TA1

Multi-site Calciphylaxis with Pulmonary Calcification in a Young Haemodialysis Patient

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TUESDAY - Moderated Poster Session, HALL Q, March 10, 2026, 16:00 - 17:00

Background:
Calciphylaxis is a rare but life-threatening complication of end-stage renal failure (ESRF), usually affecting the skin and subcutaneous tissues. Pulmonary involvement is uncommon and may represent metastatic pulmonary calcification (MPC) or pulmonary calciphylaxis, the latter typically requiring histological confirmation and associated with an extremely poor prognosis. We describe a young haemodialysis (HD) patient with cutaneous and penile calciphylaxis and concurrent pulmonary calcification, highlighting the diagnostic value of bone scintigraphy in differentiating these entities.

Case Presentation:
A 34-year-old man with a congenital horseshoe kidney and recurrent bladder urothelial carcinoma developed ESRF from obstructive uropathy. He commenced HD in September 2023 but demonstrated prolonged non-adherence, including a four-month period without dialysis. In February 2025, after three weeks without HD, he presented with lethargy, weakness, haematuria, and severe hyperkalaemia (8.6 mmol/L). Despite understanding the risks, he intermittently refused dialysis during admission.

He subsequently developed painful necrotic plaques on the thighs and penile shaft, clinically consistent with calciphylaxis. Concurrently, he experienced progressive breathlessness and cough. CT chest revealed bilateral ground-glass opacities with septal thickening; bronchoscopy and microbiology were negative. Persistent abnormalities prompted bone scintigraphy, which demonstrated increased tracer uptake in the lungs and multiple skeletal sites, confirming MPC.

Management & Outcome:
He was treated with intensified HD using low-calcium dialysate, intravenous sodium thiosulphate, and non-calcium phosphate binders. Broad-spectrum antibiotics were given empirically for concurrent infections. Pulmonary symptoms improved with optimised dialysis and medical therapy; however, cutaneous and penile lesions remained painful with poor healing. Multidisciplinary discussions concluded that prognosis was poor due to multi-site calcification, active bladder cancer, and ongoing dialysis non-adherence. He was referred to palliative care, and ward-based ceilings of care were agreed.

Discussion & Conclusion:
This case illustrates rare multi-site calciphylaxis with concurrent pulmonary calcification in a young ESRF patient. Although both calciphylaxis and MPC share risk factors such as hyperphosphataemia and secondary hyperparathyroidism, they are pathologically distinct. Bone scintigraphy was invaluable in supporting a diagnosis of MPC over pulmonary

calciphylaxis, avoiding invasive biopsy. Importantly, dialysis non-adherence was the major modifiable driver of mineral imbalance and calcification burden. Despite aggressive intervention, outcomes in extensive multi-site disease remain poor, underscoring the need for early recognition, patient engagement, and multidisciplinary care.

Learning Points:
1. Calciphylaxis can involve unusual sites including the penis and may coexist with pulmonary calcification.
2. In ESRF patients with pulmonary opacities, both MPC and pulmonary calciphylaxis should be considered.
3. Bone scintigraphy is a sensitive, non-invasive tool to identify MPC and differentiate it from pulmonary calciphylaxis.
4. Dialysis non-adherence is a key modifiable risk factor for severe mineral imbalance and widespread calcification.
5. Prognosis in extensive calciphylaxis with visceral involvement remains poor despite aggressive management.

TA2

Obinutuzumab as salvage therapy for rituximab-refractory MPO-ANCA vasculitis in a kidney transplant recipient: a case report

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Introduction

Relapsed MPO-ANCA vasculitis after kidney transplantation represents a therapeutic dead-end when rituximab fails, as infection risk, cumulative immunosuppression, and graft preservation constrain alternatives. Type II anti-CD20 antibodies such as obinutuzumab achieve deeper, more sustained B-cell clearance and may overcome rituximab resistance. We report one of the first UK cases of obinutuzumab used successfully as salvage therapy for rituximab-refractory MPO-ANCA vasculitis in a kidney transplant recipient, highlighting mechanistic novelty and clinical feasibility. Therapeutic options in this scenario are rarely documented, with no established transplant-specific pathway once rituximab and cyclophosphamide have failed.

Case/Methods

A 45-year-old woman, diagnosed with MPO-ANCA vasculitis in 2009, developed kidney failure and underwent deceased-donor transplantation in June 2016. Despite standard immunosuppression, she experienced repeated biopsy-proven relapses: January 2023, September 2023, and July 2024. These occurred despite rituximab (two 1 g doses; January 2023) and six intravenous cyclophosphamide pulses (November 2023–March 2024). In July 2024, with rituximab-refractory disease confirmed, the multidisciplinary vasculitis and transplant teams initiated obinutuzumab induction (1,000 mg IV on days 1 and 15), with premedication and Pneumocystis prophylaxis. Mycophenolate was withheld during induction and reintroduced two weeks later. Structured surveillance was undertaken, including full blood counts, immunoglobulin levels, and viral PCR monitoring, reflecting MDT-led efforts to balance efficacy with safety.

Results

She has maintained remission for 10 months (August 2024–May 2025) with no further flares. Treatment was well tolerated, with no infusion reactions. Within three months, biopsy (November 2024) showed no active vasculitis or rejection. A first maintenance dose of obinutuzumab (500 mg IV) was administered in February 2025. Graft function improved: creatinine fell from 186 to 144 µmol/L and eGFR rose from 28–32 to 38 mL/min/1.73 m^2. Haemoglobin normalised with iron therapy. Transient leukopenia (WCC 2.9 $\times 10^9$/L) resolved after withholding mycophenolate, which was then permanently discontinued. There were no major infections, hypogammaglobulinaemia, or rejection episodes, demonstrating efficacy without compromising transplant safety.

Discussion

This case demonstrates the feasibility of obinutuzumab as salvage therapy for rituximab-refractory MPO-ANCA vasculitis in the transplant setting. Mechanistically, rituximab relies more on complement-mediated killing, whereas obinutuzumab achieves deeper tissue B-cell depletion and stronger FcγRIIIa engagement, overcoming recognised resistance pathways. Clinically, MDT-guided titration of immunosuppression achieved durable remission, improved allograft function, and manageable cytopenia. While a single case, it provides practice-based evidence that obinutuzumab can be delivered safely alongside transplant immunosuppression and should prompt systematic evaluation in real-world registries.

Implication

Obinutuzumab may represent a rescue strategy in rituximab-refractory ANCA vasculitis after kidney transplantation, where conventional therapies are exhausted.

Next step

Prospective registries and carefully designed studies with transplant-specific safety endpoints are required to confirm feasibility, refine dosing, and define obinutuzumab's place within multidisciplinary vasculitis–transplant practice.

TA3

When the stones keeps hitting ,leave no stones unturned: A case report of APRT deficiency progressing to ESRF

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Introduction

Adenine phosphoribosyl transferase (APRT) deficiency is a rare autosomal recessive disorder of purine metabolism that is characterized by development of radiolucent stones in the urinary tract and resultant progressive CKD secondary to crystal nephropathy. We report a case of 2,8-DHA crystalline nephropathy caused by APRT deficiency in a 19 year old male.

Case Summary

A 19 year old male presented with several episodes of diarrhea and vomiting and had AKI with a creatinine of 208 µmol/l.(baseline-116 µmol/l) Initial diagnosis of pre-renal AKI secondary to viral gastroenteritis was made and he was rehydrated which led to a marginal improvement to 176 µmol/l. He was discharged following resolution of symptoms and planned for outpatient follow-up .

His ultrasound showed normal sized kidneys with increased cortical echogenicity and reduced cortico-medullary differentiation. The right kidney was also mildly hydronephrotic and there was 17 mm hyperechoic foci in the urinary bladder suggesting possibility of stones. His urine was bland.

On follow up appointment , it was noted that his renal function was worse with creatinine now 201µmol/l but immunology screen was unremarkable . About 7 weeks after his initial presentation, he was readmitted to hospital with right flank pain, raised inflammatory markers and his creatinine was 328µmol/l . Repeat ultrasound showed a moderately hydronephrotic right kidney but there was still no clear evidence of an obstructing calculi. CT KUB then revealed a 8mm obstructive calculus in the proximal right ureter with hydroureteronephrosis and he had a right retrograde ureteric stent inserted. Left kidney was unobstructed.

Few months after, while on holiday abroad with his family, he was admitted to hospital with worsening loin pain and deteriorating kidney function .Imaging showed blockage of his right ureteric stent and he had a right nephrostomy inserted. He returned back to the UK and was readmitted due to worsening symptoms . CT then showed a patent right nephrostomy tube and there was no visible calculus but his creatinine had peaked at 600 µmol/l.

The presence of significant renal dysfunction, absence of proteinuria and unobstructed kidneys led to a decision to perform a kidney biopsy. This showed features of obstructive intraluminal crystalline casts in many tubules(Fig 1 and 2), moderate acute tubular injury and mild acute pyelonephritis. After MDT discussion, he was commenced on a trial of steroids and the possibility of crystallite/tubular nephropathy was considered. 24 hr urine oxalate excretion was normal and following discussion with the local tertiary specialist center, he was screened for APRT deficiency and commenced on empirical Allopurinol and Pyridoxine.

He was commenced on hemodialysis due to worsening renal function. His genetic testing confirmed he had homozygous APRT deficiency and APRT in red blood cell was 2nmol/Hr/mgHb(Ref range 14-39).

Conclusion

Due to the rarity of the disease and huge variability in presentation, delay in diagnosis is often the norm. A high index of suspicion is required and this should be considered in patients presenting with urinary stones and renal impairment, early onset urinary stones, frequent recurrence of stones and unexplained renal failure.

TA4

Euglycaemic ketoacidosis after initiation of tirzepatide in a patient on chronic SGLT2 inhibition and intercurrent colitis

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Introduction

The rapid adoption of SGLT2 inhibitors and tirzepatide is reshaping kidney care but exposing clinicians to an under-recognised threat: euglycaemic diabetic ketoacidosis (euDKA). Without blood ketone testing it may be overlooked, delaying life-saving therapy. We describe a rare case of tirzepatide-associated euDKA in a patient on chronic SGLT2 inhibition, complicated by colitis, to highlight this emerging diagnostic and therapeutic challenge.

Case

A 44-year-old woman with type 2 diabetes, multiple sclerosis, and coronary artery disease had been on canagliflozin since January 2021. In July 2025 she received her first dose of tirzepatide 2.5 mg weekly. Within a week she developed bloody diarrhoea, abdominal pain, and dehydration; CT confirmed descending and sigmoid colitis. On admission euDKA was not recognised for nearly 48 hours and was only identified when nephrology reviewed unexplained acidosis. Arterial pH was 7.00–7.33, bicarbonate 8–19 mmol/L, anion gap elevated, serum β-hydroxybutyrate 5.5 mmol/L, and glucose remained in the euglycaemic range. CRP peaked at 212 mg/L, WCC 9.2–15.8×10^9/L, potassium 3.1–3.2 mmol/L, albumin 25–31 g/L; lactate was normal and eGFR >90 mL/min/1.73 m². Stool cultures were negative. Starvation ketosis and sepsis were considered but excluded: lactate was normal, stool studies were negative, and the severity of acidosis with β-hydroxybutyrate 5.5 mmol/L pointed decisively to euDKA.

Results

Tirzepatide was discontinued and canagliflozin withheld. Management included isotonic fluids, variable-rate intravenous insulin with dextrose, potassium replacement, and antibiotics. Acidosis and ketosis resolved rapidly. She was discharged on 23 July 2025 on NovoMix 30 insulin (12 units AM, 10 units PM). Tirzepatide was permanently discontinued. A Naranjo score of 5–6 indicated a probable adverse drug reaction.

Discussion

This case illustrates a “three-hit” model for tirzepatide-associated euDKA. Predisposition: chronic SGLT2 inhibition lowered the glycaemic threshold for ketosis and masked hyperglycaemia. Trigger: first-dose tirzepatide induced gastrointestinal intolerance and reduced intake, generating starvation physiology. Amplifier: colitis added inflammation, dehydration, and counter-regulatory stress. Together these produced profound ketonaemia and acidosis despite euglycaemia. Dechallenge confirmed causality; rechallenge was avoided. The prolonged half-life of tirzepatide (~5 days) means its gastrointestinal and metabolic effects persist through peri-illness fasting and bowel preparation, making this

signal directly relevant to transplant and CKD care pathways. This is among the first UK renal reports of tirzepatide-triggered euDKA, highlighting a diagnostic gap where absence of hyperglycaemia can delay recognition for days.

Implication

Nephrologists should maintain high suspicion for euDKA in any patient on SGLT2i and tirzepatide presenting with acidosis, and should measure blood ketones before attributing symptoms to sepsis or starvation. Sick-day rules and peri-illness drug holds must be embedded into renal and transplant care pathways.

Next step

Renal services should implement peri-procedure protocols including early suspension of SGLT2i/tirzepatide, ketone-first monitoring, and insulin–dextrose bridging. National audit is needed to quantify incidence, refine drug-hold intervals, and establish euDKA prevention as a measurable NHS safety standard.

TA5

Calcified fibrin sheath mimicking a retained dialysis catheter on CT imaging after dialysis Permcath removal

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TUESDAY - Moderated Poster Session, HALL Q, March 10, 2026, 16:00 - 17:00

Case:
A 58-year-old woman with diabetic chronic kidney disease undergoing thrice-weekly haemodialysis via a right internal jugular tunneled permacath presented with a febrile episode during a session of hemodialysis .She had previously exhausted the options of peritoneal dialysis due to peritonitis and AV fistula access secondary to steal syndrome.

The patient reported blood on her permacath dressing and had changed it herself one day before the hemodialysis session , raising suspicion for CVC catheter contamination .Her Blood cultures grew Enterobacter cloacae, while catheter cultures showed E. cloacae and Klebsiella oxytoca. Treatment with intravenous piperacillin–tazobactam and post dialysis gentamicin was initiated.

The catheter was removed shortly afterward, with significant resistance noted during extraction .On removal, it was found to be fully intact and heavily coated with calcifications. A contrast-enhanced CT scan performed soon after revealed a linear hyperdense structure along the course of the catheter.This was initially reported as a retained catheter tip. The scan also showed extensive calcifications in multiple blood vessels, indicating widespread vascular calcification.

After multidisciplinary evaluation, it was determined that the hyperdense linear structure seen on CT was actually a calcified fibrin sheath, not a retained catheter fragment.
A new femoral dialysis catheter was inserted soon after removal of the contaminated catheter. Although repeat peripheral cultures initially grew Enterobacter, subsequent cultures were negative .Haemodialysis was continued without further complications and the patient's recovery thereafter was uneventful .

Conclusion:
Significant calcification encasing a catheter can closely resemble a retained catheter tip on imaging, particularly when widespread vascular calcification is present. Recognizing this "phantom catheter" phenomenon is vital to avoid unnecessary invasive procedures, such as surgical removal or catheter replacement, which can increase patient morbidity, healthcare costs, and anxiety. A multidisciplinary approach and careful image interpretation are essential for appropriate management, especially when bacteremia occurs around the time of catheter removal.

TA7

Vascular Ehlers-Danlos Syndrome presenting with bilateral renal infarcts

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TUESDAY - Moderated Poster Session, HALL Q, March 10, 2026, 16:00 - 17:00

Introduction:
Malfait et al updated the classification of Ehlers-Danlos Syndrome (EDS) in 2017 to include 13 distinct subtypes. This incorporated novel research into genetic testing building on the original six subtypes published in 1998. Described within these is Vascular EDS, caused by an autosomal dominant variant of COL3A1 decreasing synthesis of type III collagen. It's a rare and diagnostically complex subtype of EDS, estimated to affect 1 in 5,000 to 10,000. Derangement and rarefication of elastic fibres in vessel walls leads to complications such as arterial rupture and occlusions at a young age.

Case:
A 35-year-old man, with no significant past medical history, presented with 7-days of vomiting with associated central and right flank pain. He had an acute kidney injury, creatinine 111 µmol/L, GFR 74, with a CRP of 110 mg/L and microscopic haematuria with a protein: creatinine ratio of 76 mg/mmol. Initial CT imaging showed a splenic infarct and large infarct in the mid-pole of the right kidney. CT angiography and direct renal angiography demonstrated a large right and smaller left renal infarct, an enlarging splenic aneurysm requiring coil embolisation and right extracranial vertebral artery, hepatic and coeliac dissection without microaneurysms (Figures 1&2).
On discussion between nephrology, vascular and rheumatology teams, given rapidly evolving vascular changes, this was felt to potentially represent a large vessel vasculitis and he received 3 doses of intravenous methylprednisolone followed by oral prednisolone and pulsed intravenous cyclophosphamide. Renal biopsy was contraindicated due to concerns regarding polyarteritis nodosa and associated bleeding risk. He had a negative ANCA, ANA positive (1:80), negative dsDNA and lupus anticoagulant, normal complement and rheumatoid factor.
On review of his family history, his mother was diagnosed with joint hypermobility aged 38, and presented age 51 with an acute type B dissection of the thoracic aorta. She underwent successful stent grafting but died suddenly post discharge therefore the R125 thoracic aortic aneurysm or dissection panel was requested. This detected a likely pathogenic missense COL3A1 variant c.1294G>A p.(Gly432Ser) two months after his initial presentation. By this time, a PET-CT was completed showing no evidence of vasculitis. His prednisolone was weaned and stopped and cyclophosphamide stopped. Although he requires ongoing treatment for hypertension, his latest GFR is 80 and CRP 4 mg/L.

Discussion:
The acute kidney injury, microscopic haematuria, proteinuria and raised CRP at presentation was potentially driven by renal necrosis however time pressure from the expansile splenic artery required urgent management despite diagnostic uncertainty. Family history

represents a minor criterion in the vascular EDS classification, but genetic testing, a major criterion, was received two months later.

There is no specific treatment for vascular EDS beyond tight blood pressure control including the use of beta blockers. The patient has received education about the condition including advice to avoid trauma, contact sports and large increases in blood pressure and he will have annual screening of his vascular tree by MR angiography.

In conclusion, vascular EDS is a rare cause of renal infarction where the use of genetic testing is important to establish a diagnosis.

TA8

Remission of PR3 associated vasculitis in a 16 year old patient with avacopan

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TUESDAY - Moderated Poster Session, HALL Q, March 10, 2026, 16:00 - 17:00

Avacopan is quickly becoming the standard of care for ANCA associated vasculitis, providing a steroid sparing alternative; however, it is currently only licensed for patients over 18. We present a case of PR3 associated vasculitis in an adolescent who was treated with avacopan, including the patient as an author, to describe the improvement in his quality of life.

A 16 year old male of South Asian ethnicity presented with two weeks of a dry cough that later became productive of brown sputum, associated with intermittent fevers, epistaxis, haemoptysis, and reduced appetite with weight loss. No rashes or lymphadenopathy were noted but further questioning revealed mild arthralgia in wrists and ankles.

He had travelled to Turkey over the summer holidays but had no contact with TB. He was a non-smoker. He was initially treated for a lower respiratory tract infection with ceftriaxone and clarithromycin.

An initial chest radiograph showed several ill-defined bilateral pulmonary nodules; these were further characterised with a CT that demonstrated multiple areas of large rounded dense consolidation with surrounding ground glass changes, alongside numerous bilateral pulmonary nodules with similar ground-glass halos scattered throughout both lungs.

He had markedly elevated inflammatory markers, with a CRP 145, ESR 85. Renal function, liver function, and bone profile were normal. Immunology was noted for negative ANA and dsDNA, but he had a raised IgG/IgA and positive ANCA antibodies in a c-ANCA pattern. PR3 titres were elevated at 91 and MPO negative. Blood cultures, procalcitonin, sputum cultures (including for acid fast bacilli), fungal markers, and Quantiferon tests were all unremarkable. Virology was also negative. Urinalysis showed trace of blood, but all else was negative. A blood film showed microcytic hypochromic anaemia with pencil and target cells in keeping with iron deficiency anaemia. Bronchoscopy was attempted but abandoned early due to poor tolerance by the patient. However, images taken showed abnormal inflamed and friable airways and gross pulmonary haemorrhage.

He was treated for ANCA associated vasculitis with three pulses of 500mg methylprednisolone daily, followed by a weaning course of prednisolone, and 2 doses of rituximab alongside co-trimoxazole, omeprazole, and vitamin D prophylaxis: cyclophosphamide was avoided due to concerns regarding fertility. Due to side effects of fatigue and myalgia, which impeded his football playing and revision for A levels, avacopan was funded by the trust on compassionate grounds. This was well tolerated with no lymphopaenia, and his disease has remained well controlled over with undetectable ANCA levels. Repeat imaging six months after the first CT showed that the previously demonstrated multiple bilateral pulmonary nodules had completely resolved with residual lung cysts (Figure 1).

This case provides an example of the potential role of avacopan in treatment of ANCA associated vasculitis in adolescents, where avoiding prolonged glucocorticoid use and preservation of fertility is key to reducing treatment related morbidity and supporting long term psychosocial and wellbeing.

TA9

A case of peritonitis as a risk factor for transvaginal dialysate leak in the presence of a normal genitourinary anatomy

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Title
Peritonitis as a risk factor for Transvaginal dialysate leak in the presence of a normal genitourinary anatomy.

Introduction
This case involves a 78yr old female with a background of end stage renal failure on continuous ambulatory peritoneal dialysis. She presented with a one-week history of intermittent per vaginal (PV) leakage of clear fluid.
Speculum examination revealed pooling of clear fluid in the vaginal canal, though no identifiable source of leakage was found.
CT peritoneography demonstrated a continuous tract of contrast-enhanced dialysate through patent fallopian tubes into the endometrial and vaginal cavities with no evidence of vaginal wall perforation or abnormal fistula formation.
The findings were consistent with physiological patency of fallopian tubes serving as a conduit for dialysate leakage.
The patient was transitioned to haemodialysis, and her PD catheter was removed. After this intervention, the per vaginal leakage was fully resolved, and no additional episodes were observed.

Discussion
Peritoneal dialysis is associated with several recognised Infectious and non-infectious complications.
Trans vaginal dialysate leakage remains an uncommon non-infectious complication of Peritoneal dialysis. While peritoneo-vaginal fistulas are most implicated, this case report demonstrates that dialysate leakage may also occur via the fallopian tubes in the context of a normal genitourinary anatomy.
Normally, the luminal pressure within the fallopian tubes prevents retrograde passage of fluid; however, tissue friability or altered peritoneal dynamics may compromise this barrier, allowing dialysate to track into the uterine and vaginal cavities. This patient had a history of recent peritonitis and had completed a course of antibiotic a week before onset of symptoms.
Initial assessment of a patient presenting with vaginal discharge requires a thorough clinical history and examination, including speculum evaluation by a gynaecologist. If the diagnosis remains unclear, further investigations are warranted.
Treatment options include surgical repair of abnormal fistulas, temporary haemodialysis, and low volume automated peritoneal dialysis (APD). In this patient, bilateral tubal ligation was considered, however, given the absence of a pathological fistula and the patient's age a decision was made to transition to haemodialysis, and her PD catheter was removed.

Conclusion

This case underscores a rare complication of peritoneal dialysis—transvaginal dialysate leakage via anatomically intact fallopian tubes. Although uncommon, such presentations should be considered in female PD patients with unexplained vaginal fluid loss, particularly in the context of elevated intra-abdominal pressure or prior peritonitis. In select cases, transition to haemodialysis may be necessary to resolve symptoms and prevent recurrence.

TA10

Sustained parvovirus infection in pancreas and kidney transplant receipt causing recurrent red cell aplasia

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Background

Parvovirus B19, a member of the Erythroparvovirus genus, can cause symptomatic or asymptomatic infection depending on age and immune status. In patients lacking effective humoral immunity, persistent viraemia may lead to red cell aplasia, cytopenias, hepatitis, myocarditis, neurological disease, or graft dysfunction. Most infections are self-limiting and improve with reduced immunosuppression.

Clinical case

A 57-year-old gentleman with simultaneous pancreas and kidney transplantation in July 2018 (1:2:1 mismatch) for end-stage renal failure secondary to diabetic nephropathy, presented with new-onset anaemia (Hb 60 g/L) of unclear cause with supressed reticulocytes. Azathioprine 75 mg once daily was withheld due to the anaemia, while he remained on tacrolimus (Adoport 6 mg twice daily). Initial workup revealed a low reticulocyte count suggestive of bone marrow suppression; however, the improvement in haemoglobin after discontinuing azathioprine made primary bone marrow disease less likely, and a biopsy was not performed after seeking haematologist opinion. Subsequently, Parvovirus B19 PCR returned positive (log 4.6; 4687 copies). The patient was treated with intravenous immunoglobulins (IVIG), resulting in viral load reduction and normalization of haemoglobin. Despite this, he experienced recurrent anaemia (Hb 58 g/L with viral log 10.4, and Hb 70 g/L with viral log 10.0, Hb 99 with viral log 10.5) at five, nine and thirteen months after the first episode. He required four courses of IVIG and multiple blood transfusions, with azathioprine permanently discontinued and Tacrolimus supplemented with Sirolimus (with the intention of taking advantage of its direct anti-viral effect). The patient has stable transplant functions and currently has stable haemoglobin but persistent low level viraemia.

Conclusion

Parvovirus B19, though rare in pancreas and renal transplant recipients, is an important cause of refractory anaemia. Chronic infection is rare and difficult to manage due to the balance between reducing immunosuppression and infection risk. Clinicians should suspect B19 in transplant patients with unexplained or persistent anaemia.

Figure 1. Haemoglobin trend in a simultaneous pancreas–kidney transplant recipient with Parvovirus B19 infection, demonstrating recurrent anaemia and subsequent improvement following intravenous immunoglobulin (IVIG) therapy.