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Category: Diabetes management and renal replacement therapies

Masked by dialysis: diabetic ketoacidosis diagnosed post-dialysis in end-stage kidney disease

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Background: Diabetic ketoacidosis (DKA) is uncommon in patients with end-stage kidney disease (ESKD) on haemodialysis (HD), due to altered insulin metabolism, reduced renal gluconeogenesis and the absence of osmotic diuresis. When it does occur, DKA may present atypically, creating diagnostic and management challenges. Worsening metabolic acidosis and hyperglycaemia after HD are particularly unusual. We report a case of DKA diagnosed after dialysis in a young woman with type 1 diabetes mellitus (T1DM), precipitated by Methicillin-Sensitive Staphylococcus aureus (MSSA) bacteraemia from a dialysis catheter infection.

Case presentation:A 30-year-old woman with longstanding T1DM who had recently started HD three times weekly for ESKD presented with fever, malaise and purulent tunnelled dialysis catheter discharge. Her diabetes regimen included subcutaneous Degludec and Aspart insulin. A recent HbA_{1c} was 56 mmol/mol. She was under evaluation for simultaneous pancreas–kidney transplantation and was not on an insulin pump.

Upon transfer, she underwent HD and commenced broad-spectrum intravenous antibiotics. Pre-dialysis investigations showed serum glucose 27.7 mmol/L, venous pH 7.36 and bicarbonate 15.3 mmol/L. DKA was not initially suspected and ketones were not checked. The infected catheter was removed after dialysis.

Several hours later, she developed worsening hyperglycaemia (29.2 mmol/L), metabolic acidosis (pH 7.30, HCO_3 14.1 mmol/L) and elevated capillary ketones (6.0 mmol/L), confirming DKA. Following JBDS guidance, a modified fixed-rate intravenous insulin infusion was started; fluid and potassium replacement were omitted due to overload and hyperkalaemia risks. DKA resolved within hours, though transient ketone rebound occurred, likely from substrate deficiency. MSSA bacteraemia was confirmed; antibiotics were rationalised, and she was discharged in a stable condition.

Discussion: DKA in ESKD is rare and can be masked by HD, which transiently corrects acidosis and may obscure early derangements. Altered insulin kinetics and absence of osmotic diuresis complicate recognition. JBDS guidelines (2023) recommend ketone testing in all T1DM patients on dialysis with glucose >15 mmol/L, regardless of symptoms.

Conclusion: DKA can develop after dialysis in ESKD patients. Worsening acidosis and hyperglycaemia after HD are unusual. Pre- and post-dialysis investigations may be misleading. Early ketone testing, cautious fluid and potassium replacement, and tailored insulin dosing are essential. Collaboration between diabetes, nephrology and critical care teams ensurs safe and effective management.

Category: Cardiovascular disease and diabetes

Urgent management of severe hypertriglyceridemia

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Severe hypertriglyceridemia is an urgent presentation that requires acute treatment.We describe two cases of hypertriglyceridemia which presented at the same time with different clinical features. There is no national guideline on the emergency management of clinically significant hypertriglyceridemia and we managed these cases using the local guidelines and input from a lipid consultant.

Case 1. A 38-year-old female with a history of non-alcoholic fatty liver disease, type 2 diabetes (T2DM), multiple myeloma and fibromyalgia presented to the emergency department with abdominal pain. Her medications were lenalidomide, acyclovir, fluconazole, dexamethasone, and insulin(100 U Toujeo,12 u Fiasp). She was diagnosed with acute pancreatitis based on her symptoms and an elevated amylase. The likely precipitating factor appeared to be either drug-induced or due to severe hypertriglyceridemia (38.6 mmol/L). Her cholesterol was 8.9 and LDL 1.5.There was no family history of hyperlipidaemia. She was initiated on insulin infusion to lower the triglycerides (TG), which came down to 18.8 mmol/L by the next day.

Case 2. A 29-year-old male patient, a no-smoker and non-alcoholic, presented to his GP with osmotic symptoms and feeling lethargic and generally unwell. The GP ran blood tests and found triglyceride levels of 58. He called the hospital and was asked to send the patient to the emergency department. The patient was started on an insulin infusion and his TG came down to 17 the following day.He did not have a family history of

cardiovascular or cerebrovascular disease. His HbA1c was 56 (a newly diagnosed diabetic on this admission), cholesterol 10.6 and LDL 2.4.

Given the improved triglyceride levels in both cases, they were weaned off insulin infusion over the next 24–48 hours. For long-term management, fenofibrate 160 mg/day was initiated. High-dose omega-3 fatty acids (4 g/day) were also started and strict dietary fat restriction was reinforced.In both cases, long-term goals were to reduce TG levels to <10 mmol/L to prevent recurrence of pancreatitis, and ideally <5 mmol/L for ongoing cardiovascular risk reduction. After discharge advice on measuring fasting lipid profile was given and they were also referred to the lipid clinic.

Lipoprotein lipase is the primary enzyme of TG lipolysis and hydrolyzes TG to form fatty acids. Hypertriglyceridemia has polygenic aetiology but can be associated with obesity, metabolic syndrome, poorly controlled diabetes and certain medications.Patients presenting with hypertriglyceridemia of this severity are rare. When they do present, the highest priority is to lower TG as fast as possible to prevent morbidity and mortality that result from end-organ damage (acute pancreatitis, acute kidney injury, acute respiratory failure and myocardial infarction) due to the increased viscosity of TG-rich plasma. Intravenous insulin increases TG breakdown by upregulating lipoprotein lipase activity and reducing its production.

Category: Diabetic emergencies

Utility of urine C-peptide creatinine ratio at first presentation with hyperglycaemia to guide diabetes classification and initial insulin therapy

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Background: Accurate classification of diabetes at first presentation is critical for guiding appropriate therapy. Current GIRFT guidance acknowledges diagnostic uncertainty in distinguishing type 1 from type 2 diabetes, particularly in adults who present with hyperglycaemia and overlapping clinical features. Traditional biomarkers such as islet autoantibodies have limitations, including false positives and negatives and reduced utility outside the early diagnostic window. The urine C-peptide creatinine ratio (UCPCR) offers a non-invasive, stable measure of endogenous insulin secretion and may help to identify absolute insulin deficiency at the time of the test.

Objective: To evaluate the utility of UCPCR at first presentation with hyperglycaemia and symptoms suggestive of type 1 diabetes (T1DM) in determining insulin requirement and refining diabetes classification.

Methods: We assessed UCPCR in patients presenting with hyperglycaemia and osmotic symptoms, prior to or shortly after initiation of insulin at first presentation to our Same Day Emergency Care (SDEC). UCPCR was measured in post-prandial urine samples collected in boric acid containers. Thresholds were applied based on validated cut-offs: <0.2 nmol/mmol indicating severe insulin deficiency (consistent with T1DM), 0.2 - 0.6 nmol/mmol intermediate secretion, and >0.6 nmol/mmol suggesting preserved insulin secretion (more typical of type 2 diabetes [T2DM] or MODY).

Results: Of 17 patients reviewed over a six-week period that were initially treated as T1DM, six (35%) were confirmed as having T1DM, 10 (58%) as T2DM and one (5%) as MODY on clinic review at six weeks utilizing serum antibodies, C-peptide function and clinical presentation. Of those with T2DM, the urine C-peptides led to a safe early discontinuation of basal/bolus therapy for nine patients, with one patient choosing to remain on basal/bolus therapy for planned pregnancy. Of these nine patients, five were switched entirely to metformin without incident and four remained on low-dose basal insulin. For the patients with T1DM, a significantly preserved C-peptide allowed reclassification as honeymoon and de-escalation in one patient (17%).

Conclusion: Preliminary data suggest that UCPCR can identify patients with preserved insulin secretion who may not require ongoing insulin therapy, allowing safe weaning and reclassification. In cases of diagnostic uncertainty, UCPCR complements clinical features and antibody testing, improving confidence in classification and reducing inappropriate insulin use. UCPCR is a practical outpatient tool that supports early differentiation between T1DM and T2DM. Its use at first presentation may reduce diagnostic ambiguity, align treatment with pathophysiology, and support personalised care. Integration into diagnostic pathways could enhance GIRFT recommendations and reduce unnecessary insulin exposure.

Category: Foot disease in diabetes

Overcoming limb weakness and foot drop: from immobility to independence in diabetic amyotrophy

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Background: Diabetic amyotrophy, or diabetic lumbosacral radiculoplexus neuropathy, is a disabling complication of diabetes characterized by asymmetrical proximal muscle weakness and pain, particularly in the lower limbs. This condition is attributed to microvascular ischaemic injury to the lumbosacral plexus and may coexist with distal symmetric neuropathy. While full structural recovery is uncommon, functional improvements are achievable with effective glycemic control and rehabilitation.

Case presentation: A 65-year-old male diagnosed with T2DM in early 2023 presented with bilateral lower-limb neuropathic pain and weakness, more pronounced on the left. His symptoms resulted in multiple falls and growing dependence on others for daily activities. Neurological examination revealed bilateral sensory loss in the lower limbs, left ankle dorsiflexion weakness and proximal muscle wasting. His HbA_{1c} at the time was 111 mmol/mol, indicating poorly controlled diabetes.

Nerve conduction studies confirmed the diagnosis of sensorimotor neuropathy with proximal involvement, consistent with diabetic amyotrophy. He was started on intensive insulin therapy, including Toujeo basal insulin and NovoRapid bolus. Shortly after starting insulin, he experienced a transient worsening of his leg weakness and pain, including foot drop, consistent with "insulin neuritis". This temporary worsening

resolved with continued therapy, and duloxetine was added for neuropathic pain, providing symptomatic relief.

By 2024, his HbA_{1c} improved to 78 mmol/mol and dropped to 37 mmol/mol by 2025. As his sugars stabilized, his functional status improved dramatically. From near immobility and dependence, he progressed to walking with a stick and, eventually, to regular gym workouts, including leg presses. Despite persistent mild distal sensory neuropathy, motor function and endurance improved significantly. His time in range now exceeds 98%, reflecting excellent diabetes control.

Discussion: This case demonstrates the typical clinical course of diabetic amyotrophy, with asymmetrical proximal weakness and pain. The phenomenon of "insulin neuritis," marked by transient worsening including foot drop following rapid glycemic correction, highlights the complexity of managing diabetic neuropathies. A multidisciplinary approach including glycemic optimization, pain management and physiotherapy was crucial in facilitating this patient's functional recovery.

Conclusion: Diabetic amyotrophy, while challenging, can show meaningful functional improvement with early recognition and comprehensive management. Although full structural reversal is uncommon, this case illustrates how a multidisciplinary approach can significantly improve quality of life, as observed in this patient who recovered from near immobility to working out in the gym. Awareness of insulin neuritis as a transient phase is critical for guiding patient expectations and ensuring adherence to therapy.

Category: Mental health and diabetes

A revolving door failure: T1DM, factitious disorder, self-harm and the limits of autonomy and integrated care

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Case report: A 37-year-old man with type 1 diabetes mellitus (T1DM) since the age of 12 had recurrent presentations with severe hypoglycaemia and hyperglycaemia.

His social history is notable for long-term unemployment, financial instability, social isolation and a lack of family support, which he identifies as key triggers for his poor mental health. The drastic fluctuations in hypoglycaemia and hyperglycaemia led to suspicions that he was self-harming by administering insulin overdoses.

Despite this, he received a donation-after-brainstem-death simultaneous pancreas-kidney (DBD-SPK) transplant in 2024.

However, his self-harming behaviours escalated post-transplant, including non-adherence to immunosuppressants. Unfortunately, the pancreas transplant has now failed.

Subsequent attempts to transition him to Continuous Subcutaneous Insulin Infusion (CSII) pump therapy have been consistently refused.

Conclusion: Management of T1DM is uniquely complex when co-morbid mental illness leads to the disease itself being used for self-harm. This case illustrates the ethical dilemma of managing a sabotaged, life-saving organ transplant and the limitations of our standard multidisciplinary model, and the costs to both the patient and our services due to recurrent admissions.

Despite involvement of our mental health specialist teams, we have not been able prevent deterioration of his mental health or progression of his diabetes. Refusal of CSII highlights this conundrum: he is rejecting the treatment because it would prevent its use for self-harm.

Type 1DM services have the platform to be truly integrated, from the psychiatry teams, social workers, substance misuse teams and community support, to provide early intervention.

Questions for consideration:

How should transplant assessment committees practically and ethically weigh a patient's history of medical self-sabotage? Is it discriminatory to deny a life-saving transplant, or is it a necessary act of resource stewardship?

Would use of CSII be of significant benefit to his outcomes?

Is 'glycaemic control' or 'transplant preservation' the correct primary outcome for this patient? Or should our main goal be harm reduction and psychological stability?

Category: Diabetes technology in people with T1DM

HCL roll-out and agesm: is this a problem?

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NICE TA943 (2023) recommends hybrid closed-loop (HCL) therapy in type 1 diabetes (T1DM), irrespective of age. Older adults are often underrepresented in clinical trials and there are scant data in this group. Possible barriers include frailty, cognition and inability to use new technology. This audit assessed whether adults aged 70 years and over meeting eligibility criteria were appropriately identified and offered HCL/pump therapy.

Methods: A retrospective audit was carried out of adults 70 years and over with T1DM attending the diabetes specialist service at York Hospital (March 2024–March 2025). Clinic records, GP summaries and CGM data were accessed. Exclusions included diabetes misclassification, non-adherence to appointments or existing HCL use. Eligibility was evaluated against NICE TA943 hypoglycaemia criteria (Gold score ≥4, CGM time <4 mmol/L >7% or time <3 mmol/L >1%) and HbA_{1c} threshold ≥69 mmol/mol (aligned with older TA151; our ICB guidance at that time).

Results: Of 71 patients, 62 were included (mean age 76.4 years, range 70–92; 27 male, 35 female).

- 38 met the eligibility criteria
- Hypoglycaemia (TA943): 5 eligible, 0 offered HCL. Possible reasons for not offering HCL were: patient refusal to use CGM (n = 2), improved hypoglycaemia awareness after insulin adjustments, inconsistent Gold score reporting, absence of formal pump offer/ discussion for pump eligibility despite clear qualifying hypoglycemia history.
- HbA_{1c} ≥69 mmol/mol: 33 eligible, 0 offered pump/HCL.

Barriers documented included frailty, dementia, third-party insulin administration, incomplete structured education, CGM non-adherence and anxiety about technology. In 22/33 (67%), no eligibility discussion was recorded.

Conclusion: None of the 38 eligible patients were offered advanced diabetes technology. Documentation of assessment and discussion was inconsistent, indicating a gap between NICE recommendations and clinical practice. Standard proforma-based eligibility review with documentation and an interval re-audit is recommended.

Category: Obesity management in diabetes

From extreme insulin resistance to reactive hypoglycemia: a metabolic transition following bariatric surgery

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Background: Severe insulin resistance (IR) represents one of the most difficult clinical phenotypes of type 2 diabetes mellitus (T2DM), often associated with obesity, non-alcoholic fatty liver disease (NAFLD) and polycystic ovarian syndrome (PCOS). In such cases, extreme hyperglycemia persists despite maximal therapy. Major weight reduction can restore insulin sensitivity and enable remission of diabetes. This case demonstrates complete metabolic remission following profound improvement in insulin sensitivity after structured weight-loss interventions.

Case presentation: A 52-year-old woman with T2DM, NAFLD, central obesity, hypertension, PCOS and hypothyroidism was referred in 2020 for uncontrolled diabetes. She was intolerant to metformin, GLP-1 receptor agonists and SGLT2 inhibitors, and required high-dose basal–bolus insulin (>200 U/day; Toujeo and Tresiba together, along with Humalog). Capillary glucose values often exceeded 30 mmol/L without hypoglycemia, consistent with marked insulin resistance.

Baseline investigations showed HbA_{1c} 86 mmol/mol, cortisol 1200 nmol/L (Cushing's was excluded by ODST and 24 hour urine cortisol), ALT/AST mildly elevated and ultrasound evidence of NAFLD. The thyroid profile was consistent with adequately treated hypothyroidism. By 2022 her HbA_{1c} rose to 90 mmol/mol despite escalating insulin therapy. She exhibited central adiposity, muscle wasting and clinical lipoatrophy. Owing to extreme insulin requirements and metabolic complexity, she was referred to a national institute for evaluation of severe insulin resistance.

In 2023, she underwent sleeve gastrectomy which resulted in significant weight loss, improved glycemic control (HbA1c 53 mmol/mol) and insulin discontinuation. Persistent postprandial hyperglycemia prompted a gastric bypass in 2024, following which she achieved a total weight loss of 5.5 stone (≈35 kg) and normalization of glycemia (HbA_{1c} 37 mmol/mol) without pharmacotherapy.

By late 2024, the patient reported nocturnal and exertional hypoglycemia, with post-prandial dips confirmed on continuous glucose monitoring. Laboratory tests showed normal fasting glucose, insulin and C-peptide, as well as normal cortisol, thyroid and liver profiles.

Discussion and conclusion: This case illustrates the remarkable metabolic adaptability of T2DM. Restoration of insulin sensitivity through major weight loss transformed a state of extreme insulin resistance into sustained diabetes remission. The emergence of reactive hypoglycemia reflects heightened post-prandial insulin responsiveness in the remission phase. Long-term monitoring and nutritional guidance remain crucial to maintain stability and prevent hypoglycemia.

Category: Diagnosis and management of monogenic diabetes

A novel KCNJ11 mutation of uncertain significance

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A 22-year-old man was referred to the diabetes clinic. He was diagnosed aged 20 in Pakistan after presenting with weight loss, polydipsia, polyuria and HbA_{1c} of 64mM/M. Treatment was initiated with gliclazide 60mg OD but stopped due to hypoglycaemia. In the UK metformin was started and gliclazide 80mg restarted but again it was stopped due to hypoglycaemia.

At his review in clinic it was noted that he had no other past medical history and did not drink alcohol nor smoke. He was from Peshawar in Pakistan; both his parents are from Pakistan and are cousins. His BMI was 22.57kg/m^2.

Blood samples taken in clinic showed an HbA_{1c} of 89mM/M. Diabetes antibodies were negative and C-peptide was 226pmol/l (glucose 16.9mmol/l). Metformin was continued and once daily Lantus was started.

At a later follow-up it was noted that he had stopped insulin for a period of 10 days when his prescription ran out. Fasting blood glucoses had been in the teens, but he had not felt unwell.

Family history was revisited. Many family members had been diagnosed with T2DM, his mother in her 30s, his father in his 20s and both maternal grandparents. His brother has pre-diabetes. Of note, his father had been very sensitive to sulfonylureas.

Given the strong family history and unclear diagnosis, monogenic diabetes was considered. Testing identified a novel missense variant in the KCNJ11 gene. The significance of this is uncertain. Of note, specific gain of function variants in KCNJ11 cause transient neonatal diabetes and this can present as MODY with non-penetrance of diabetes mellitus in the neonatal period. It is characterised by sensitivity to sulfonylurea therapy, which both our patient and his father have experienced.

We have discussed genetic testing of family members, which our patient is considering. Low-dose gliclazide 20mg has been started as a trial with a plan to titrate down Lantus. We are interested to see how our patient responds to very low dose sulfonylurea treatment.

This case illustrates the importance of taking a thorough history around the time of diagnosis and recognising features which are suggestive of monogenic diabetes. MODY is less frequently investigated and diagnosed in non-white populations in the UK. We need to work to remove biases around genetic testing. This is a newly identified mutation of uncertain significance which could be further investigated to see if it is pathogenic. However, this requires samples from affected family members, which can be difficult if they are abroad.