Journal of
Medical Case Reports

CASE REPORT

Open Access

Oxalate nephropathy causing renal failure in small bowel Crohn's disease: a case report

Aishwarya Samir Chitnis[1], Saumya Gupta[2], Ian S. Roberts[3] and David A. Gorard[1*]

Abstract

Background Crohn's disease of the small intestine typically causes abdominal symptoms and nutritional problems, but renal consequences are rare.

Case presentation We report a 70 year-old British Caucasian man with longstanding Crohn's disease of the small intestine complicated by multiple fibrotic strictures, who developed a rapid deterioration in his renal function around the same time as initiation of biologic therapy.

Following investigation into the cause of his renal deterioration and eventually a renal biopsy, he was diagnosed with oxalate nephropathy leading to end-stage renal failure with creatinine peaking at 604 umol/l. The pathophysiology of enteric hyperoxaluria in Crohn's disease is discussed.

Conclusion Awareness of hyperoxaluria and potential oxalate nephrotoxicity in patients with small intestinal Crohn's and an intact colon should help clinicians advise such patients on dietary and fluid intake modification at an early stage.

Keywords Oxalate, Crohn's disease, Renal failure, Case report

Background

Crohn's disease is an inflammatory disease of the gastrointestinal tract, most commonly involving the small intestine and/or colon. In addition to abdominal symptoms (diarrhea, rectal bleeding, abdominal pain, perianal sepsis) and nutritional problems (weight loss, anemia, vitamin deficiencies), Crohn's disease can cause a multitude of extraintestinal symptoms including arthropathy, skin and eye problems, fevers, mouth ulcers and fatigue. Kidney problems are not typically considered a consequence of Crohn's disease.

*Correspondence:
David A. Gorard
david.gorard@nhs.net
[1] Gastroenterology Department, Stoke Mandeville Hospital, Buckinghamshire NHS Trust, Aylesbury HP21 8AL, UK
[2] Nephrology Department, Oxford University Hospitals NHS Trust, Oxford OX3 9DU, UK
[3] Department of Cellular Pathology, John Radcliffe Hospital, Oxford OX3 9DU, UK

Case presentation

A 70 year-old British Caucasian man with longstanding Crohn's disease reported increased bowel frequency and was found to be anemic when attending his annual gastroenterology clinic appointment.

He had been diagnosed with ileal Crohn's disease in his early twenties at a laparotomy for presumed appendicitis. Following a right hemicolectomy, he had a stormy hospital course complicated by the need for further surgery and a temporary ileostomy, following which he had further surgery to re-establish intestinal continuity. Over the subsequent decades he had recurrent adhesional small bowel obstructive episodes before entering a long period of apparent Crohn's disease quiescence. During many years of apparent Crohn's disease inactivity, he opened his bowels 2–3 times daily, passing formed stool without urgency. At times when he experienced excess borborygmi and his abdomen felt gaseous, he empirically received courses of metronidazole for presumed small intestinal bacterial overgrowth. His inflammatory

© The Author(s) 2025. **Open Access** This article is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License, which permits any non-commercial use, sharing, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if you modified the licensed material. You do not have permission under this licence to share adapted material derived from this article or parts of it. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit http://creativecommons.org/licenses/by-nc-nd/4.0/.

markers were persistently unremarkable but there was evidence of small bowel stricturing on cross-sectional imaging (Fig. 1). Since his disease seemed to be clinically inactive and there were no objective blood markers of significant inflammation (C-reactive protein (CRP) persistently 1–8 mg/l) he had not previously been treated with immunomodulator or biologic treatments.

Apart from Crohn's disease he had a history of asthma, eczema and a left inguinal hernia repair. His regular medications were 20 mg omeprazole daily, a vitamin D tablet daily and 3-monthly vitamin B12 injections. He was a nonsmoker and consumed little alcohol.

At his clinic review he reported 2–6 loose bowel actions daily, fatigue and some weight loss. His blood tests showed iron deficiency anemia with haemoglobin 61 g/l (serum ferritin18 ng/ml) and normal white cell and platelet counts. His serum albumin was 25 g/l and CRP 6.1 mg/l. Renal function tests showed unremarkable serum creatinine at 86 umol/l and eGFR 80 ml/min.

He received a blood transfusion and iron infusion but his iron deficiency anemia kept recurring over the subsequent months. A gastroscopy and colonoscopy failed to identify any bleeding source. Despite his unremarkable CRP measurements, his fecal calprotectin concentration rose to 420 ug/g. Although repeat Magnetic Resonance Enterography showed little inflammation associated with his small bowel Crohn's strictures, it was considered most likely that he had active small bowel Crohn's disease accounting for his symptom deterioration, recurrent iron deficiency anemia and elevated fecal calprotectin concentration. Therefore he was started on azathioprine and infliximab.

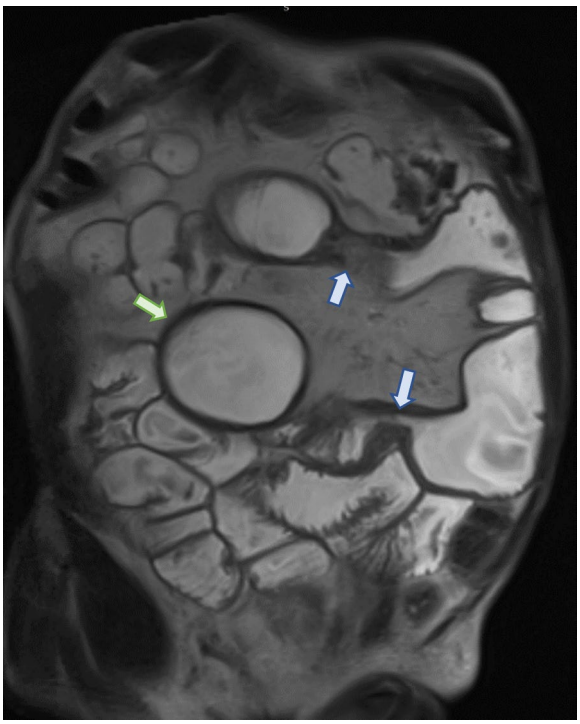

Fig. 1 Magnetic resonance enteroclysis image showing multiple smooth diffuse small intestinal strictures (blue arrows) with mild wall thickening, mesenteric fat hypertrophy and minimal inflammation. There is marked intestinal dilatation with pseudo-diverticulation (green arrow), consistent with long standing fibro stenotic disease

Blood testing 4 months before commencing Azathioprine and Infliximab unexpectedly showed a decline in his renal function with serum creatinine 137 umol/l (previously 80–90 umol/l) and eGFR 57 ml/min (previously 70–90 ml/min). There had been no worsening of diarrheal symptoms associated with this and his blood pressure was unremarkable. An initial fluid challenge did not improve his renal function. Stopping his omeprazole (in case proton pump inhibitor therapy was contributory) did not improve his renal function.

Over the subsequent 15 months, his serum creatinine climbed to 500 umol/l and peaked at 604 umol/l. His eGFR gradually dropped to 9 ml/min. This deteriorating renal function was associated with increased fatigue and poor appetite.

Investigations into the etiology of his renal decline included renal imaging (ultrasound scanning and non-contrast computed tomography (CT) scanning), which ruled out any obstructive pathology and showed no renal calculi. Myeloma screening and vasculitis (anti-nuclear antibody, anti-neutrophil cytoplasmic antibody, anti-glomerular basement membrane antibody) screening, were all negative. His blood pressure was not elevated and was typically 105–115/70–80 mmHg. Urinalysis showed no hematuria, casts or proteinuria.

A renal biopsy showed acute tubular injury with intratubular oxalate crystals. It showed seven glomeruli with two of them showing sclerosis. There was moderate chronic tubule-interstitial damage with 50% of the cortex showing interstitial fibrosis and tubular atrophy. There was no evidence of a glomerulonephritis or tubulointerstitial nephritis (Fig. 2). A diagnosis of oxalate nephropathy was made.

Once oxalate nephropathy was diagnosed, a low-oxalate diet was commenced and and an increase in his oral fluid intake advised. However the low-oxalate diet and efforts to drink more fluid each day had little beneficial effect on his renal function. An arteriovenous fistula was inserted in preparation for dialysis, but his worsening renal function then plateaued. He is currently being regularly followed up in the renal clinic and is awaiting dialysis. His Crohn's disease symptoms have stabilized with azathioprine and infliximab, although he has not gained weight.

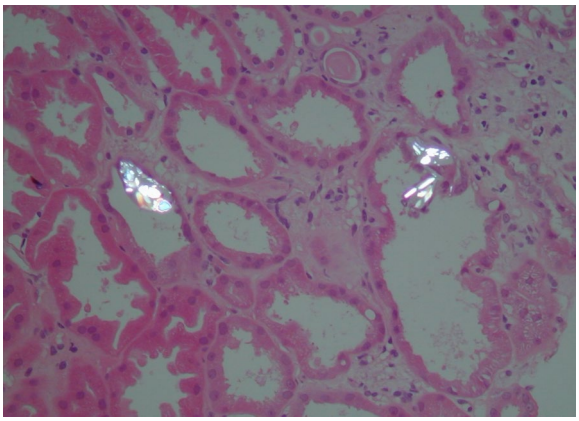

Fig. 2 Renal biopsy (polarized hematoxylin and eosin stain, 20 × objective magnification) showing dilated renal tubules containing birefringent calcium oxalate crystals

Discussion

Oxalate is an end product of amino acid metabolism, but dietary absorption of oxalate is also a major contributor to serum (and consequently urinary) oxalate levels. Oxalate nephropathy results from elevated serum and urine oxalate levels with consequent deposition of damaging calcium oxalate crystals in the renal tubules [1, 2].

Primary hyperoxaluria (a group of autosomal recessive metabolic disorders causing hepatic overproduction of oxalate), and excessive dietary ingestion of oxalate-rich foods, as causes of oxalate nephropathy are not discussed here [3].

In distinction our Crohn's disease patient had enteric hyperoxaluria. Factors in understanding the underlying pathophysiology in enteric hyperoxaluria include recognition that the colon is an important site of oxalate absorption and that enteric hyperoxaluria primarily results from fat malabsorption.

Normally, calcium binds to dietary oxalate in the small intestine and the insoluble calcium oxalate is excreted in the feces. In conditions associated with fat malabsorption (steatorrhea), free fatty acids bind with luminal calcium, making it unavailable for binding with oxalate. [4] Large amounts of soluble free oxalate then pass into the colon and passively diffuse from the colon into plasma. Furthermore, increased luminal concentrations of fatty acids and bile salts arriving in the colon will increase the permeability of the colonic mucosa to oxalate. The increased quantities of plasma oxalate reach the kidneys for excretion, resulting in hyperoxaluria [5].

A long period of increased urinary calcium oxalate concentration results in the formation and retention of calcium oxalate crystals in renal tubules, causing progressive renal injury and / or oxalate renal stones [6].

Crohn's disease of the small intestine is the most common cause of fat malabsorption leading to enteric hyperoxaluria [7, 8] .Since the colon is the site of the excess oxalate absorption, it is only Crohn's disease patients with small bowel disease / resections who have an intact colon that are prone to hyperoxaluria. Crohn's disease patients with small bowel disease/resections and an ileostomy will not be prone to hyperoxaluria.

In Crohn's disease, enteric hyperoxaluria more commonly causes oxalate renal stones which may be symptomatic or incidentally detected on imaging. [9] Nephropathy with progression to renal failure as in the patient reported here, is far less common.

Management strategies in enteric hyperoxaluria are largely based around dietary modifications. These include a high fluid intake to aim for a daily urine output of more than 2–3 L. Such a high urine output reduces urinary supersaturation with oxalate, which has been shown to reduce stone formation [10].

Dietary measures to reduce intestinal oxalate absorption include a low-oxalate, low-fat, and normal calcium diet. [11] Foods rich in oxalates, such as spinach, rhubarb, collard greens, nuts, beets, and tea should be restricted. Although high dietary oxalate has been thought to play a relatively small (20%) contributory role in oxaluria, [12, 13] it is likely to be a more significant factor in the pathophysiology of enteric hyperoxaluria.

While there is no need to restrict dietary vitamin C, patients with hyperoxaluria should be dissuaded from taking high-dose vitamin C supplements. Excess vitamin C supplementation is metabolized to oxalate and can contribute to hyperoxaluria [14].

Since dietary calcium binds with oxalate to form insoluble calcium oxalate that is then excreted in feces, patients should maintain a normal dietary calcium intake and should not restrict calcium.

The bile salt sequestrant, cholestyramine should theoretically reduce gut absorption of oxalate by binding it directly as well as by binding bile salts. However convincing reductions in colonic oxalate absorption have not been demonstrated in human studies [15].

Most recently, oxalate decarboxylase, an oxalate-degrading enzyme has been investigated as an oral agent to lower urinary oxalate excretion with initial trial evidence of efficacy [16].

Patients with small bowel Crohn's disease and a colon in continuity, particularly where there is suspected steatorrhea, should be considered for early introduction of a low oxalate, low fat diet in addition to a high oral fluid intake. Such measures may help to prevent renal damage from hyperoxaluria. In reality, however, many patients with small bowel Crohn's disease and steatorrhea are malnourished and restricting dietary fat may worsen

their nutritional state. The development of oxalate decarboxylase drugs will be a welcome adjunct since despite dietary/ fluid interventions, renal replacement therapy is required for patients developing end-stage renal failure [17].

Conclusion

Hyperoxaluria should be considered as a cause of impaired renal function in patients who have a history of small intestinal Crohn's disease and a colon in continuity. Early consideration of hyperoxaluria will allow dietary strategies, which may mitigate subsequent renal decline.

Acknowledgements
Not applicable

Author contributions
AC wrote the first draft of the manuscript. IR performed the histological examination of the kidney biopsy. SG and DG amended the manuscript. All authors read and approved the final manuscript. DG is guarantor and corresponding author.

Funding
Not applicable.

Data availability
Not applicable.

Declarations

Ethics approval and consent to participate
Ethical approval not required.

Consent for publication
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests
The authors declare that they have no competing interests.

Received: 10 January 2025 Accepted: 10 October 2025
Published online: 21 November 2025

References

1. Rosenstock JL, Joab TMJ, DeVita MV, Yang Y, Sharma PD, Bijol V. Oxalate nephropathy: a review. Clin Kidney J. 2021;15(2):194–204. https://doi.org/10.1093/ckj/sfab145.
2. Buysschaert B, Aydin S, Morelle J, *et al*. Etiologies, clinical features, and outcome of oxalate nephropathy. Kidney Int Rep. 2020;5:1503–9.
3. Hoppe B. An update on primary hyperoxaluria. Nat Rev Nephrol. 2012;8(8):467–75. https://doi.org/10.1038/nrneph.2012.113.
4. Dobbins JW, Binder HJ. Effect of bile salts and fatty acids on the colonic absorption of oxalate. Gastroenterology. 1976;70(6):1096–100.
5. Hylander E, Jarnum S, Jensen HJ, Thale M. Enteric hyperoxaluria: dependence on small intestinal resection, colectomy, and steatorrhoea in chronic inflammatory bowel disease. Scand J Gastroenterol. 1978;13(5):577–88. https://doi.org/10.3109/00365527809181767.
6. Robijn S, Hoppe B, Vervaet BA, D'Haese PC, Verhulst A. Hyperoxaluria: a gut-kidney axis? Kidney Int. 2011;80(11):1146–58. https://doi.org/10.1038/ki.2011.287.
7. Lumlertgul N, Siribamrungwong M, Jaber BL, Susantitaphong P. Secondary oxalate nephropathy: a systematic review. Kidney Int Rep. 2018;3(6):1363–72. https://doi.org/10.1016/j.ekir.2018.07.020.
8. Nazzal L, Puri S, Goldfarb DS. Enteric hyperoxaluria: an important cause of end-stage kidney disease. Nephrol Dial Transplant. 2016;31(3):375–82. https://doi.org/10.1093/ndt/gfv005.
9. Gaspar SR, Mendonça T, Oliveira P, Oliveira T, Dias J, Lopes T. Urolithiasis and Crohn's disease. Urol Ann. 2016;8(3):297–304. https://doi.org/10.4103/0974-7796.184879.
10. Borghi L, Guerra A, Meschi T, *et al*. Relationship between supersaturation and calcium oxalate crystallization in normal and idiopathic calcium oxalate stone formers. Kidney Int. 1999;55:1041–50. https://doi.org/10.1046/j.1523-1755.1999.0550031041.x.
11. Witting C, Langman CB, Assimos D, *et al*. Pathophysiology and treatment of enteric hyperoxaluria. Clin J Am Soc Nephrol. 2021;16:487–95. https://doi.org/10.2215/CJN.08000520.
12. Holmes RP, Goodman HO, Assimos DG. Contribution of dietary oxalate to urinary oxalate excretion. Kidney Int. 2001;59(1):270–6. https://doi.org/10.1046/j.1523-1755.2001.00488.x.
13. Taylor EN, Curhan GC. Determinants of 24-hour urinary oxalate excretion. Clin J Am Soc Nephrol. 2008;3(5):1453–60. https://doi.org/10.2215/CJN.01410308.
14. Traxer O, Huet B, Poindexter J, Pak CY, Pearle MS. Effect of ascorbic acid consumption on urinary stone risk factors. J Urol. 2003;170(2 Pt 1):397–401. https://doi.org/10.1097/01.ju.0000076001.21606.53.
15. Caspary WF, Tönissen J, Lankisch PG. "Enteral" hyperoxaluria. Effect of cholestyramine, calcium, neomycin, and bile acids on intestinal oxalate absorption in man. Acta Hepatogastroenterol. 1977;24(3):193–200.
16. Lieske JC, Lingeman JE, Ferraro PM, Wyatt CM, Tosone C, Kausz AT, Knauf F. Randomized placebo-controlled trial of Reloxaliase in enteric hyperoxaluria. N Engl J Med Evid. 2022;1(7): EVIDoa2100053. https://doi.org/10.1056/EVIDoa2100053.
17. Asplin JR. The management of patients with enteric hyperoxaluria. Urolithiasis. 2016;44(1):33–43. https://doi.org/10.1007/s00240-015-0846-5.

Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.