Dear Sir,

Renal failure is uncommon in sarcoidosis [1] and is mainly due to hypercalcaemia [2]. Other rare causes described are granulomatous interstitial inflammation and various types of glomerulonephritis. We report a case of severe renal failure in sarcoidosis in which, despite normocalcaemia and lack of renal calcification on X-ray, renal biopsy showed diffuse nephrocalcinosis. Creatinine clearance and hypercalciuria returned to normal following treatment with oral prednisolone.

A 37-year-old man was referred in 1983 for investigation of renal failure. He had been investigated 4 years previously because of urinary calculi, at which time serum calcium was normal (2.21 mmol/l) and blood urea minimally elevated (7.8 mmol/l). In 1982 sarcoidosis had been diagnosed based on bilateral hilar adenopathy, non-caseating granulomata on supraclavicular lymph node biopsy, and a positive Kveim test. Other investigations in 1982 showed normal serum calcium (2.65 mmol/l), normal serum phosphate (0.9 mmol/l) and mild impairment of renal function (blood urea 15.4 mmol/l, serum creatinine 146 µmol/l, creatinine clearance 72 ml/min). Intravenous pyelogram showed no abnormal findings. In 1983 he was re-admitted to hospital because of nausea and vomiting. He was found to have hypertension (190/140), early pulmonary fibrosis, abnormal liver function tests and progressing uraemia (blood urea 21.6 mmol/l, serum creatinine 373 µmol/l, creatinine clearance 32 ml/min). Significant hypercalciuria was present (14.9 mmol/24 h), but serum calcium was normal (2.3 mmol/l) as were serum phosphate (0.87 mmol/l) and serum parathyroid hormone (0.2 µg/l), intravenous pyelogram showed only a small stone in the left kidney with no associated scarring, obstructive changes or nephrocalcinosis. Pulmonary function tests showed moderately reduced diffusion, normal spirometry and lung volumes.

![Fig. 1. Calcium deposits in tubular epithelium. Von Kossa. ×375.](image-url)

In view of his progressive renal failure, oral prednisolone, 20 mg daily, was commenced in addition to anti-hypertensive treatment with atenolol 50 mg and cyclopenthiazide 0.5 mg daily. Percutaneous renal biopsy showed nephrocalcinosis and mild interstitial fibrosis (fig. 1), with no
evidence of glomerular lesion, vasculitis, granulomata or hypertensive nephrosclerosis. Four weeks after commencing treatment, repeat renal function tests showed creatinine clearance 67 ml/min, serum creatinine 164 μmol/l, proteinuria 0.18 g/24 h, serum urea 11.9 mmol/l. Serum calcium remained normal (2.59 mmol/l) and urine calcium excretion had fallen to within the normal range (6.1 mmol/24 h). Serum phosphate and serum parathyroid hormone were both within the normal range. After a further follow-up period of 12 months to date, creatinine clearance measures 114 ml/min, serum creatinine 118 μmol/l and serum urea 9.0 mmol/l. Liver function tests remain mildly abnormal and pulmonary function tests are unchanged.

We have been able to find only 1 other case in the literature where renal failure has occurred in sarcoidosis due to diffuse nephrocalcinosis despite absence of hyper-calcaemia [3] and radiologically evident renal calcification.


References