Jejuno-ileal bypass, enteric hyperoxaluria, and oxalate nephrosis: a role for polarised light in the renal biopsy

K Hicks, G B Evans, M E Rogerson, P Bass

Abstract

Enteric hyperoxaluria, a complication of jejuno-ileal bypass, is associated with renal failure owing to oxalate nephrosis or tubulo-interstitial nephritis. A 54 year old woman developed renal failure 17 months after jejuno-ileal bypass for morbid obesity. Renal biopsy showed widespread acute on chronic damage to the tubulo-interstitial compartment with extensive deposition of oxalate crystals. The extent of oxalate deposition was only evident on polarisation of the biopsy.

Keywords: jejuno-ileal bypass; hyperoxaluria; renal failure; tubulo-interstitial nephritis

Jejuno-ileal bypass, a procedure which peaked in popularity in the mid-1970s as a treatment for morbid obesity, is a recognised cause of enteric hyperoxaluria. Some series have shown up to 30% of these patients develop renal calculi. Less commonly reported is the occurrence of renal failure owing to oxalate nephrosis or oxalate tubulo-interstitial nephritis. We present the case of a patient with rapidly progressive renal failure following jejuno-ileal bypass.

Case report

In May 1994 a 54 year old woman underwent jejuno-ileal bypass for morbid obesity (weight 140 kg, body mass index 49 kg/m²) owing to severe compulsive eating disorder. Endocrine investigations had been unremarkable. Her medical history revealed only osteoarthritis of a knee for which she had been taking diclofenac periodically for several years and only occasionally for the previous year. She had taken no other medicinal or vitamin preparation. At the time of the end–side jejuno-ileostomy, baseline serum biochemistry was normal: urea 5.6 mmol/litre, creatinine 84 µmol/litre. Formal creatinine clearance was not performed.

Postoperatively she suffered from diarrhoea, on average three times a day, passing soft or watery stools. Her weight fell steadily to 83 kg and remained stable at this level for six months before presentation.

In October 1995 she presented with a one month history of increasing fatigue, breathlessness, and dizziness. Physical examination revealed pallor and bradycardia confirmed by ECG as complete heart block. The heart sounds were normal with no evidence of heart failure or oedema. She was mildly hypovolaemic with a central venous pressure of +2 cm and blood pressure of 110/70 mm Hg. A laparotomy scar was noted, with no organomegaly. Blood chemistry findings were as follows: potassium 4.6 mmol/litre, urea 33.9 mmol/litre, creatinine 1176 µmol/litre, thyroid stimulating hormone 2.2 mU/litre, free thyroxine 11.2 pmol/litre, albumin 24 g/litre (other liver function tests were normal), haemoglobin 57 g/litre, serum vitamin B-12 181 ng/litre (normal range 150–700), serum ferritin 284 µg/litre (normal range 7–75), and red cell folate 333 µg/litre (normal range 150–700). Cardiac enzymes were not raised and an autoimmune profile was positive for smooth muscle antibody only. Urine microscopy revealed sterile pyuria with no casts. There was no proteinuria. Renal ultrasound showed kidneys of 10 cm and 10.5 cm, with normal cortical thickness and echogenicity and no evidence of obstruction.

Within two days of admission and before any improvement in her biochemistry, the complete heart block resolved spontaneously. Renal function showed a progressive decline with plasma creatinine rising from 1176 to 1318 µmol/litre, when a renal biopsy was performed. Plasma oxalate was measured at 90 µmol/litre (normal range 1–3).

Clinically, her renal function failed to improve and she has remained dialysis dependent.

Pathological findings

The specimen consisted of a 13 mm long needle biopsy of renal tissue composed of cortex and medulla.

Light microscopy (fig 1A) showed widespread acute on chronic damage to the tubulo-interstitial compartment, with focal interstitial fibrosis, tubular atrophy, and patchy interstitial oedema. Superimposed on this was a scanty interstitial acute and chronic inflammatory cell infiltrate with neutrophils.
and eosinophils which spilled into the medulla. No tubulitis was seen. Granular debris and oxalate deposits were seen in some tubular lumina. Examination with polarised light (fig 1B) showed marked deposition of oxalate crystals within tubular lumina and in the cytoplasm of many tubular epithelial cells. Occasional crystals appeared to be lying free within the interstitium and had provoked a giant cell response. In polarised light, the presence of oxalate was seen to be much more extensive than had appeared with normal light microscopy.

The glomeruli showed variably severe ischaemic changes but no evidence of necrosis or crescent formation. Vessels showed moderate degenerative changes only. There was no amylloid deposition.

Immunohistochemistry showed occasional mesangial deposits of IgM and C1q.

Electron microscopy revealed widespread acute on chronic tubulo-interstitial damage with prominent tubular crystals. The glomeruli showed segmental foot process fusion, variable glomerular capillary wall wrinkling, and prominent mesangial matrix. Tuft cellularity, the glomerular basement membrane, and endothelium were normal and there was no evidence of immune complex deposition.

**Discussion**

Primary hyperoxaluria occurs as a result of defects of the enzymes alanine glyoxalate amino transferase (type 1 or PH1) and D-glycerate dehydrogenase (type 2 or PH2), or as an idiopathic primary enteric hyperabsorption of oxalate (type 3 or PH3). PH1 presents in childhood with severe calculus disease, renal failure, and variable degrees of systemic oxalate deposition. Mild metabolic hyperoxaluria (PH3) can present in adult life with renal calculi and hyperoxaluria.

Secondary hyperoxaluria can occur as a result of pyridoxine deficiency, excessive oxalate precursor intake, and principally owing to enteric hyperabsorption of oxalate. Such hyperabsorption occurs in malabsorptive states, for example following extensive small bowel disease or resection or in pancreatic failure.

In such situations the increased concentrations of intraluminal free fatty acids competitively inhibit the precipitation of dietary oxalate and calcium by complexing the available intraluminal calcium ions, thereby leaving greater quantities of soluble uncomplexed oxalate for absorption. Additionally, excess bile salts and free fatty acids are toxic to the colonic mucosa and result in increased permeability to oxalate. These mechanisms can lead to an increase in dietary oxalate absorption from 10% to 40%.

Previous case reports have shown deposits of oxalate crystals associated with patchy tubulo-interstitial disease and variable degrees of atrophy and inflammatory reaction. In our patient, a scanty interstitial acute and chronic inflammatory cell infiltrate was noted, along with interstitial fibrosis and tubular atrophy. The clinical history included the use of diclofenac, a non-steroidal anti-inflammatory drug. However, we feel that the irregular use of this drug coupled with the lack of a tubulitis would make this very unlikely to be the cause of her renal failure.

Oxalate crystal deposits can be seen in the kidneys of patients with acute renal failure, but the degree is never as great as in patients with end stage chronic renal failure who have been treated by dialysis over a prolonged period. In our case, the renal failure was acute and the histological changes were not those of an end stage kidney. These findings would support the premise that the changes seen in the kidney were secondary to the deposition of oxalate crystals.
We have presented a case of a 54 year old woman who underwent jejuno-ileal bypass in 1994 and had suffered from diarrhoea postoperatively until her presentation on this occasion, 17 months later. No other predisposing cause of oxalate nephrosis was present. This case therefore serves to highlight the relation between jejuno-ileal bypass, malabsorption, hyperoxaluria, oxalate nephrosis, and its histological features. The striking amount of tubular cell cytoplasmic, luminal, and interstitial oxalate deposition could only be appreciated with polarisation of the biopsy. We suggest that polarisation be used in all cases of chronic tubulo-interstitial disease, and particularly those cases with a previous history of gastrointestinal surgery.

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