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Renal complications of jejuno-ileal bypass for obesity

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Summary

Jejuno-ileal bypass has until recently been an accepted treatment for refractory morbid obesity. Although hyperoxaluria causing renal tract calculi is a well-recognized complication, we describe eight patients who developed significant renal failure attributable to hyperoxaluria resulting from this procedure, three requiring renal replacement therapy. We review the literature, describing 18 other cases with renal failure, the mechanisms of hyperoxaluria and its treatment. Because reversal of the bypass may result in stabilization or partial improvement of renal function, these patients require long-term follow-up of renal function.

Introduction

Morbid obesity is rare but can have such devastating consequences that patients resistant to dietary advice and medical therapy were sometimes offered surgical bypass of the small bowel. This procedure was effective in reducing weight, and had other beneficial effects including mild reduction in blood pressure, improved glucose tolerance, and a reduction in serum cholesterol.[1] The operation entailed excluding the majority of the small bowel, leaving just 30–35 cm of jejunum and 10 cm of ileum in continuity. The remainder of the small bowel was anastamosed to the caecum as a 'blind loop', allowing the possibility of later reversal. There are unfortunately a number of complications of this operation, which are fully described elsewhere,[1–5] and for these reasons the operation is not now used. While nephrolithiasis from hyperoxaluria is well recognized,[3–12] the development of renal failure is less frequently described. We report eight patients who developed renal failure of varying severity following jejuno-ileal bypass.

Patients

Eight patients with varying degrees of renal impairment following jejuno-ileal bypass were identified from the databases of the Oxford and Bristol Kidney Units (Table 1). There was an equal sex distribution, and mean age at presentation was 58 years. The mean interval from surgery to presentation was 17 years (range 17 months–27 years).

Patient 1

After failure of conservative treatment for her obesity, a 54-year-old woman underwent a jejuno-ileal bypass, with 30 cm of small bowel left in continuity between the duodeno-jejunal flexure

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Table 1 Summary of clinical data and investigations in all eight patients

Patient	Gender	Age at bypass	Time from bypass to presentation with renal impairment	Level of oxaluria (μmol/day)*	Histology	Clinical course
1	F	54	17 months		Chronic interstitial nephritis, calcium oxalate crystals	Maintenance dialysis, bypass subsequently reversed
2	F	40	18 years		Chronic interstitial nephritis, calcium oxalate crystals	Maintenance dialysis
3	M	42	13 years		Chronic interstitial nephritis	Reversal of bypass. Creatinine 211 μmol/l
4	M	20	20 years	1090 before, 480 after	No histology	Bypass reversal. Creatinine 173 μmol/l.
5	M	28	25 years	1530	Chronic interstitial nephritis, calcium oxalate crystals	Creatinine 376 μmol/l
6	F	48	27 years		No histology	Creatinine 369 μmol/l
7	M	41	24 years	800–1200	No histology	Creatinine clearance stable at 37 ml/min
8	F	46	10 years	410–1000	No histology	Maintenance dialysis, transplant failed due to renal-artery thrombosis

*Normal range 110–440 μmol/day.

and the ileo-caecal valve. At 13 months, she presented with lethargy, and at 16 months, was found to be in renal failure. A renal biopsy showed chronic tubulointerstitial disease with deposition of birefringent calcium oxalate crystals (Figure 1). Within 17 months, she had reached end-stage renal failure and was established on continuous ambulatory peritoneal dialysis (CAPD).

Because she was persistently hypoalbuminaemic (albumin 25–27 g/l) with oedema despite adequate protein intake, she was switched to haemodialysis. At 28 months she developed complete heart block (possibly as a result of oxalate deposition in the conducting system) and required placement of a permanent pacemaker.

Three years after surgery, despite the switch to haemodialysis, she remained malnourished, and her jejuno-ileal bypass was therefore reversed. However, her eating disorder persists, with poor compliance to dietary restrictions of potassium, phosphate and fluid and with renewed weight gain of 38 kg over 3 years. To prevent interdialytic hyperkalaemia, she has been changed back to CAPD. The initial presentation and histological findings of this case have been reported previously.[13]

Patient 2

A 40-year-old woman underwent a jejuno-ileal bypass for obesity. She subsequently developed small-bowel obstruction due to intussusception which necessitated resection of the majority of her small bowel.

Following surgery, she suffered recurrent oxalate stones and recurrent urinary tract infections and had an episode of acute renal failure caused by obstruction and infection which required a left nephrectomy. Her renal function deteriorated, and 18 years after her initial surgery she started maintenance haemodialysis. One month after this, she developed obstructive pyonephrosis and septicaemia requiring right nephrectomy. Histology of the kidneys showed chronic interstitial nephritis with deposition of oxalate crystals. She died of an access-related infection 31 months after starting dialysis.

Patient 3

A 63-year-old man who developed obstruction and failure of his right kidney had undergone jejuno-ileal bypass for morbid obesity at the age of 42. This was followed by apronectomy and incisional hernia repair 4 years later. From 7 years after surgery, he suffered recurrent calcium oxalate stones. By 13 years, he was known to have a large right pelvic

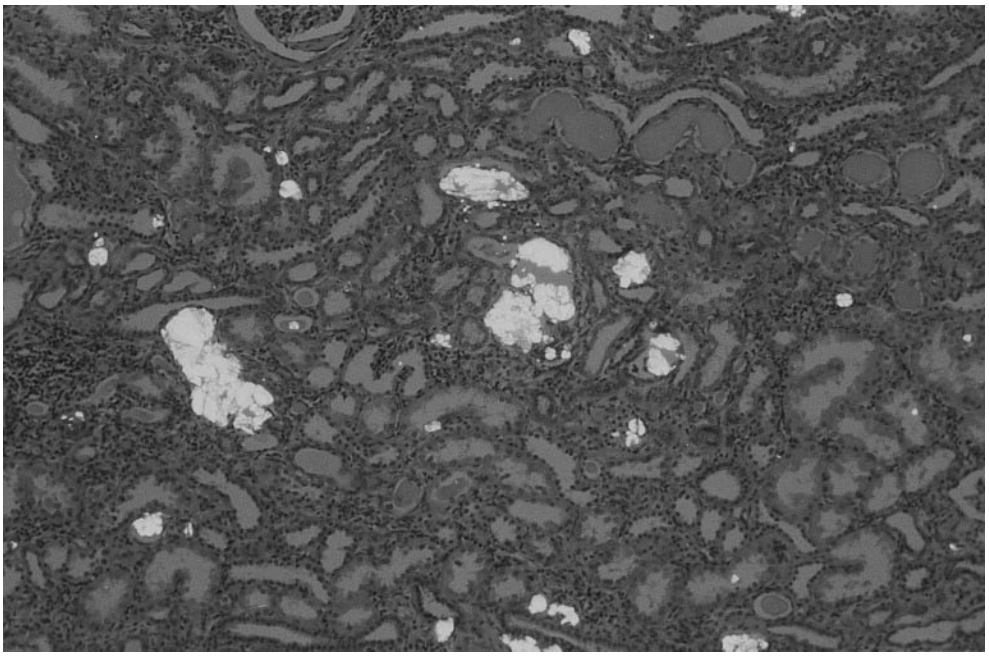

Figure 1. Renal histology from patient 1. H&E staining viewed through crossed polaroids demonstrating deposition of birefringent calcium oxalate in the interstitium.

calculus, and isotope renography showed less than 10% of normal function on this side. At 14 years, the bypass was reversed and a year later the right kidney was removed. Histology showed chronic changes but there was no comment on the presence of oxalate crystals. The creatinine has risen very slightly from 187 to 211 μmol/l over the 5 years of renal follow-up.

Patient 4

A 43-year-old man had undergone a jejuno-ileal bypass for morbid obesity at the age of 20. Despite this he still weighed 115 kg with a BMI of 29.6 kg/m^2. Twenty years after the surgery, he suffered renal colic secondary to bilateral renal calculi which required treatment with external shock-wave lithotripsy. He had mild chronic renal impairment with a creatinine of 178 μmol/l and a creatinine clearance of 78 ml/min. A renal biopsy was not performed, but 24-h urine showed a protein loss of only 0.24 g/day and an oxalate excretion of 1090 μmol/day (normal 110–440 μmol/day). The bypass was reversed, after which the oxalate excretion fell to 480 μmols/day. Sixteen months later, his plasma creatinine remains stable at 173 μmol/l.

Patient 5

This 54-year-old male had undergone a jejuno-ileal bypass for morbid obesity at the age of 28. His weight had fallen dramatically following bypass, but then stabilized. At the age of 53 he developed severe acute pancreatitis complicated by Gram-negative sepsis requiring cardiorespiratory support. At discharge, his serum creatinine was 177 μmol/l. However a month later he was treated for an *E. coli* urinary tract infection and pyelonephritis. His serum creatinine was 322 μmol/l. Two weeks later (26 years after his jejuno-ileal bypass) he developed confusion, renal failure (creatinine 502 μmol/l), and a severe metabolic acidosis (arterial pH 7.07, pCO_2 1.2 kPa, venous bicarbonate 2.7 mM). Microscopy of his urine revealed numerous calcium oxalate crystals (Figure 2) consistent with hyperoxaluria, which was quantified at 1530 μmol/24 h (normal 110–440 μmol/day). He underwent haemofiltration to correct the acidosis, followed by sodium bicarbonate supplementation. His creatinine stabilized at 376 μmol/l. A renal ultrasound revealed kidneys of 10.5 cm bilaterally but no stones or hydronephrosis. A renal biopsy confirmed chronic interstitial nephritis with interstitial deposition of calcium oxalate crystals. He has been lost to follow-up since moving overseas.

Patient 6

A 77-year-old female had undergone a jejuno-ileal bypass for obesity at the age of 48. Her weight fell from 106 kg to 76 kg. Four years after her bypass, she required repair of a hernia and also underwent excision of abdominal skin fold. At 27 years after

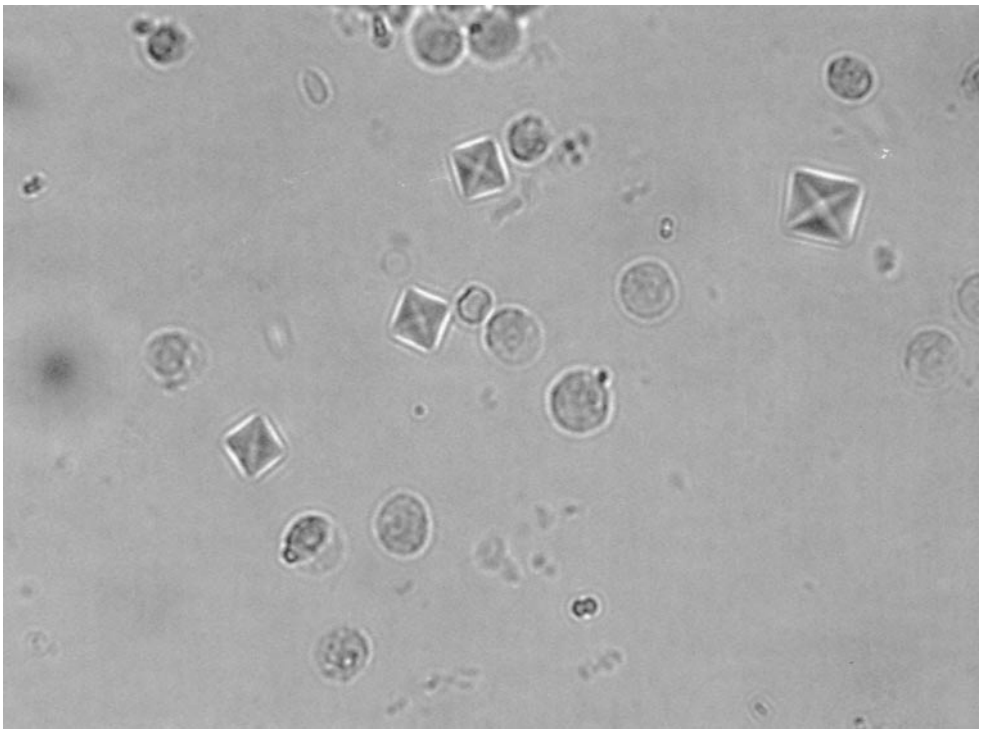

Figure 2. Urine microscopy from patient 5 showing numerous bipyramidal calcium oxalate crystals.

her operation, she was found to have chronic renal failure with a creatinine of 421 μmol/l. Ultrasound showed small kidneys of 9.4 and 8.9 cm, with cortical thinning, precluding renal biopsy. A clinical diagnosis of oxalate nephropathy was made. Her latest creatinine is 369 mol/l.

Patient 7

Twenty-four years after a jejuno-ileal bypass for chronic morbid obesity, a 65-year-old man presented with severe renal impairment (creatinine 1047 μmol/l). Renal impairment was first noted at the age of 64, but was not investigated further. Ultrasound showed normal-sized kidneys with right-sided hydronephrosis. Plain radiology showed multiple stones on both sides. Bilateral retrograde pyelograms showed an obstructing ureteric stone on the right, but no evidence of obstruction on the left. He was successfully treated with extracorporeal shock wave lithotripsy. Following partial recovery of renal function, urinary oxalate excretion was 1300 μmol/24 h. He was advised to maintain a high fluid intake and tight restriction of dietary oxalate intake, and was prescribed calcium carbonate with meals (2 g elemental calcium t.d.s) together with codeine phosphate to minimize gut fluid losses. Despite this, urinary oxalate excretion has remained between 800 and 1200 μmol/24 h, with no reduction after a trial of pyridoxine supplementation. Renal function remains stable with a serum creatinine of 312 μmol/l and a calculated creatinine clearance of 37 ml/min.

Patient 8

A 62-year-old woman who had undergone jejuno-ileal bypass at the age of 46 presented with end-stage renal failure. Her right kidney had been removed, prior to her bypass, at the age of 32, because of non-function and infection associated with a staghorn calculus. Ten years after the bypass operation she was found to be in end-stage renal failure. No evidence of obstruction or urinary tract stone was found at that time, and a renal biopsy was not performed. A clinical diagnosis of oxalate nephropathy was made. Two years after starting haemodialysis, she underwent successful cadaveric renal transplantation. Severe hyperparathyroidism necessitated parathyroidectomy 20 months later, complicated by severe hypocalcaemia thought to be due to calcium malabsorption. Urine oxalate excretion varied between 410 and 1000 μmol/24 h, the lowest levels being associated with very high doses of oral calcium supplements (12 g elemental calcium daily). Her transplant failed suddenly after 41 months as a result of renal artery thrombosis, and she was re-established on haemodialysis. On standard haemodialysis, pre-dialysis plasma creatinine was 629 μmol/l and oxalate was 93.6 μmol/l (reference range 0–3 μmol/l, enzymic assay), much higher than normally seen in patients

on haemodialysis.[15] Her dialysis dose was subsequently increased to give a calculated Kt/V of 1.7, after which pre-dialysis plasma oxalate concentration was 39.8 μmol/l.

Discussion

The association between jejuno-ileal bypass, hyperoxaluria and renal calculi is well recognized, with rates of between 4% and 29% (Table 2).[3–12] Although hereditary forms of hyperoxaluria are well known to cause chronic renal failure, reports of chronic renal failure after jejuno-ileal bypass are remarkably uncommon. Early descriptions of the outcome of jejuno-ileal bypass surgery failed to recognize this complication at all.[1,2,5,11,12,14] The first description of oxalate nephropathy causing renal failure following jejuno-ileal bypass was by Cryer *et al*. in 1975.[16] Since then, there have been a further 17 cases in the literature (summarized in Table 3), making a combined total of 26.

Our series is the only one to report within the last decade and to include cases presenting more than 10 years after bypass. It is likely that this complication is under-reported.

This concern is strengthened by the data of Drenick *et al*.,[21] who published a series of renal biopsies on 18 patients who had undergone jejuno-ileal bypass for morbid obesity. Although only four had evidence of impaired renal function at the time, all showed evidence of tubulo-interstitial damage, and 8/18 had deposition of oxalate crystals within the interstitium. There are no data on the long-term follow-up of these patients.

All the published cases in whom measurements were made have had a raised urinary oxalate excretion. These however are not markedly raised and, in Drenick's series,[21] did not distinguish between patients with and without renal failure. Moreover, urinary oxalate excretion becomes increasingly unreliable as a way of detecting increased oxalate absorption as renal function declines.

The surgical reversal of a jejuno-ileal bypass is usually straightforward. There can be some disproportion between the proximal small bowel and the defunctioned sections, and appropriate measures must be taken to ensure optimal fluid balance so that a good urine output is maintained.

Mechanism of hyperoxaluria

Oxalate is a useless end-product of metabolism in man, produced by one of two main pathways, the ascorbic acid pathway or the glyoxylate pathway. It is also absorbed from the bowel (Figure 3). Its calcium salt is ~1000 times less soluble than the sodium form, explaining why it is calcium oxalate which precipitates. Absorption of dietary oxalate is normally limited by the formation of insoluble precipitates of calcium oxalate in the bowel. In normal fasting subjects, 2.3–12% of an oral oxalate load is absorbed by the bowel.

Two studies in patients with extensive ileal resection[27,28] and one in patients with jejuno-ileal bypass[29] have shown an increase in absorption of oxalate as assessed by urinary excretion of an oral load of ^{14}C oxalate. Chadwick *et al*. investigated the absorption of bile salt precursors of oxalate by measuring oxalate excretion after oral ^{14}C-cholyl glycine, and i.v. ^{14}C-glyoxylate administration.[27] Neither led to increased urinary excretion of ^{14}C oxalate, suggesting that biliary cholyl glycine and endogenous glyoxylate do not contribute to the hyperoxaluria. This is supported by the data of Stauffer *et al*., who showed that binding bile salts with cholestyramine did not reduce hyperoxaluria in patients with extensive ileal resection.[30]

Earnest *et al*. demonstrated a direct correlation between dietary fat malabsorption and urinary oxalate excretion.[28] Increasing the dietary fat content

Table 2 Previous reports of renal calculi complicating jejuno-ileal bypass

Authors	Patients (*n*)	Duration of follow-up (months)	Calculi (%)	Composition
Dickstein *et al*.[6]	34	NG	29	6/6 analysed were Ca oxalate
Fikri & Cassella[7]	52	NG	10	All Ca oxalate
O'Leary *et al*.[8]	31	6–66**	23	4/4 analysed were Ca oxalate
Wise & Stein[9]	93	17.6*	4	All Ca oxalate
Jewell *et al*.[3]	52	6–42**	6	All Ca oxalate
Drenick *et al*.[5]	26	NG	17	All Ca oxalate
Thomas & Madura[10]	172	NG	10–14	7/7 analysed were Ca oxalate
Gregory *et al*.[11,12]	543	12–72**	12	30/32 analysed were Ca oxalate
Halverson *et al*.[1]	101	32.1*	7	Not stated

*Mean; **range. NG, not given.

Table 3 Summary of previously reported cases of renal failure following jejuno-ileal bypass surgery

Reference	Age at time of renal failure	Gender	Time from bypass to renal failure (months)	Level of oxaluria (mg/dl)	Histology	Clinical course
Cryer *et al.*[16]	41	M	35	69	Interstitial nephritis, calcium oxalate crystals	Reversal of bypass, GFR 15, CrCl rose from 13–15 to 23 ml/min
Barbour *et al.*[17]		M	24	56		Reversal of bypass, long-term dialysis
		Not given	48			Reversal of bypass, long-term dialysis
Ehlers *et al.*[18]	58	M	48	113	Interstitial nephritis, calcium oxalate crystals	Reversal of bypass, CrCl rose from 9 to 23 ml/min
Gelbart *et al.*[19]	45	M	20		Interstitial nephritis, calcium oxalate crystals	Long-term dialysis
Miller *et al.*[20]	50	F	60	49	IVP-nephrocalcinosis	CrCl 30 ml/min and declining at time of report
Drenick *et al.*[21]	38	F	22	Anuric	Interstitial nephritis, calcium oxalate crystals	
	41	M	36	250	Interstitial nephritis, calcium oxalate crystals	
	60	M	63	222	Interstitial nephritis	
	53	M	45	210	Interstitial nephritis, calcium oxalate crystals	
Cryer & Kissane[22]	56	M	30		Interstitial nephritis, calcium oxalate crystals	Reversal of bypass, GFR stabilized at 9 ml/min
Das *et al.*[23]	48	M	54		Oxalate nephropathy	Long-term dialysis
	50	M	18	56	Oxalate nephrosis	Long-term dialysis, reversal of bypass
	35	F	13	63	Oxalate nephrosis	Long-term dialysis
	37	F	6		Oxalate nephropathy	Long-term dialysis
Bischel *et al.*[24]	39	F	60	26	Granulomatous interstitial nephritis, oxalate crystals	Creatinine fell to 1.8 g/dl prior to reversal of bypass, then stable
Canos *et al.*[25]	54	F	20	45	Interstitial nephritis, calcium oxalate crystals	Reversal of bypass, CrCl rose from 32 to 44 ml/min
Verani *et al.*[26]	58	F	108	61	Granulomatous interstitial nephritis, calcium oxalate crystals	Reversal of bypass, creatinine fell from 3.4 mg/dl to 2.1 mg/dl

led to a further increase in hyperoxaluria. Conversely, reducing fat or oxalate intake or increasing calcium intake reduced urinary oxalate excretion. Furthermore, other conditions causing steatorrhoea (e.g. chronic pancreatitis) may also lead to hyperoxaluria. This led to the suggestion that the increased free fatty acids in the gut bind to calcium, inhibiting the formation of insoluble calcium oxalate. This has the effect of maintaining the solubility of the oxalate and rendering it available for absorption.

Hofmann *et al.* showed that the excretion of orally administered ^{14}C-oxalate was far slower in patients than controls, and suggested that this was due to colonic absorption.[29] Dobbin and Binder, who have studied urinary oxalate excretion and bowel absorption of ^{14}C-oxalate in a variety of gastrointestinal diseases with steatorrhoea, showed that an intact colon was necessary for increased absorption and excretion of oxalate.[31] In Sprague-Dawley rats, the presence of bile salts in the colon increased colonic permeability and hence absorption of oxalate independently of calcium levels.[32] This suggests that the increased absorption of oxalate following jejuno-ileal bypass occurs in the colon as a result of increased delivery of soluble oxalate and possibly increased colonic permeability. The role of unabsorbed bile salts in increasing colonic permeability was demonstrated in man by Fairclough *et al.*, who demonstrated increased absorption of radiolabelled oxalate in the presence of chenodeoxycholate during retrograde perfusion of the colon in three patients who had undergone colonic exclusion for hepatic encephalopathy.[33]

Oxalate is excreted both by glomerular filtration and tubular secretion. As renal function declines, oxalate clearance also falls, causing higher plasma and tissue levels, which eventually cause accelerating tissue deposition of calcium oxalate. In the late stages of chronic renal failure, absolute oxalate excretion falls, probably as a result of tissue deposition, making estimation of 24-h oxalate clearance unreliable for the diagnosis of hyperoxaluria in this situation.[34] The risk factors for developing nephropathy, as opposed to calculi, are however not known.

Treatment

Given the mechanism of hyperoxaluria following jejuno-ileal bypass, it is not surprising that strategies to reduce oxalate production such as pyridoxine, allopurinol, or reducing ascorbate intake have had little effect.[35] Dietary restrictions of oxalate or fat are obvious measures, but oxalate is present in a wide range of foodstuffs, and patients who have undergone jejunoileal bypass for obesity are unlikely to be able to adhere to tight restriction of dietary fat intake.[8,27,36]

Strategies to increase binding of oxalate in the bowel and prevent its absorption are attractive alternatives. Cholestyramine has been used to bind oxalate in the gut and reduce hyperoxaluria, but with varying results.[8,14,30,37] There are, however, no data to show that this will prevent nephrolithiasis or renal impairment. Four short-term studies have examined the effects of oral calcium supplementation following jejuno-ileal bypass,[37,38] in pancreatic insufficiency,[39] and in a variety of conditions leading to enteric hyperoxaluria.[40] Three showed a significant fall in urinary oxalate excretion of approximately 50%. However, no data on renal function or nephrolithiasis were provided.

In six reported cases of oxalate nephropathy, and two further cases in this series, bypass was reversed before the onset of ESRF (Table 3). Of these eight cases, only five have provided data on urinary oxalate excretion, all showing a significant fall. Three patients showed an improvement in creatinine clearance, with two showing stabilization. A further patient showed a fall in serum creatinine from 299 μmol/l to 185 μmol/l. However, the two patients described by Barbour[17] showed a progressive decline in renal function, both eventually reaching end-stage renal failure. Thus an important aspect of managing these patients in the early stages of their renal failure is reversal of the bowel diversion.

Apart from patient 8, there are only two other recorded cases of renal transplantation for oxalate nephropathy from enteric causes. One was following ileal resection rather than bypass.[41] Despite poor absorption of cyclosporin, the graft was still functioning well after 7 years. The second case was a patient following jejuno-ileal bypass, and was performed because of failure to comply with salt and water restriction.[16] He died from acute pulmonary oedema 36 h after discharge from hospital.

In summary, jejuno-ileal bypass surgery for morbid obesity may result in hyperoxaluria, which commonly leads to obstructing renal calculi, but may also lead to renal failure through interstitial deposition of calcium oxalate and, chronic interstitial nephritis. This may occur at any stage after bypass, and it is likely that this complication is being under-reported. The mechanism of hyperoxaluria is through fat malabsorption and increased delivery of faecal fat to the colon. The fatty acids bind calcium to form soaps, leading to an increase in the concentration of the more soluble sodium oxalate. It is therefore possible that any of the recently introduced pharmacological measures to reduce weight by reducing intestinal fat absorption will also result in a similar complication. Strategies

to bind oxalate in the bowel may reduce urinary oxalate excretion, but there are no data on the effect of this on the rate of renal calculi formation or renal function. The only effective treatment for the nephropathy is reversal of the bypass which results in stabilization or partial improvement of renal function in the majority of patients. It is therefore suggested that patients who undergo jejuno-ileal bypass procedures have regular follow-up of renal function with reversal of the procedure if interstitial nephritis and renal impairment develops.

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