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Images in Nephrology
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Nephrocalcinosis in a patient with secondary hyperoxaluria

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Case

A 74-year-old man presented to the renal unit 5 years ago with end-stage renal failure due to nephrocalcinosis. He has had numerous small bowel resections in the past for Crohn’s disease, resulting in short bowel syndrome. The nephrocalcinosis was due to hyperoxaluria. His abdominal radiograph (Figure 1) shows the cortical pattern of calcium oxalate crystal deposition seen in hyperoxaluria.

He had a renal transplant 3 years ago. There is no recurrence of the primary disease in his graft radiographically. However, his transplant biopsy at month 7 showed intraglomerular, intratubular and interstitial calcification in keeping with recurrence of oxalate crystals in his graft. He has since been told to maintain a urine output of 3 l a day, with a dietary restriction of low fat, low protein and low salt. He was started on potassium citrate to prevent further oxalate stone formation and calcichew to bind oxalate in his gut [1]. His Crohn’s disease remains in remission and his current creatinine is stable at 160 μmol/l.

Discussion

Oxalate is a metabolically inert ion. Oxalate homeostasis can be affected by renal failure and when there is overproduction or overabsorption of oxalate. The hyperoxalurias are divided into primary and secondary groups.

Types I and II primary hyperoxaluria are caused by an enzyme defect, which results in the increased biosynthesis of oxalate. Type III primary hyperoxaluria has been attributed to oxalate hyperabsorption in the presence of normal intestinal anatomy, histology and absorptive function.

Secondary hyperoxaluria can be due to enteric causes, as in this case, or non-enteric causes, such as oxalate ingestion from acute poisoning or excessive intake of ascorbic acid [2].

Conflict of interest statement. None declared.

References