficiency [3]. This is a case having typical clinical manifestations with a disproportionate elevation in serum uric acid in relation to the degree of renal insufficiency. However, we also need to consider *UMOD*-related kidney disease (uromodulin-associated kidney disease) when the patient has renal impairment between age 15 and 40 years, precocious gout and hyperuricaemia, although urate crystals are said to be rare [4].

Conflict of interest statement. None declared.

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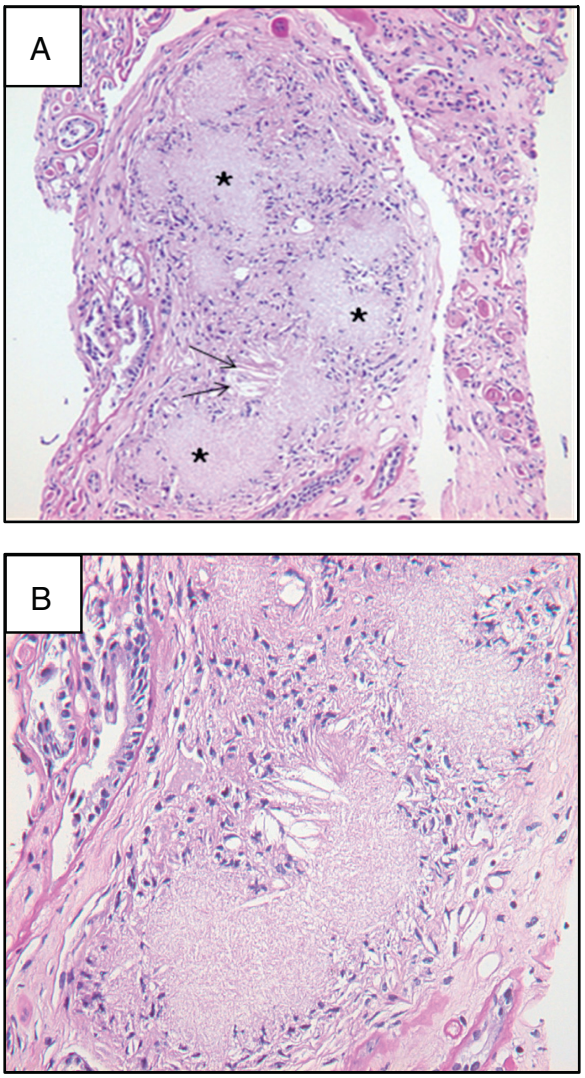

Fig. 1. Uric acid crystals, in amorphous (asterisks) or spindle shape (arrows), deposited within the renal medulla surrounded by mononuclear inflammatory cells (periodic acid Schiff stain; magnification: **A** ×200, **B** ×400).