WA1

Unraveling the clinical heterogeneity of NELL1-positive membranous nephropathy: A case report of an idiopathic presentation

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WEDNESDAY - Moderated Poster Session, HALL Q, March 11, 2026, 13:45 - 14:45

Background:
Neural epidermal growth factor-like 1 (NELL1) is a podocyte antigen, first identified in 2019 in a subset of membranous nephropathy (MN) with distinct clinicopathologic features. NELL1-positive MN accounts for approximately 16–35% of PLA_2R-negative primary MN and typically demonstrates granular subepithelial deposits with IgG1 or IgG4 dominance.Unlike PLA_2R-MN, anti-NELL1 serology is not yet widely available, so histopathology remains the diagnostic standard.Initially considered largely primary, subsequent studies revealed strong associations with malignancy, medications (thiol drugs, lipoic acid, NSAIDs), heavy metal exposure, and traditional remedies, with secondary causes identified in up to 89% of patients in some cohorts. However, the relationship with malignancy remains controversial: some series reported no tumors, whereas others found malignancy in up to one-third of cases. We wanted to present a case illustrating the heterogeneity of presentation of NELL1-MN.

Case Presentation:
We report a 78-year-old male with atrial fibrillation, mitral regurgitation, heart failure with preserved ejection fraction(HFpEF) and bronchiectasis, who had recurrent presentations with dyspnea and peripheral edema around November 2024. Symptoms worsened despite optimum management of his HFpEF. Development of generalised edema, ascites, pleural effusion prompted work up for nephrotic syndrome. Significant findings include urine protein creatinine ratio(UPCR)-873 mg/mmol, serum albumin-14g/L, cholesterol-8.2 mmol/L., negative for serum anti-PLA2R antibody. He had eGFR of 76ml/min/1.72m2 and creatine of 84 umol//L at this stage. A renal biopsy was organised as part of the work up which demonstrated changes consistent with PLA_2R-negative MN. Upon requesting for specific immunohistochemical tests, a strong NELL1 positivity and IgG4/IgG1-dominant staining was noted. Extensive evaluation—including CT imaging, hepatitis and HIV serology, myeloma screen, liver/renal ultrasound—showed no evidence of malignancy, autoimmune disease, or relevant exposures. Supportive treatment with diuretics, human albumin solution and fluid management was commenced. He struggled significantly with postural hypotension, requiring treatment with midodrine. Due to persistence of heavy proteinuria, decline in renal function (eGFR-44ml/min/1.73m2, creatinine-130umol/L in December 2025) and frailty, immune suppression with Rituximab was offered. He received 2 doses in January 2025. Partial remission with stabilization of renal function (creatinine 95ml/min/2.73m2), remarkable drop in urine PCR (505mg/mol from peak of 1212mg/mol) and improvement in serum albumin (33g/L) was noted after treatment. He remains on regular follow up under our care.

Discussion:

While secondary associations, particularly with malignancy, are well described, a subset of patients—including ours—have no identifiable triggers. Emerging evidence suggests a spectrum of clinical behavior. A few recent reviews emphasize that some NELL1-MN patients, especially those without secondary associations, may follow an indolent course under supportive care alone. Conversely, others require immunosuppression for persistent proteinuria. Taken together, current data indicate that NELL1-MN cannot be regarded as uniformly malignancy-associated or uniformly aggressive. Careful exclusion of secondary causes, individualized treatment decisions, and close monitoring remain key.

Conclusion:

We present a case of NELL1-MN without secondary associations, contributing to the expanding clinical spectrum of this entity. Recognition of both high-risk (eg: malignancy/ drug-associated) and potentially lower-risk idiopathic phenotypes should support clinical practice. Larger prospective studies are needed to clarify pathogenesis, define risk stratification and optimise treatment strategies of this evolving condition.

WA2

Pyroglutamic Acidosis

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WEDNESDAY - Moderated Poster Session, HALL Q, March 11, 2026, 13:45 - 14:45

Background

High anion gap metabolic acidosis (HAGMA)- is a common acid-base derangement resulting from a variety of metabolic changes -normally remembered by the acronym MUDPILES
A rare cause of HAGMA is pyroglutamic acidosis.
Pyroglutamic acidosis also known as 5-oxoprolinemia -is produced from γ-glutamyl cysteine by the enzyme γ-glutamyl acyltransferase catabolized by 5-oxoprolinase.When glutathione levels are low, the activity of γ-glutamyl acyltransferase is increased, resulting in pyroglutamic acid accumulation in glutathione-depleted states
Can be inherited - due inborn errors of metabolism
Acquired causes include paracetamol use ,flucloxacillin (inhibits 5-oxoprolinase) ,hepatic ,renal impairment and malnutrition
Flucloxacillin and paracetamol can cause pyroglutamic acidosis by disrupting the glutathione cycle.
This cumulative effect results in High Anion gap metabolic acidosis due to build up of pyroglutamic acid.

Case presentation

We present the case of a 62 years old lady -who developed high anion gap metabolic acidosis - was found to have pyroglutamic acidosis
BG of - Multiple previous bilateral hip surgeries -, previous MSSA bacteremia of right hip.
Admitted to hospital with sepsis-secondary to infected right hip prosthesis -was taken to theatres by the orthopedics team.
Admitted to ITU post op following -Partial Excision Arthroplasty of Hip with MSSA Bacteremia and AKI. Bloods and orthopedics fluid cultures were positive for MSSA – (sensitive to flucloxacillin) so commenced on flucloxacillin - also had therapeutic doses of paracetamol prescribed for pain control.
Admitted to ITU post-operatively for BP support and stage -III AKI which both resolved . However, continued to have worsening inflammatory markers - CT- TAP done to look for focus of infection showed edematous pancreas.
Escalated from 2G flucloxacillin to Piperacillin-tazobactam and clindamycin on day -15 of flucloxacillin. Her blood gas showed metabolic acidosis This lady was also severely deconditioned, mal-nourished and frail because of prolonged hospital stay and intercurrent illness
Venous blood gases showed progressively worsening metabolic acidosis. The patient displayed a high-anion gap metabolic acidosis (HAGMA): serum anion gap (AG), corrected for hypoalbuminemia (low -albumin 17) equaled AG = 31.8 -calculated using -AG + 2.5 (4-albumine in g/dL).

After appropriate antibiotic escalation her inflammatory markers were improving, she was no longer septic, her nutrition status had also improved after commencement of NG feeding. Alternative causes of HAGMA were ruled out. However, the metabolic acidosis continued to worsen - The possibility of pyroglutamic acidosis due to the concomitant use of paracetamol and flucloxacillin was suspected. Paracetamol was withheld; she was commenced on bicarbonate replacement and urinary organic acids including pyroglutamic acids were sent off

She was pre-emptively given N-acetyl cysteine for high suspicion of pyroglutamic acidosis, gradually she got better - the acidosis resolved with normalization of her bicarbonate levels. The diagnosis was further confirmed by urine organic acids measurement, showing a markedly elevated level of pyroglutamic acid

Conclusion

Pyroglutamic acidosis is usually a diagnosis of exclusion supported by the appropriate clinical scenario combined with plasma and/or urine pyroglutamic acid levels.
This lady developed pyroglutamic acidosis due to co-prescription of flucloxacillin and paracetamol with underlying malnutrition , sepsis as risk factor

WA3

Asymptomatic Large Renal Arteriovenous Malformation: From Incidental Finding to Intervention

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WEDNESDAY - Moderated Poster Session, HALL Q, March 11, 2026, 13:45 - 14:45

Background: Renal arteriovenous malformations (AVM) are rare abnormalities of the vascular wall of renal vessels and literature suggest that incidence is <1%. Frequent symptoms constitute haematuria, sometimes life-threatening, flank pain or even symptoms of high output heart failure. The gold standard diagnosis is made with Digital Subtraction Angiography and embolization is the treatment of choice when feasible. If embolization is unsuccessful, then surgical intervention with partial or total nephrectomy in severe cases is indicated.

Case presentation: A 63 year old man with coincidental finding of an asymptomatic left renal AVM as a result of investigations performed to investigate nocturia, itself the result of benign prostate hyperplasia (BPH). From his past medical history, he was being treated with losartan for hypertension. The initial ultrasound revealed pelvic ureteric dilatation, and a subsequent CT urogram showed a parapelvic cyst, and raised the suspicion of a left renal AVM. An MRA demonstrated early venous filling and dilatation of the left renal vein with the presence of an AVM (measurements 35x33mm). A yearly follow-up MRA showed increase in size and measurments 36x35mm and on discussion in multidisciplinary meeting, he was followed up with annual interval scanning with CT Angiography. The CT angiography revealed the AVM feeding artery measuring 11 mm distally, which directly feeds into the draining veins via a couple of defined small branches. The left renal vein was markedly aneurysmal at 38x36 mm. His initial kidney function, with a creatinine of 95 mmol/l (eGFR 75 ml/min/1.73 m^2), remained stable over this period, and he remained asymptomatic. A cardiac MRI was performed to assess cardiac compromise caused by the enlarging AVM; it was normal. Due to the growing size of the aneurysmal renal vein, we decided to proceed with embolization. This was because we were concerned it might grow beyond a size that would complicate the intervention, as well as the potential risks of rupture and heart failure. The first procedure involved planning an angiogram for the definitive intervention. The second procedure involved embolization with multiple coils. The AVM was partially excluded, with some residual sluggish flow. A week later, a super selective angiogram of the left renal artery confirmed complete exclusion of the fistula with good perfusion of the kidney parenchyma. No further intervention was required.

Conclusion: Although renal AVMs are infrequently encountered and imaging is key in obtaining the diagnosis, symptoms may be absent or nonspecific. Rupture and life-threatening bleeding has been well described in the literature, when surgical intervention was required either because of the urgency or because of embolization had been unsuccessful. Our patient has remained completely asymptomatic throughout follow-up and

underwent successful embolization, despite a markedly dilated and increasing in size renal vein, his kidney function was unaffected, blood pressure well controlled and no signs of cardiopulmonary compromise, unlike similar AVM cases. Management can prove to be challenging, especially when balancing the risks of delayed intervention against procedural complications.

WA4

A case report of distal renal tubular acidosis presenting with hypokalaemia

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WEDNESDAY - Moderated Poster Session, HALL Q, March 11, 2026, 13:45 - 14:45

Introduction:
Distal renal tubular acidosis (dRTA) is a relatively uncommon disorder caused by failure of hydrogen ion secretion in the distal nephron. This results in a non-anion gap, hyperchloremic metabolic acidosis and can lead to life-threatening electrolyte disturbances. Clinical recognition is often delayed as the condition may present with non-specific symptoms.

Case Presentation:
A 43-year-old woman presented to the Emergency Department with symptomatic hypokalaemia (serum potassium 2.3 mmol/L). She reported generalised muscle aches, weakness, paraesthesia in her limbs and trunk, intermittent dizziness, light-headedness and shortness of breath. She had lost 5 kg over the preceding 2–3 months and also described subjective dry eyes and dry mouth. There was no significant past medical history other than gastritis and irritable bowel symptoms under gastroenterology review. She is an active smoker and had recently been reviewed by rheumatology for possible autoimmune disease. On examination, her vital signs were stable. Cardiovascular and respiratory assessments were normal. Neurological examination revealed mild symmetrical weakness in the lower limbs, both proximally and distally.

Investigation:
Blood tests confirmed hypokalaemia, hyperchloremia, low serum bicarbonate and metabolic acidosis (low pH). Renal, thyroid and liver function were normal. Renin-aldosterone levels ruled out Conn's syndrome (hyperaldosteronism) as a cause of hypokalaemia. Urine studies showed high urinary potassium (>15 mmol/L) and a persistently high urinary pH (>5.5). These findings supported the diagnosis of dRTA. Unfortunately, urinary chloride was not measured so the urinary anion gap could not be calculated.
Further immunological workup showed a positive ENA screen with raised Ro (SSA) antibody (162 U/ml), raising the possibility of an autoimmune aetiology such as Sjögren's syndrome.

Management and Outcome:
The patient was treated with intravenous potassium chloride (40 mmol × 3 doses) followed by oral potassium supplementation. Sodium bicarbonate 500 mg three times daily was commenced after renal consultation. Her potassium levels improved and remained stable on follow-up. She was discharged with ongoing oral potassium replacement and remains under renal and rheumatology follow-up for further evaluation.

Discussion:

This case demonstrates the importance of considering renal tubular disorders when evaluating patients with hypokalaemia and metabolic acidosis. dRTA can easily be overlooked unless a physiology-based approach is used. The association with autoimmune diseases, particularly Sjögren's syndrome, is well recognised and should be actively investigated. Early recognition is important, as untreated dRTA can result in recurrent hypokalaemia, nephrocalcinosis, bone disease and progressive renal impairment. In this case, the presence of positive Ro (SSA) antibodies highlights the importance of screening for autoimmune causes.

Conclusion:
Distal renal tubular acidosis is rare but potentially life-threatening if undiagnosed. This case underlines the need for systematic evaluation of metabolic acidosis with careful interpretation of urine studies. Early identification allows prompt treatment, prevents complications and can uncover an underlying systemic disease requiring long-term management.

WA5

Why bet when you can pet!!

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WEDNESDAY - Moderated Poster Session, HALL Q, March 11, 2026, 13:45 - 14:45

Introduction

Granulomatosis with polyangiitis(GPA) is a form of systemic vasculitis is characterized by clinical manifestations which are sometimes difficult to distinguish from infective pathology. It's immunosuppressive treatment is frequently complicated by infection. Differentiating active GPA from infection is critical to management and patient outcome

Case Summary

A 37 year old man was diagnosed with GPA, having presented with nasopharyngeal disease, multiple cranial nerve palsies (7,11, 12), cerebral vein thrombosis, cavitating chest disease and pauci-immune glomerulonephritis.
He regained independent kidney function 2 months following Rituximab induction. His early clinical course was complicated by lung hemorrhage and pseudoaneurysm rupture (left kidney) requiring embolization. Attempts to establish the patient on maintenance immunosuppression were hampered by pancytopenia (azathioprine), recurrent chest infection (aspiration related to cranial nerve palsies), steroid induced psychosis and nutritional compromise (BMI 18 from 31) requiring PEG feeding.

Re-induction with rituximab was required after 12 months (nasopharyngeal and chest disease progression), followed by a prompt relapse requiring cyclophosphamide. Despite an excellent response, 2 months later he developed multi-organ failure secondary to sepsis (source unknown) resulting in a 2-month hiatus in immunosuppression except for steroid. A recrudescent PR3 and inflammatory response led to a comprehensive reassessment of disease activity and infection screen, further Rituximab and Tertiary service referral.

A persistent inflammatory response and neutrophilia were noted despite B-cell depletion. Avacopan was added pending ENT assessment and CT-PET. The latter revealed a mass or abscess replacing most of the left kidney without other evidence of vasculitis activity. Further imaging, infection screen and kidney biopsy did not establish a specific diagnosis. A prolonged course of ertapenem was unhelpful and the patient’s clinical condition and GFR deteriorated.

A left nephrectomy was undertaken (non-functioning on DMSA). Histology did not reveal malignancy or active vasculitis, and microbiological studies were negative despite the presence of widespread abscess formation.

4 weeks later, the patient was recovering well with normalized CRP and WCC, though unfortunately, dialysis dependent.

Conclusion.

This case highlights the challenges differentiating active vasculitis from infective complications of immunosuppression, as well as the value of a second opinion and CT-PET when the clinical course is atypical

WA6

Hormone Replacement Therapy and Venous Thromboembolism Risk in the Peri-Operative Setting: A Living Kidney Donor Case Report

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WEDNESDAY - Moderated Poster Session, HALL Q, March 11, 2026, 13:45 - 14:45

Introduction

Menopause is the permanent cessation of menstruation which affects all women. Up to 80–90% report some symptoms, with 25% describing them as severe and debilitating. Symptoms may include hot flushes, night sweats, disturbed sleep, anxiety, joint pain, and vaginal or urinary issues. Hormone replacement therapy (HRT) is first-line treatment. Peri-operative HRT is often withheld due to concerns about venous thromboembolism (VTE). Oral combined HRT increases VTE risk and is typically stopped 4–6 weeks before major surgery, whereas transdermal and vaginal preparations avoid first-pass metabolism and therefore have less effect on coagulation factors. The VTE risk associated with transdermal and vaginal HRT at standard therapeutic doses is no greater than baseline population risk.

This case report highlights the management of HRT in a kidney donor with menopausal symptoms. It emphasises the importance of individualising care, improving understanding of HRT use, avoiding unnecessary discontinuation, and optimising symptom control during the peri-operative period.

Case Presentation

A 55-year-old Caucasian female presented as a planned directed living kidney donor for her son. She was reviewed at the pharmacist pre-assessment transplant clinic in March 2025. Her past medical history included four pregnancies and a history of smoking; she had quit 25 years earlier. She was using a hormone replacement pellet implant in her thigh, sourced from Barbados, replaced every three months and last changed in January 2025. The pellet contained oestradiol 25 mcg (Dermestril Septem) and progesterone 100 mg (Susten), but it was unlicensed and unavailable in the UK. She remained symptomatic and expressed concern about managing her HRT in preparation for surgery.

Limited UK data exist for pellet implants and risk of VTE. One centre reported using implants, noting that residual estradiol release could persist for up to 18–24 months with six-monthly implants. Thrombosis and gynaecological specialists at our hospital were also consulted.

Results

The patient received standard VTE prophylaxis (seven days of post-operative enoxaparin for kidney donation), without extended prophylaxis. Post-operatively, her implant was removed and switched to oestradiol gel and oral progesterone at night, with additional testosterone 2% gel to aid symptom control. She tolerated this regimen until returning to Barbados.

Discussion and Conclusion

This case demonstrates that peri-operative HRT can be safely managed through individualised risk–benefit assessment with careful consideration of VTE prophylaxis. A multidisciplinary approach may reduce unnecessary HRT discontinuation in kidney donors and recipients in the peri-operative period. Clearer guidance is needed to support patients and clinicians in managing HRT in the renal transplant population.

WA7

From Weight Loss to Kidney Loss: Off-Label Semaglutide-Induced Acute Kidney Injury

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[1]Manchester Royal Infirmary

WEDNESDAY - Moderated Poster Session, HALL Q, March 11, 2026, 13:45 - 14:45

Introduction:

Semaglutide is GLP-1 receptor agonist (GLP-1RA) mainly used in Type 2 diabetes to improve glycaemic control and promote weight loss. These advantages have resulted in a rise in off-label purchase and use. There are important considerations regarding its safety and effectiveness when initiated, particularly outside approved prescriptions. These concerns especially regard the renal health implications.

Case Summary

A male patient in his late 20s presented with two days of severe lower abdominal pain, which progressed to bilateral flank pain radiating to the groin. He reported haematemesis, oliguria and constipation. He had no prior history of renal problems. The patient had been taking one month of off-label Semaglutide for weight loss. Physical examination revealed only lower abdominal tenderness. Blood pressure on first inspection was 133/99, which then continued increased.

Admission bloods showed AKI3, CRP: 38 and Creatinine: 270. ECG: NAD. The patient was given IV fluids, antiemetics and analgesics. CT KUB: no radiopaque urinary tract calculus and no hydronephrosis. The patient did not respond to fluids; 16 hours later, bloods showed CRP: 52 and Creatinine: 338. CT abdomen: NAD. IgG, IgA and C3: Negative. IgM, C1q: Trace. Kappa and Lambda showed the same staining intensity.

Antihypertensives were started. Three doses of IV methylprednisolone 250 mg were given, which lead to improved kidney function. The patient was then transitioned to Prednisolone 60 mg OD. Kidney biopsy was performed once blood pressure stabilised.

16 day CRP trend: 38, 52, 101, 101, 38, 19, 9.
16 day Creatinine trend: 270, 338, 450, 467, 300, 218, 157, 119

Biopsy Results (images available) showed widespread interstitial inflammation with lymphocytes, histiocytes and several eosinophils. Interstitial oedema with diffuse acute tubular damage and cytoplasmic vacuolation and regenerative epithelial changes were seen. Uromodulin casts, with associated rupture of the tubular basement membrane were reported.

With clinical and biochemical improvement, the patient was discharged on a prednisolone tapering regime. In Day 23 follow up, Doxazosin was stopped. Bloods showed CRP <1, Creatinine 84 and no AKI. Blood pressure was 112/75.

Discussion

This case highlights the serious risks of GLP-1RAs. AKI was primarily driven by interstitial nephritis, evidenced through the biopsy and improvement only after steroids and not fluids. Whether solely due to GLP-1RA, contamination, or substandard product, remains uncertain.

While GLP-1RAs are increasingly recognised to slow CKD progression in diabetics, unlike SGLT2 inhibitors and ACE inhibitors, there are no reports linking GLP-1RAs to reversible eGFR reduction. Standard CKD treatments involve monitoring kidney function at initiation. This practice is not routinely applied to GLP-1RA use, especially off-label. Nevertheless, AKI post-GLP-1RA initiation highlights the need for close monitoring, further research, and caution when used off-label without clinician supervision.

Conclusion

Early detection of renal impairment and monitoring of GLP-1RAs is essential to prevent irreversible kidney damage. Further research and reported cases are needed to understand individual risk factors for AKI in GLP-1RA use. The rise in off-label use is concerning and warrants more research and monitoring protocols.

WA8

Anti-GBM disease secondary to ipilimumab/nivolumab immunotherapy

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WEDNESDAY - Moderated Poster Session, HALL Q, March 11, 2026, 13:45 - 14:45

We describe a case of a 73 year old male treated for dialysis dependent AKI, who presented with rapidly progressive glomerulonephritis secondary to anti-GBM disease, associated with ipilimumab and nivolumab immunotherapy.

He had a past medical history of clear cell renal cell carcinoma with sarcomatoid differentiation of his right kidney in 2022, which had metastasised to liver, bone, and spleen. He had received four cycles of ipilimumab/nivolumab combination immunotherapy and had continued on nivolumab since, with denosumab being added for bone protection. His last dose of nivolumab had been one month prior to presentation.

He had recently returned from a holiday in Malaysia, after which he had experienced a 3 week history of diarrhoea, generalised unwellness, reduced appetite, and fatigue. He was seen in oncology clinic on his return and recalled after blood tests showed a severe AKI with resultant hyperkalaemia and metabolic acidosis (Ur 55.7, Cr 1670, K+ 7.4, HCO3 12); his renal function had been normal two months prior.

Frank haematuria was noted upon catheterisation and a uPCR was 983mg/mmol. A CT renal tract revealed enlarging right pelvic side wall lymph nodes but no obstruction was seen. He was initially managed in ITU as a pre-renal AKI secondary to diarrhoea in a returning traveller, although steroids for immunotherapy induced colitis were considered. However, his anti-GBM levels returned as 392 and he was treated with 3x pulses of daily 500mg IV methylprednisolone, PO cyclophosphamide 1.5mg/kg, and four cycles of plasma exchange.

Renal biopsy showed severe necrotising glomerulonephritis with linear IgG and C3 staining of the glomerular basement membrane with diffuse acute tubulo-interstitial nephritis, all in keeping with anti-GBM disease. These results, together with his developing anuria, informed the decision to stop PLEX and immunosuppression.

Discussion

This is the first described case of anti-GBM disease associated with ipililumab/nivolumab immunotherapy for renal cell carcinoma. Immunotherapeutic agents, especially checkpoint inhibitors, are well known to cause tubulo-interstitial nephritis , however, anti-GBM disease secondary to immunotherapy is unusual, and such a presentation three years into treatment even more so. In addition, renal cell carcinoma itself has also been described to trigger production of anti-GBM autoantibodies. Proposed mechanisms include development of autoreactive CD4 T cells specific for α3NC1, and loss of self-tolerance mechanisms for the former, and invasive tumour growth causing exposure of the glomerular basement membrane for the latter. Frequent urinalysis on immunotherapy is required to allow for early biopsy to exclude rarer glomerulopathies.

WA9

Pyroglutamic Acidosis

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WEDNESDAY - Moderated Poster Session, HALL Q, March 11, 2026, 13:45 - 14:45

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Can be inherited - due inborn errors of metabolism
Acquired causes include paracetamol use ,flucloxacillin (inhibits 5-oxoprolinase) ,hepatic ,renal impairment and malnutrition
Flucloxacillin and paracetamol can cause pyroglutamic acidosis by disrupting the glutathione cycle.
This cumulative effect results in High Anion gap metabolic acidosis due to build up of pyroglutamic acid.
Case presentation

We present the case of a 62 years old lady , admitted to ITU - who developed high anion gap metabolic acidosis - found to have pyroglutamic acidosis
Multiple previous bilateral hip surgeries -, previous MSSA bacteremia of right hip. Admitted to hospital with sepsis-secondary to infected right hip prosthesis -was taken to theatres by the orthopedics team.
Admitted ITU post op following -Partial Excision Arthroplasty of Right Hip with MSSA Bacteremia and AKI.. Bloods and orthopedics fluid cultures were positive for MSSA – (sensitive to flucloxacillin) so commenced on flucloxacillin - also had therapeutic doses of paracetamol prescribed for pain control.
- Admitted to ITU post-operatively for BP support and stage -III AKI which both resolved . However, continued to have worsening inflammatory markers - CT- TAP done to look for focus of infection showed edematous pancreas.
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After appropriate antibiotic escalation her inflammatory markers were improving, she was no longer septic, her nutrition status had also improved after commencement of NG feeding. Alternative causes of HAGMA were ruled out. However, the metabolic acidosis continued to worsen - The possibility of pyroglutamic acidosis due to the concomitant use of paracetamol and flucloxacillin was suspected. Paracetamol was withheld; she was commenced on bicarbonate replacement and urinary organic acids including pyroglutamic acids were sent off

She was pre-emptively given N-acetyl cysteine for high suspicion of pyroglutamic acidosis, gradually she got better - the acidosis resolved with normalization of her bicarbonate levels. The diagnosis was further confirmed by urine organic acids measurement, showing a markedly elevated level of pyroglutamic acid

Conclusion

Pyroglutamic acidosis is usually a diagnosis of exclusion supported by the appropriate clinical scenario combined with plasma and/or urine pyroglutamic acid levels.
This lady developed pyroglutamic acidosis due to co-prescription of flucloxacillin and paracetamol with underlying malnutrition , sepsis as risk factor

WA10

Minimal change disease following termination of pregnancy: A rare case report

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WEDNESDAY - Moderated Poster Session, HALL Q, March 11, 2026, 13:45 - 14:45

Introduction:
Minimal Change Disease (MCD) is a leading cause of nephrotic syndrome, but its onset following pregnancy termination is extremely rare. Limited literature exists on this association, with only two prior case reports documented. We present a rare and informative case of MCD in a young woman following surgical termination of pregnancy for hyperemesis gravidarum. In this case of steroid dependent MCD we have treated her successfully with Rituximab.

Case Report:
A 24-year-old woman with a background of asthma and atopic dermatitis developed nephrotic syndrome in May 2024, one month after elective termination of pregnancy at 13 weeks' gestation due to hyperemesis gravidarum. She presented with nephrotic-range proteinuria (urine PCR > 900 mg/mmol), hypoalbuminemia and preserved renal function. Renal biopsy confirmed MCD. She responded well initially to high-dose corticosteroids. However, she experienced multiple relapses within a year-first in December 2024 and again in April and June 2025-demonstrating steroid dependency. Introduction of tacrolimus as a steroid-sparing agent was partially effective but failed to prevent further relapses despite therapeutic drug levels. She has achieved complete remission following Rituximab (June 2025) and has remained in remission since then.
Conclusion:
This case underscores a potential temporal association between pregnancy termination and the development of podocytopathies emphasizing the importance of considering glomerular disease in post termination of pregnancy. It also highlights the challenges of managing frequently relapsing and steroid-dependent Minimal Change Disease in adults with a need to consider early introduction of steroid-sparing agents like Calcineurin Inhibitor or Rituximab.