Abstracts from ABCD Diabetes Update 2026

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The winning abstracts were published in the June issue. The following are all the abstracts presented

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Category: Diagnosis and management of monogenic diabetes

A case of mitochondrial diabetes – seven years on

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A 34-year-old Indian man was referred to the diabetes centre by his GP. He was originally diagnosed with type 2 diabetes (T2DM) in India seven years previously as he was antibody-negative. On direct questioning at the time of presentation, however, he said that he had lost a significant amount of weight and was suffering with symptoms of hyperglycaemia. He was commenced on once-daily Tresiba and has continued on this ever since. He does not suffer with hypoglycaemia and has never had a hospital admission for complications of diabetes. He has no other past medical history and is taking no other medications.

He has a strong family history of diabetes. Both his mother and father were diagnosed in their fourth decade and two of his three siblings were diagnosed in their twenties, one with GDM and the other withT2DM. All require insulin therapy.

His symptoms of hyperglycaemia resolved with insulin therapy but he has struggled to gain weight, which is his main concern. His weight was 50 Kg, with a BMI 17 Kg/m^2 and his examination was unremarkable except for an area of lipohypertrophy on his thigh. His ongoing difficulty with weight gain raised the suspicion of insulin deficiency, so he underwent further investigations. His blood tests demonstrated a HbA_{1c} 68mmol/mol, C-peptide 287 pmol/L with glucose 8.5 mmol/L, normal renal and thyroid function and normal ferritin level. MRI pancreas was normal.

He was referred for genetic testing, which revealed a mitochondrial variant m.3243A>G MT-TL in keeping with a diagnosis of maternally inherited diabetes and deafness. He was provided with a Freestyle Libre 3 which demonstrated a postprandial glucose rise. Novorapid was added to his insulin therapy. He reports no symptoms of hearing impairment but has been referred for formal audiology and an echocardiogram.

Category: Mental health and diabetes

Eating disorder and type 1 diabetes - a difficult combination

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A 34-year-old woman was diagnosed with type 1 diabetes (T1DM) at the age of 20 years. She has struggled with her diabetes, predominantly because she also suffers with an eating disorder. Throughout the first 3-4 years of her diagnosis she had frequent admissions in diabetic ketoacidosis (DKA), mainly because she was omitting for fear of weight gain.

In 2019 her HbA_{1c} was 95mmol/mol. She was commenced on Freestyle Libre and this helped her to focus and improved her blood glucose control. She began to take her Tresiba more regularly; however, her weight also increased with tighter blood glucose control and this caused much anxiety for her. Her consistency in using her glucose sensor over the years has been variable, with average twice-daily scans and glucose running in the 20-25mmol/L range.

She was referred to the King's College Type 1 diabetes disordered eating programme in January 2020 and underwent a course of CBT to help with thoughts around insulin omission. She attended regularly reviews with a psychiatry consultant and three-monthly review with the specialist diabetes psychiatrist.

She struggled to engage with services during the pandemic with virtual appointments and telephone calls. She also experienced two family bereavements in this time. She participated in weekly CBT virtual sessions from April to October 2020 with a psychotherapist and during this time she moved in with her boyfriend. Her baseline HbA_{1c} was 109mmol/mol, which improved to 85mmol/mol. Following this therapy she reported a 75% improvement in binge eating behaviour and 50% improvement with insulin doses and started giving an evening meal dose of Novorapid. Unfortunately, these both deteriorated again after these regularly sessions ceased.

She had a hospital admission in April 2022 for treatment of an abscess on the scalp and at this point was binging 3-4 times per week, though her boyfriend was unaware of the ongoing problem.

She got married in August 2023. The build-up to this prompted concerns over weight and a preoccupation with her body appearance on her wedding day. Therefore she stopped taking Novorapid again but did continue with her Tresiba. Just before their wedding her fiancé began attending appointments with her as he wanted to be more involved. On review after her wedding she agreed to take 1 unit Novorapid with meals. This was subsequently increased to 2 units a few months later, with good compliance and no deterioration in her mental health.

She was reviewed in pre-conception clinic in February 2024. She was now using her FSL more regularly and giving herself insulin injections four times per day, with TIR 21%, average glucose 14.3 mmol/L and HbA_{1c} 81 mmol/mol. She was being reviewed regularly by the dietician and diabetes psychiatrist in the eating disorder type 1 clinic at Mile End hospital. In August 2024 she was started on an Ypsopump insulin pump with a Dexcom G6 CGM, with a starting total daily dose of 29 units. There was concern over her eyes; she had avoided retinal screening appointments for four years. Attending this appointment was one of the conditions of starting an insulin pump. This revealed pre-proliferative R2 bilaterally and she was referred for laser therapy.

From a glycaemic point of view she has done extremely well on the insulin pump, although this has been associated with

weight gain, which has caused her some increased anxiety. She conceived in May 2025 with a pre-pregnancy HbA_{1c} of 45 mmol/mol and TIR of 74%. She is currently 28 weeks pregnant and is doing very well, although concern remains regarding a future relapse in her eating disorder. She continues to be reviewed regularly both within the diabetes disordered eating clinic and the Type 1 diabetes clinic.

Category: Diabetes management and renal replacement therapies

Mind the weekend gap: improving DKA transitions and discharge efficiency at a district general hospital in Essex

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Background: This quality improvement project (QIP) evaluates whether implementing a structured Friday transition plan improves discharge efficiency and reduces weekend-related delays for diabetic ketoacidosis (DKA) patients at a district general hospital in Essex.

DKA management requires timely transition from fixed-rate insulin infusions (FRI) to variable rate insulin (VRI), followed by stepdown to subcutaneous insulin. Delays in this process—especially over weekends—can prolong admissions and increase staff workload, as patients require frequent venous blood gases and medical reviews. Prompt and structured transitions can reduce both staff burden and patient risk. Prolonged stays also increase costs, as an NHS hospital bed costs approximately £345 per day. Princess Alexandra Hospital (PAH), a district general hospital in Essex, admits around 100 DKA patients per month. Improving insulin transitions and discharge efficiency could therefore enhance patient flow and generate significant cost savings.

Methods: A retrospective review of 16 DKA inpatients (March–April 2025) was conducted. Data included insulin transition timelines, presence of a documented stepdown plan, weekend handover practices, length of stay (LoS), and discharge delays. Statistical analyses compared LoS between weekday and weekend discharges, and between patients with and without a documented stepdown plan. Cost-effectiveness was also determined.

Results: Patients discharged over the weekend had a mean LoS of 8.4 days, compared with 14.9 days for weekday discharges, though this difference was not statistically significant ($t = –1.24$, $p = 0.24$). Patients with a documented stepdown plan had a mean LoS of 4.7 days, compared with 13.4 days for those without, a difference of 8.7 days saved per admission ($t = –1.74$, $p = 0.18$). Although underpowered, this finding is clinically important. Since each inpatient bed costs £345 per day, this equates to approximately £3,000 saved per admission. Applied to 17 eligible DKA patients per month, this could save £51,000 monthly — approximately £612,000 per annum in unnecessary bed use.

Conclusion: This QIP demonstrates a strong trend toward shorter hospital stays in DKA patients with structured stepdown planning. Implementing a standardized Friday stepdown protocol, improving weekend handovers and ensuring DSN support could enhance discharge efficiency, reduce unnecessary bed occupancy and yield substantial annual savings—potentially over £3.6 million at PAH.

Reference
1. https://questions-statements.parliament.uk/written-questions/detail/2023-03-14/165361

Category: Diagnosis and management of monogenic diabetes

Familial case of transient neonatal diabetes due to paternal 6q24 duplication

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Background: Transient neonatal diabetes mellitus (TNDM) is a rare form of monogenic diabetes that presents within the first six months of life. It is typically caused by abnormalities in the imprinted region of chromosome 6q24, most commonly due to paternal duplication or uniparental disomy. Although it resolves in infancy, relapse often occurs in adolescence or adulthood. Relapsed TNDM can be misclassified as type 1 or type 2 diabetes (T1DM or T2DM), leading to inappropriate long-term management strategies.

Case summary: A 62-year-old woman with insulin-treated diabetes was referred for optimization of glycaemic management. History identified that she had diabetes as a newborn baby, which resolved spontaneously. She was re-diagnosed with diabetes in her twenties during pregnancy. Despite large insulin doses (approximately 200 units/day) combined with metformin and dapagliflozin, glycaemic control remained suboptimal. She had a body mass index of 28 kg/m2 and had no dysmorphic features or learning difficulties. There was no evidence of insulin resistance on clinical examination. Her

C-peptide level was 309 pmol/L (reference 298–2350), indicating preserved endogenous insulin secretion. Autoimmune antibodies (GAD, IA2, ZnT8) were negative.

There was a strong family history of diabetes, affecting her father and four siblings. One sister had also been diagnosed with diabetes during pregnancy at age 24. She was treated as T1DM, though she had never developed ketoacidosis and did not have any history of neonatal diabetes.

Initial testing was undertaken of the sister with a history of neonatal diabetes. Methylation-specific MLPA (MS-MLPA) identified a paternal duplication of the 6q24 locus, confirming the diagnosis of 6q24-related TNDM. There was a further positive test in the second sister. They were referred for genetic counselling and were advised that each offspring has a 50% chance of inheriting the duplication; only paternal transmission leads to disease expression. The patients were referred for cascade screening of relatives.

Discussion: This case shows the importance of a full history to identify features that may support the diagnosis of monogenic diabetes in adults with a history of neonatal diabetes, preserved C-peptide and negative autoantibodies. Identification of a 6q24 duplication refines diagnosis, guides management and informs genetic counselling. Women with 6q24-related TNDM are at risk of relapse during pregnancy and may present with gestational diabetes. Genetic counselling is essential to clarify inheritance patterns and recurrence risks.

Reference
Temple IK, Mackay DJG. Diabetes Mellitus, 6q24-Related Transient Neonatal. GeneReviews®, University of Washington, Seattle, 2018.

Category: Diabetes technology in people with T1DM

Optimising insulin pump initiation: a contact-mapped model for HCL transition and follow-up

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The NHS's five-year rollout of Hybrid Closed Loop (HCL) technology represents a transformative shift in type 1 diabetes (T1DM) care, offering patients improved glycaemic control through automated insulin delivery. However, the success of this technology hinges not only on its clinical efficacy but also on the robustness of the onboarding process. Historically, insulin pump initiation has been fragmented, with multiple unstructured contacts leading to inefficiencies, variable patient experiences and inconsistent outcomes.

In response, Harrogate and District NHS Foundation Trust has developed a streamlined, patient-centred pathway to support safe and effective transition to HCL therapy. This model prioritises structured education, timely clinical oversight and efficient use of resources, while maintaining flexibility to accommodate varying levels of patient complexity and prior experience.

The pathway begins with an initial outpatient or ad-hoc clinic decision to progress to HCL therapy. Patients are then referred for tailored dietetic education, delivered via a blend of face-to-face, telehealth and online modalities. Following this, a dedicated technology session introduces pump options and enables joint decision-making. Patients who complete both education components are reviewed in a multidisciplinary Pump MDT to confirm their eligibility and readiness.

All patients then undergo a pre-pump telehealth appointment to set individualised targets and expectations. The planned start appointment occurs at week 6. A face-to-face review at week 12 ensures that basal delivery has stabilised; therapy can be fine-tuned, allowing time for algorithmic adaptation and behavioural adjustment. Patients are then transitioned to a Patient-Initiated Follow-Up (PIFU) model with a two-year recall, ensuring long-term safety and autonomy.

Each pathway is mapped with a "lived experience" schedule of contacts, including outpatient attendances, telephone and email support, and education sessions. This mapping enables services to anticipate workload, allocate resources effectively and maintain consistent standards of care. Contact intensity ranges from 6 to 13 interactions over a 16-week period, depending on patient group:

- Pump-naïve patients require early face-to-face education and then early remote support.
- Existing CSII users benefit from a lighter-touch approach with remote modalities.
- Complex patients need frequent, personalised input.
- HCL mass upgrades are supported through industry-led mass upgrade days and remote education.

A key enabler of this model is the Diabetes Technologist role, which provides administrative, financial and clinical support throughout the pathway. This role allows consultant-led contacts to be converted into ad-hoc interactions within working hours, improving responsiveness and reducing reliance on fixed clinic sessions.

By embedding structure, flexibility and multidisciplinary collaboration, this pathway ensures that patients receive the right support at the right time, promoting safety, self-management and long-term engagement with HCL technology. It helps to solidify a clinician's expectations of the support generally required for each case.

Category: Diabetic emergencies

When life-saving therapy turns deadly: hybrid closed-loop as the solution to insulin allergy

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Insulin allergy is a rare but potentially life-threatening complication in patients with diabetes, with a prevalence below 2%. It may present as local reactions or systemic hypersensitivity, limiting insulin use and glycaemic control. Management requires a multidisciplinary approach, and in severe cases, immunosuppressants may be considered.

Case presentation: A 60-year-old male with type 3c diabetes after pancreatic cancer surgery was started on a basal–bolus insulin regimen. Within one week, he developed widespread urticaria and pruritus, leading to diabetic ketoacidosis (DKA) as he was unable to continue insulin. Testing for insulin delivery devices (needles, pen materials) was negative, but intradermal and serological testing confirmed hypersensitivity to all insulin preparations, with lowest reactivity to insulin glulisine (Apidra®).

Following multidisciplinary discussion, immunologists considered immunosuppressive therapy but opted first for desensitisation. The patient was gradually desensitized to insulin glulisine and subsequently commenced on a CamAPS FX hybrid closed-loop (HCL) insulin pump using glulisine.

Discussion: True insulin allergy is a major therapeutic dilemma, particularly in absolute insulin deficiency. Traditional strategies include switching analogues, antihistamines, corticosteroids or desensitization. Immunosuppressants are sometimes required but carry significant risks. In this case, HCL therapy allowed continuous, precise, low-dose insulin delivery, which minimised immune reactivity and avoided immunosuppression. The patient achieved >70% time-in-range and excellent glycaemic control, and had no recurrence of systemic allergic reactions.

Conclusion: When insulin becomes the "enemy," combining desensitisation with advanced diabetes technology offers a safe and effective solution. HCL therapy can obviate the need for high-risk immunosuppressants in patients with true insulin allergy, providing both glycaemic stability and improved quality of life.

Category: Mental health and diabetes

Breaking the cycle: psychosocial drivers of recurrent admissions in adults with type 1 diabetes: a case series

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Background: Type 1 diabetes mellitus (T1DM) poses a significant challenge, with a heavy psychological burden that impacts patient engagement, glycaemic control and the progression of complications. This is often compounded by mental health disorders, lack of family support, unemployment, homelessness and substance misuse. Many patients experience recurrent hospital admissions due to these complex issues and the absence of adequate support. This case series presents seven

patients with recurrent admissions associated with T1DM at our tertiary hospital in London.

Case presentation: We identified seven patients who were well known to our diabetes teams, each facing psychosocial challenges that may contribute to recurrent hospital admissions. Common themes included:

Psychiatric co-morbidity: five patients had formal diagnoses, including personality disorders, schizoaffective disorder and mood disorders, often linked to impulsivity and self-neglect.

Substance misuse: seen in five patients, complicating glycaemic control and treatment adherence.

Social determinants: one patient was unemployed, two had unstable housing, two faced social isolation and two had limited family support.

Disengagement from care: many patients demonstrated poor engagement with diabetes services despite access to specialists.

Medical complexity and burnout: Chronic complications and repeated admissions contributed to psychological distress, feelings of hopelessness and poor adherence.

One patient had more than 50 emergency admissions in a year, due to hyperglycaemia, hypoglycaemia, anxiety, depression, and complications such as diabetic gastroparesis and syncope. These admissions were greatly reduced after a multidisciplinary team (MDT) approach and pancreas transplant.

All seven patients received regular input from psychiatry and psychology services, with four referred to our High Intensity User (HIU) team for close collaboration with primary care to help reduce readmission rates.

Discussion: These cases highlight the need for integrated, multidisciplinary care involving diabetes specialists, mental health professionals, social workers and addiction services. Early identification of psychiatric disorders and substance misuse, along with involvement of HIU teams, may help reduce readmissions and improve patient outcomes through better coordination between secondary and primary care.

Conclusion: We are conducting a Trust-wide audit of recurrent diabetes-related admissions in T1DM patients to understand the medical and psychosocial factors that may contribute to these readmissions. Our goal is to create a pathway linking T1DM services with the HIU team, providing integrated support from psychiatry, substance misuse services, and community resources like the London Ambulance Service (LAS) and primary care, to improve care consistency, reduce length of stay and prevent avoidable admissions.

Category: Diabetic emergencies

Paclitaxel-induced hyperglycaemia leading to diabetic ketoacidosis in a patient with type 1 diabetes: a case report

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Background: Hyperglycaemia occurs in up to 30% of patients receiving chemotherapy. Individuals with type 1 diabetes mellitus (T1DM) are particularly vulnerable to ketosis and diabetic ketoacidosis (DKA). While glucocorticoids are well-recognised contributors, emerging evidence suggests that the chemotherapeutic agent paclitaxel may also exacerbate hyperglycaemia. Animal studies have demonstrated paclitaxel-induced hyperglycaemia, but human data remain limited.

Case presentation: A 61-year-old woman with T1DM and metastatic breast cancer developed DKA following her first cycle of paclitaxel. She had previously maintained good glycaemic control using multiple daily injections and carbohydrate counting. Within 24 hours of chemotherapy, she became drowsy and confused. In the emergency department, laboratory tests confirmed DKA. She was treated with intravenous insulin and fluids. Paclitaxel was withheld until recovery. The diabetes team reinforced sick-day rules, including maintaining basal insulin, increasing total daily insulin by 10–20% if ketones persist, ensuring hydration, and seeking urgent care if ketones fail to resolve.

Outcome: The patient resumed chemotherapy with enhanced glycaemic monitoring and multidisciplinary support, without further DKA episodes.

Conclusion: Paclitaxel may precipitate hyperglycaemia that can lead to DKA in T1DM patients when insulin adjustments are inadequate. Education on sick-day rules and proactive collaboration between oncology and diabetes teams are essential to improve safety.

Category: Diabetes technology in people with T1DM

Optimizing glycaemic control in a patient with type 1 diabetes using a hybrid closed-loop insulin pump: addressing hypoglycaemia unawareness and bolus management challenges

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Background: Hybrid closed-loop (HCL) insulin delivery systems significantly improve glycaemic control in individuals with type 1 diabetes mellitus (T1DM). However, effective use relies on appropriate user engagement, including accurate carbohydrate estimation and timely pre-meal bolusing. We present a case highlighting the importance of structured education and behavioural support in optimizing HCL therapy, particularly in a patient with hypoglycemia unawareness. Misuse of bolus dosing features can lead to significant postprandial glucose variability and hypoglycaemia.

A 68-year-old woman with T1DM was being managed on the Omnipod 5 HCL system integrated with a Freestyle Libre 2 Plus sensor. Despite prior structured education (DAFNE), the patient reported ongoing difficulty with carbohydrate counting and had been administering insulin boluses based on personal estimates rather than using the pump's bolus wizard. She reported increased stress related to family illness and moving house, resulting in inconsistent pre-meal bolusing and variable carbohydrate estimation. This approach led to inconsistent bolus insulin dosing and frequent post-meal hypoglycaemic episodes, as evidenced by continuous glucose monitoring (CGM) data showing 8% time below range and multiple postprandial lows.

Intervention and plan: The patient was counselled on the importance of pre-bolusing 15 minutes before meals and using the bolus wizard for accurate dose calculation based on carbohydrate intake and insulin sensitivity factors. Given persistent challenges with carbohydrate counting, a customised or simplified meal plan was also proposed on Omnipod5, incorporating pre-programmed typical meals or approximate carbohydrate groupings to support safer insulin dosing and improve time in range.

Outcome and follow-up: A referral was made for further diabetes specialist nurse input to reinforce carbohydrate

estimation skills and assess the impact of simplified meal strategies. Follow-up is planned to review glycaemic trends and assess improvement in time below range after implementing structured bolus management strategies.

Discussion: This case highlights that even with advanced insulin delivery technology, user behaviour remains critical to achieving optimal outcomes. Lack of pre-bolusing and inconsistent carbohydrate estimation can significantly impact glycaemic stability and increase hypoglycaemia risk. Structured education, simplified meal management strategies and ongoing multidisciplinary support are essential to maximize the benefits of HCL systems in real-world settings.

Conclusion: Effective utilization of HCL systems requires patient engagement and continuous education and support. Early identification of behavioural barriers, such as failure to pre-bolus or reluctance to use dosing algorithms and carbohydrate counting, allows timely intervention to enhance glycaemic outcomes and safety.

Category: Foot disease in diabetes

Beyond 'disengagement': the critical role of communication in high-risk diabetic foot disease

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Background: Diabetic foot ulcers complicated by infection and critical limb ischaemia frequently result in amputation. Communication barriers and unrecognised psychosocial factors may lead to premature assumptions about treatment limitations. This case illustrates how interpreter-mediated multidisciplinary care can facilitate successful limb salvage.

Case report: A 62-year-old Romanian man with a 25-year history of type 2 diabetes mellitus (T2DM) complicated by peripheral neuropathy, peripheral vascular disease, retinopathy, chronic kidney disease, hypertension and heart failure, was admitted with a septic, gangrenous left fifth toe.

He had previously undergone right below-knee amputation (in 2023) but had remained ambulant until progressive left foot ischaemia led to immobility in July 2025. He had received extensive antimicrobial therapy previously, with wound cultures growing multi-drug-resistant MRSA and carbapenem-resistant organisms (CRO).

MRI confirmed osteomyelitis with exposure of the fifth metatarsal head. CT angiography demonstrated diffuse arterial calcification, severe popliteal stenosis, near-complete occlusion of the anterior tibial artery, chronic total occlusions of the posterior tibial and peroneal arteries, and single-vessel runoff.

Previous admissions had not consistently used translation services, leading to the patient being perceived as disengaged and unlikely to engage with therapies. During this admission, translation services were consistently engaged across the diabetic foot multidisciplinary team (MDT) and the vascular, interventional radiology and podiatry teams.

Three management options were discussed: (1) staged debridement and revascularisation; (2) above-knee amputation; or (3) palliative management.

Outcome: Collateral history from family and interpreter input revealed that his apparent disengagement stemmed from untreated depression related to his immobility. Reassessment confirmed his motivation for limb salvage.

He underwent percutaneous transluminal angioplasty of the left superficial femoral, popliteal and posterior tibial arteries using 5–7 mm and 2–2.5 mm balloons, achieving excellent angiographic flow to the wound. Post-procedure, he demonstrated improved mood and motivation to engage with diabetic foot care and rehabilitation.

Discussion:This case highlights the dangers of misinterpreting language barriers, and the importance of formal translation services in reframing the patient's perceived non-compliance. Effective multidisciplinary care for high-risk patients must include robust systems to overcome logistical, language and psychosocial barriers to ensure truly informed, shared decision-making.

Despite revascularisation, his prognosis remains guarded, since he has grown multi-drug-resistant organisms and has significant co-morbidities, underscoring the need for close MDT follow-up.

Diabetic foot patients often require multiple hospital follow-ups across different specialties. Addressing logistical barriers to coordinate care is essential, as these patients are at high risk of missing appointments.

Category: Mental health and diabetes

Dilemma of diabetes management in the context of complex mental health needs

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Diabetes mellitus is a long-term multisystem condition that interacts closely with psychological wellbeing, neuro-developmental conditions and health system factors. We present the case of a young man living with type 1 diabetes mellitus (T1DM) for more than 20 years. Achieving safe and sustained glycaemic management has been particularly challenging due to co-existing complex mental health and learning needs.

He has a background of autism spectrum disorder, moderate learning difficulties (including dyslexia, dyspraxia and dyscalculia), anxiety, depression, and auditory and visual hallucinations. He has been known to diabetes services for more than a decade. Since September 2025, he has experienced three hospital admissions with diabetic ketoacidosis (DKA). His most recent admission coincided with an acute mental health crisis and suicidal ideation, during which he self-harmed, resulting in a fracture of the fifth metacarpal of the right hand.

His diabetes is managed using a multiple daily injection regimen with insulin glargine U300 (Toujeo) 24 units once daily and insulin aspart (Novorapid) 8 units with breakfast, 5 units with lunch, and 3 units with the evening meal. He commenced continuous glucose monitoring using Dexcom ONE+ in September 2025. His most recent HbA_{1c} was 84 mmol/mol (9.8%).

- Using HbA_{1c} 84 mmol/mol, the estimated GMI ≈ 9.7–9.9%
- Typical corresponding CGM pattern
    - Time in range (3.9–10 mmol/L): ~35–40%
    - Time above range (>10 mmol/L): ~50–55%
    - Very high (>13.9 mmol/L): ~25–30%
    - Time below range (<3.9 mmol/L): ~5–8%

He also takes fludrocortisone 100 micrograms daily (for suspected autonomic dysfunction with postural symptoms) and vortioxetine 5 mg daily for anxiety and depression. He has developed diabetes-related complications, including non-diabetic proliferative retinopathy (NDPR) and lipohypertrophy.

He is a non-smoker, with a BMI of 20.7 kg/m^2.

A central challenge in this case is his limited engagement with ongoing diabetes care. He does not engage with community diabetes services and is unable to sustain regular weekly or fortnightly follow-up appointments. He expresses a strong preference to be reviewed only by a specific consultant and diabetes specialist nurse, which limits continuity of care at times of service pressure. His mental health needs significantly affect his capacity to engage with insulin optimisation, glucose review and sick-day management. In turn, recurrent hyperglycaemia and hospital admissions contribute to psychological distress, reinforcing a cyclical interaction between mental health and diabetes outcomes.

Care is provided through a multidisciplinary approach involving endocrinologists, diabetes specialist nurses, liaison psychiatry, learning disability services, mental health crisis teams and inpatient services. Despite intensive input, achieving stable diabetes outcomes remains difficult, highlighting the complexity of managing T1DM in the context of significant mental health needs.

This case illustrates the importance of integrated mental and physical healthcare, flexibility in service delivery and realistic goal-setting when supporting people living with diabetes and complex mental health conditions.

Category: Diagnosis and management of monogenic diabetes

Tirzepatide as a therapeutic option in monogenic diabetes: evidence from two genetically confirmed cases

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Key words: Monogenic diabetes, HNF1A, tirzepatide, glycaemic control, weight reduction

Introduction: Monogenic diabetes is a rare, inherited form of diabetes resulting from single-gene mutations, typically following an autosomal dominant pattern. The most common subtype, accounting for approximately 70% of cases, is caused by mutations in the Hepatic Nuclear Factor 1-alpha (HNF1A) gene. Management usually involves lifestyle optimisation and oral hypoglycaemic agents—most commonly sulfonylureas—with insulin rarely required. However, hypoglycaemia and variable adherence can limit long-term effectiveness. Tirzepatide, a dual glucose-dependent insulinotropic polypeptide (GIP) and glucagon-like peptide-1 (GLP-1) receptor agonist, has demonstrated significant benefits in weight reduction and glycaemic control in type 2 diabetes (T2DM), though its use in monogenic diabetes remains scarcely reported. We present two genetically confirmed HNF1A-related monogenic diabetes cases treated with tirzepatide, highlighting notable improvements in metabolic outcomes.

Case presentations: Case 1: A 27-year-old female presented with fatigue and elevated home glucose readings. Her HbA_{1c} was 67 mmol/mol, and she had a strong maternal history of T2DM. Genetic testing confirmed an HNF1A mutation. Initial treatment with glibenclamide 2.5 mg OD and later gliclazide 20 mg OD was complicated by recurrent hypoglycaemia and inconsistent adherence. Her baseline weight was 75.7 kg (BMI 28.7 kg/m^2). Motivated to lose weight, she independently initiated tirzepatide and discontinued gliclazide. At follow-up, HbA_{1c} had improved to 32 mmol/mol and BMI reduced to 23 kg/m^2. She tolerated tirzepatide well and received pre-conception counselling with reinforcement of adherence strategies.

Case 2: Her 26-year-old brother, also confirmed to have an HNF1A mutation, initially presented with HbA_{1c} of 57 mmol/mol and was started on gliclazide 40 mg OD. Poor adherence and low physical activity contributed to deterioration in glycaemic control (HbA_{1c} 82 mmol/mol). He independently commenced tirzepatide and ceased gliclazide. His BMI improved from 30.4 to 27.3 kg/m^2, with significant glycaemic improvement and a HbA_{1c} of 55 mmol/mol with no reported adverse effects. He remains under regular follow-up.

In both cases, tirzepatide use was associated with substantial weight loss (BMI reductions from 28.7 to 23 kg/m^2 and 30.4 to 27.3 kg/m^2) and marked HbA_{1c} improvements (from 67 to 32 mmol/mol and from 82 mmol/mol to 55 mmol/mol). Both patients tolerated the therapy well and demonstrated improved adherence compared to their prior sulfonylurea-based regimens.

Conclusions:These cases suggest that tirzepatide may represent a promising therapeutic option in HNF1A-related monogenic diabetes, offering enhanced glycaemic control, weight reduction and improved treatment adherence. While preliminary, these findings support further exploration of incretin-based dual agonist therapy in monogenic diabetes.

Conflicts of interest: nothing to disclose.

Category: Diabetic emergencies

Mind the gap: lost in handover: a systems failure in joint medical-surgical diabetes care

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Background: There is a growing number of older adults with type 1 diabetes mellitus (T1DM) in the UK. They may present unique challenges in acute management, particularly during intercurrent illness.

Case presentation: An 88-year-old woman with T1DM was admitted with a neck of femur fracture (NOF) after a fall and long-lie. She had been previously independent and self-administered insulin. She developed diabetic ketoacidosis (DKA) soon after admission. It was unclear when she last administered her long-acting insulin. She was successfully treated with a fixed-rate intravenous insulin infusion (FRIII) as per local DKA policy and Joint British Diabetes Society guidelines, and the post-take ward round plan appropriately documented switching to a variable-rate intravenous insulin infusion (VRIII) when oral intake resumed. When stabilised she would be taken for NOF repair.

Management and outcome: Over the next 12 hours, she remained in Accident and Emergency and developed multiple hypoglycaemic episodes. This was flagged to her medical and orthopaedic teams, who prescribed high-concentration intravenous dextrose infusions. Despite this, her blood glucose kept dropping.

She became unresponsive (Glasgow Coma Scale [GCS] dropped from 14 to 6), and a medical emergency call was placed. On review she was found to be fluid overloaded, hypoxic, hypoglycaemic and hyperkalaemic (K^+ 6.7 mmol/L). After checking her drug chart, the FRII (and matching fluid regimen) had not been stopped. During these 12 hours, she received 60 units of insulin (her usual background dose was 3 units), and 6 litres of fluid with potassium chloride.

Her acute treatment was complicated, as we had to stabilise her acute medical problems for the NOF surgical repair. She

continued to have hypoglycaemic events despite a VRIII and her neurological function did not recover.

Discussion: There are several essential learning points. The FRIII was continued after biochemical resolution of DKA, and fluids with potassium chloride were prescribed despite rising serum potassium levels. These could have been mitigated by checking the drug chart before adding further prescriptions. These errors are common with electronic prescribing.

Delayed escalation and poor clinical handover compounded her deterioration. This could reflect a potential age or frailty bias that may inadvertently delay aggressive management in older adults.

For joint medical-surgical patients, clearly defined responsibility for insulin and fluids prescriptions is essential to avoid duplication, omission or delay.

Overall, this case highlights the importance of strict adherence to and education surrounding the diabetes emergencies protocol, in all cases, to mitigate both technical and human factors that could cause harm.

Category: Diabetic emergencies

A unique case of diabetic ketoacidosis

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Diabetic ketoacidosis (DKA) is a common diabetic emergency, particularly in patients with type 1 diabetes mellitus (T1DM).

Our patient was a 51-year-old woman who had had T1DM for the previous 43 years. She had a background history of Addison's disease and coeliac disease. She presented to the accident and emergency department having had multiple episodes of diarrhoea and vomiting for two days. This meant that she had not been able to take her regular medications, including steroids.

She was found to have both DKA and an Addisonian crisis. She was started on the DKA protocol and IV hydrocortisone.

Her blood test results revealed acute kidney injury and hyperkalaemia. She was catheterised for fluid management and started on hyperkalaemia treatment. Her DKA resolved, as did her acute kidney injury with appropriate fluid management

She was seen by diabetes inpatient nurses, who provided illness management and diabetes distress leaflets. The patient was referred for a DAFNE refresher course and a follow-up appointment was arranged.

As her symptoms improved she became able to tolerate oral intake of tablets. Her IV hydrocortisone was later switched to hydrocortisone tablet 20/20/10 (doubled) for three days before restarting her usual steroid dose .She was also seen by the endocrine specialist nurse to reinforce sick day rules and given a follow-up appointment.

This case is interesting since the patient has autoimmune polyglandular syndrome type 2. Her acute illness exacerbated both diabetes and Addison's simultaneously .

Category: Diabetic emergencies

Beyond steroid hyperglycaemia: a case of pembrolizumab-induced DKA requiring a new oncology-diabetes Standard Operating Procedure (SOP)

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Background: Immune checkpoint inhibitors (ICIs) have opened new avenues of treatment for cancers that were previously thought to be incurable. Unfortunately, various adverse events have been reported, including new presentation with diabetes mellitus (DM), particularly with programmed-cell-death-protein-1 (PD-1) inhibitors such as pembrolizumab.

Case report: A 67-year-old male, who was being treated for oesophageal cancer with pembrolizumab, had just completed his third cycle of treatment. Prior to the third cycle, he was started on 60mg prednisolone once daily.

He was reviewed in the oncology clinic for blood tests to monitor his condition after completing the third cycle. He was found to have an elevated capillary blood glucose (CBG) of 20.1 mmol/L. He did not have any background of diabetes. It was presumed that the hyperglycaemia was due to the high-dose steroids that he had recently started. He did not have any symptoms of hyperglycaemia so a follow-up appointment was planned for one week later, when his HbA_{1c} could be checked.

The day before his follow-up appointment with Oncology, he began to feel unwell with blurred vision, polydipsia, polyuria and vomiting. His GP discovered that his CBG was 29.1 mmol/L and called an ambulance.

In hospital, he was found to be in diabetic ketoacidosis (DKA) with biochemical markers pH 7.29, glucose >39mmol/L, ketones 6.7 and bicarbonate 15.

He was promptly referred to the diabetes team. They advised that although steroids can induce hyperglycaemia, development of DKA was more likely a problem with insulin production. An immunotherapy-induced DM would be the likely cause.

He was treated successfully as per the Joint British Diabetes Society guidelines with a fixed-rate intravenous insulin infusion (FRIII). He was also started on long-acting insulin in the acute setting.

Due to the prompt diagnosis, he only required a two-day hospital admission. He was taught how to administer insulin, and a continuous glucose monitor was applied. He was discharged on a basal-bolus regimen and followed up on the local Diabetes Virtual Ward.

Conclusion: The onset of ICI-related adverse events can be rapid. They are more likely to occur with combination treatment (more than one immunotherapy, in combination with chemotherapy or addition of steroids).

As the use of ICIs becomes more common, it is imperative that clinicians are aware of ICI-related adverse events. Collaboration between oncology and diabetes teams is essential to create robust SOPs to ensure early intervention. In this case, same-day initiation of long-acting insulin when initially seen in Oncology clinic could have prevented life-threatening DKA.

Category: Diagnosis and management of monogenic diabetes

When T1DM isn't T1DM: diagnosing HNF4A-MODY in everyday clinical practice

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Background: Maturity-onset diabetes of the young (MODY) is a heterogeneous group of monogenic diabetes disorders, accounting for less than 5% of all diabetes cases, which usually manifest before the age of 25 and mostly exhibit an autosomal dominant hereditary pattern. It is frequently misdiagnosed as type 1 diabetes (T1DM), particularly in individuals who are diagnosed in adolescence or early adulthood.

MODY is associated with defective pancreatic islet cell function and impaired insulin production. Fourteen different MODY mutations have been described, with the variants in hepatocyte nuclear factor 1 alpha (HNF1A), glucokinase (GCK) and hepatocyte nuclear factor 4 alpha (HNF4A) accounting for approximately 80–90% of MODY cases. Among these, HNF4A (MODY 1) represents around 5–10% of all MODY. It results from pathogenic variants in the hepatocyte nuclear factor-4 alpha gene, which leads to progressive deterioration in insulin secretion. Individuals with HNF4A MODY demonstrate macrosomia at birth, neonatal hypoglycaemia, vascular complications and low HDL levels as HNF4A is also expressed in the liver. MODY, with the exception of the GCK type (MODY 2), carries a similar rate of complications to T1DM and T2DM. Correct diagnosis has important implications for treatment selection, glycaemic control and cascade family screening.

Case presentation: A 44-year-old woman, diagnosed with T1DM at the age of 19, was managed for years with a basal–bolus insulin regimen. Her family history included genetically confirmed HNF4A-MODY in her mother, sister and three (maternal) nieces. Initial investigations showed preserved insulin secretion (urinary C-peptide:creatinine ratio 0.92; serum C-peptide 500 pmol/L), negative islet cell antibodies and an HbA_{1c} of 80 mmol/mol. Genetic testing confirmed HNF4A-MODY. She transitioned gradually off prandial insulin as gliclazide was introduced and titrated. Her regimen eventually stabilised at gliclazide 80 mg BD and Levemir 10 units nocte, maintaining fasting glucose at 6–8 mmol/L with minimal nocturnal hypoglycaemia. She remained physically active and maintained a BMI of 21 kg/m^2. Co-morbidities, including hypertension and dyslipidaemia, were managed with lifestyle measures and atorvastatin.

Conclusion: This case highlights the importance of considering MODY in adults with atypical diabetes features and a strong family history. Transition from insulin to sulfonylurea therapy can be safe and effective with multidisciplinary support; however, some individuals may continue to require low-dose insulin in combination with sulfonylureas to maintain optimal glycaemic control. Glucagon-like-peptide-1 (GLP-1) agonists have shown similar efficacy to the sulphonylureas. Accurate recognition of monogenic diabetes enables personalised therapy, avoids unnecessary insulin exposure and facilitates appropriate cascade genetic testing, with significant implications for long-term patient and family outcomes in routine clinical practice.

Category: Diagnosis and management of monogenic diabetes

A case of atypical diabetes: diagnostic and clinical challeneges.

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A 35-year-old female diagnosed with gestational diabetes mellitus (GDM) in 2010 was initially treated with diet alone. However, five years post-partum her HbA_{1c} rose to 79mmol/mol. Given her young age at presentation and her low BMI she was treated as type 1 diabetes (T1DM) and given insulin therapy (Novorapid and Levemir). Despite the low insulin doses, a subsequent HbA_{1c} was 27 mmol/mol. She continued on reducing doses of insulin but because she was having recurrent hypoglycaemic events the insulin was discontinued. Testing for diabetes antibodies gave negative results; serum and urine C-peptide levels suggested good endogenous insulin production.

Her clinical presentation was complicated by bilateral hearing loss, vertigo and tinnitus in 2018. Audiometry assessment confirmed bilateral sensorineural hearing loss (SNHL). She reported a family history of hearing issues, notably in her maternal uncle and her mother, who died at the age of 27. She had been diagnosed with diabetes but there was limited information available. The patient's HbA_{1c} rose again in 2020 and a diagnosis of atypical diabetes was considered, especially given her clinical phenotype, family history and absence of insulin dependency. MODY calculator score was 45.5% (showing a 1 in 2.2 chance of having MODY) and subsequent testing revealed a m.3243A>G mitochondrial diabetes variant, linked to MIDD (Maternally Inherited Diabetes and Deafness) and hyponatremia.

Three years later, her HbA_{1c} was again rising. Insulin was initiated once more with Humulin I and Novorapid. Continuous glucose monitoring has been utilised and her glucose control is monitored to avoid hypoglycaemia.

This case highlights the importance of considering genetic factors in atypical diabetes presentations, particularly when combined with relevant clinical features such as hearing loss and a family history of diabetes.

Category: Mental health and diabetes

Embedding psychological screening in type 1 diabetes clinics: a quality improvement project to increase the Diabetes Distress Scale-2 uptake

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Background: Type 1 diabetes (T1DM) requires lifelong self-management and continuous adaptation to daily challenges that can often lead to diabetes distress. This can negatively impact adherence to treatment, glycaemic outcomes and overall wellbeing. Psychological needs are often underassessed in routine diabetes care, partly due to time constraints and the perceived complexity of available tools. A baseline audit in our T1DM clinic identified that psychological assessment was completed in only 30% of consultations (range 16–56%).

Aim: To increase the proportion of people with T1DM receiving psychological assessment using the Diabetes Distress Scale-2 (DDS2), within a district general hospital diabetes service.

Methods: Between August 2024 and August 2025 a series of interventions was implemented following a staff survey to identify key barriers. These included:

- Education sessions highlighting the importance and ease of using DDS-2.
- Integration of a structured T1DM consultation tool for consistent psychological screening.
- Monthly reminders for reception staff to distribute the consultation tool to patients.
- Visual prompts (posters) in clinic areas to reinforce practice change.

Progress was reviewed across ten Plan–Do–Study–Act (PDSA) cycles, with data analysed using statistical process control (SPC) charts.

Results: Following the first PDSA cycle, completion of psychological assessments increased from 30% to 49%. The uptake fluctuated between 30–70% across subsequent cycles, maintaining an improved mean of 45% throughout the final six cycles.

Conclusion: This project demonstrates that embedding simple, low-cost interventions such as DDS2 screening into T1DM clinic workflows can sustainably enhance the recognition and documentation of psychological distress in people with T1DM. Regular staff engagement and visual prompts are effective strategies for integrating holistic assessment into routine diabetes care.

Category: Diagnosis and management of monogenic diabetes

Both sides of the spectrum of HNF4A MODY

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Maturity Onset Diabetes of the Young (MODY) is a rare, autosomal dominant form of diabetes that accounts for approximately 1–5% of all diabetes cases. It is characterised by early onset, typically before the age of 25, a multigenerational family history of diabetes, and a favourable response to oral hypoglycaemic agents. Among the various MODY subtypes, HNF4A accounts for 5% of cases. This can present with neonatal hypoglycaemia and later with MODY. We would like to share this case highlighting this spectrum of presentation.

A 31-year-old woman was referred for monitoring of HNF4A MODY. She had genetic testing following her twin daughters' diagnoses. She had gestational diabetes, which was managed with metformin, and had pregnancy-induced hypertension. She underwent C-section at 35 weeks due to growth restriction. Her twins required prolonged readmission after presenting with hypoglycaemia, requiring IV glucose and diazoxide. In retrospect she also required treatment for hypoglycaemia in the infantile period. Genetic testing confirmed that the patient and her daughters all carry a heterozygous pathogenic mutation in HNF4A, in keeping with congenital hyperinsulinism. There is a family history of diabetes in her mother, who is well controlled on metformin. She also has high BMI and has not yet been tested for HNF4A MODY. Our patient's BMI is 32 kg/m^2 and her post-natal HbA_{1c} at six weeks was normal. Her HbA_{1c} is now 63 mmol/mol 18 months post-delivery. She was initially commenced on low-dose gliclazide with good effect. Subsequently metformin was added because of other features.

Mutations in HNF4A are unusual in that they influence insulin secretion differently across the life course. In foetal and neonatal life, HNF4A mutations lead to increased insulin secretion, resulting in macrosomia and transient neonatal hypoglycaemia due to hyperinsulinism. During childhood, insulin secretion normalises, but in adolescence or early adulthood progressive β-cell dysfunction develops, leading to impaired glucose-stimulated insulin secretion and overt diabetes. This dual-phase presentation reflects the central role of HNF4A as a transcription factor regulating genes essential for pancreatic β-cell function. Over time, the secretory defect progresses, and patients may eventually require insulin therapy.

HNF4A MoDY should be suspected in patients with diabetes and a strong family history of neonatal hypoglycaemia. All babies of pregnancies where either parent has HNF4A should have their blood glucose tested at birth and monitored later to ensure their blood glucose levels are not low.

Category: Diagnosis and management of monogenic diabetes

When genes and autoimmunity collide: a rare case of GCK-MODY co-existing with type 1 diabetes

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A 69-year-old man, who has been managed for many years as having type 1 diabetes (T1DM), has long maintained a consistently stable glycaemic control with a remarkably low insulin dosage. He has been on Levemir 5 units twice daily and Novorapid on a carbohydrate-counting regimen, maintaining HbA_{1c} values in the mid-60 mmol/mol range. Despite his longstanding diabetes, his low insulin requirements and stable blood glucose levels with minimal microvascular complications raised the possibility of an alternative or co-existing diagnosis.

A strong family history revealed a son diagnosed with glucokinase-MODY (GCK-MODY). Hence genetic testing was pursued for our patient, which confirmed the same pathogenic GCK mutation. This finding prompted further evaluation to determine whether his diabetes was monogenic or if there was coexistence with autoimmune or type 2 diabetes (T2DM). Laboratory investigations revealed a random plasma glucose of 21.2 mmol/L, markedly positive diabetes antibodies (GAD 25 kU/L, total antibody screen 123 U/mL) and a suppressed C-peptide < 17 pmol/L, confirming an established T1DM characterized by autoimmune-mediated beta-cell destruction and absolute insulin deficiency, superimposed on GCK-MODY.

This case highlights an uncommon overlap between two distinct aetiologies: monogenic and autoimmune diabetes. While GCK-MODY typically manifests as stable non-progressive hyperglycaemia not requiring insulin therapy, antibody positivity with undetectable C-peptide supports a true insulin-deficient state, consistent with T1DM. Recognition of this dual pathology is essential to inform prognosis, tailor treatment strategies and enable targeted genetic screening for at-risk family members. In this case, management remains unchanged, with a continuation of basal-bolus insulin therapy given complete beta-cell failure due to autoimmunity, along with glycaemic monitoring. However, accurate molecular diagnosis can provide clarity on the family's genetic risk, avoiding misdiagnoses and inappropriate attempts to discontinue insulin.

This case underscores the importance of integrating clinical phenotype, thorough collection of family history during consultation, genetic testing and biochemical markers to accurately investigate atypical diabetes presentations. In clinical

practice, MODY may coexist with more common forms of diabetes, complicating diagnosis and management. Even when treatment remains unchanged, establishing an accurate dual diagnosis is vital for clinical understanding and enhanced personalised care.

Category: Diabetic emergencies

Improving inpatient hypoglycaemia management: a Quality Improvement Project at Doncaster Royal Infirmary

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Background: Hypoglycaemia is a frequent and preventable inpatient complication of diabetes management, particularly among patients treated with insulin or sulfonylureas. Prompt rechecking of blood glucose, adjustment of therapy and prevention of recurrence are vital to patient safety. Despite established guidelines, variations in clinical practice persist. This Quality Improvement Project (QIP) was undertaken at Doncaster Royal Infirmary to evaluate current practice, implement a targeted intervention, and assess its impact through re-audit.

Methods: Two audit cycles were conducted across multiple medical wards and the acute medical unit. The first cycle, from 30 March to 12 April 2025, identified 45 hypoglycaemic events. Three standards were assessed: (1) blood glucose rechecked within 15–20 minutes; (2) insulin or sulfonylurea dose adjusted; and (3) recurrence of hypoglycaemia.

An intervention followed, consisting of educational posters on hypoglycaemia recognition and management displayed in clinical areas, reinforcement of documentation in the electronic system (Nervecentre), and targeted teaching sessions for medical and nursing teams.

The second cycle, from 17 June to 22 July 2025, reviewed 41 hypoglycaemic events across an expanded set of wards. The same standards were reassessed, alongside uptake of Nervecentre documentation.

Results: In the first cycle, only 42% of events had a documented recheck within 15–20 minutes. This improved to 63% in the second cycle. Adjustment of insulin or sulfonylurea therapy remained low, with improvement from 20% to 27%. Recurrence of hypoglycaemia showed a modest reduction, from 33% in the first cycle to 29% in the second. Uptake of Nervecentre documentation improved, reflecting greater staff awareness of recording and monitoring processes.

Conclusion: This QIP demonstrated that simple, low-cost interventions such as poster campaigns and targeted teaching can positively influence inpatient hypoglycaemia management. Significant improvement was observed in timely blood glucose rechecking, though medication adjustment and recurrence rates remained areas of concern. Future recommendations include ongoing education for medical and nursing teams, integration of electronic alerts within Nervecentre to prompt timely action, and continuation of audit cycles to ensure sustained improvement. Embedding these measures will strengthen patient safety and align practice with national standards for hypoglycaemia management.

Category: Obesity management in diabetes

Weight gain in patients with type 1 diabetes on insulin pump therapy: association, challenges and management options

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Background: Weight gain is a recognised and often distressing challenge in people with type 1 diabetes mellitus (T1DM), particularly after transitioning from multiple daily injections (MDI) to continuous subcutaneous insulin infusion (CSII). While insulin pump therapy markedly improves glycaemic control and quality of life, it may also be associated with progressive weight gain. Managing obesity in individuals with T1DM remains challenging, with few effective or licensed pharmacological options available.

Case presentation: We report the case of a 36-year-old man with T1DM diagnosed at the age of 10. He experienced a substantial weight gain of 35 kg (from 86 kg to 121 kg) over five years following initiation of insulin pump therapy (Medtronic), which was commenced for persistently poor glycaemic control. His height was 167 cm (5'6"), corresponding to a body mass index (BMI) of 43.9 kg/m^2. Despite the weight increase, his glycaemic control was excellent, with a time-in-range (TIR) of 86%, time below range of 1%, and HbA_{1c} of 44 mmol/mol. He reported no change in dietary pattern, caloric intake or physical activity. He worked full-time as a delivery driver, with consistent daily exertion. He had background diabetic retinopathy, normal urine albumin–creatinine ratio and renal function, and no neuropathy. Dyslipidaemia was identified and managed with atorvastatin; his blood pressure remained normal. Despite lifestyle adjustments and dietetic input, his weight continued to rise. Concerned that his rapid weight gain was linked to pump use, he queried whether discontinuing the pump or intermittently using CSII might promote weight loss. He had been declined glucagon-like peptide-1 receptor agonist (GLP-1RA) therapy by private providers because of his T1DM diagnosis and was not keen on bariatric surgery. He was counselled against discontinuing CSII and referred to a Tier 3 weight management service, where he was commenced on tirzepatide (Mounjaro) following multidisciplinary discussion regarding potential benefits and risks, including ketosis.

Discussion: Weight gain after CSII initiation is well documented in adults with T1DM, though the extent varies. Long-term studies suggest this reflects metabolic adaptation rather than the device itself, with both CSII and MDI users demonstrating gradual increases in body weight. Younger age and higher baseline HbA_{1c} predict greater weight gain, and the magnitude observed in this case suggests individual susceptibility. Pharmacological options are limited; GLP-1RAs have shown modest but consistent weight reduction and insulin-sparing effects in randomised and real-world T1DM cohorts, though their use remains off-label.

Conclusion: This case illustrates marked weight gain temporally associated with CSII in T1DM despite excellent glycaemic control. While CSII remains the optimal method for achieving glucose targets, proactive and individualised weight management strategies are essential. Off-label GLP-1RA therapy may represent a viable adjunct for selected individuals under specialist supervision, pending further evidence and regulatory approval.

Category: Diabetes technology in people with T1DM

Starting insulin pump therapy: is the diagnosis correct?

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Background: Accurate classification of diabetes mellitus is fundamental to structuring patient education, guiding optimal therapy and informing prognosis. Nonetheless, misclassification between type 1 and type 2 diabetes (T1DM and T2DM) remains frequent in clinical practice, especially at initial presentation. Diagnostic ambiguity in diabetes classification is increasingly recognized due to overlapping phenotypic features between types.

Case report: A 66-year-old female, diagnosed with T1DM at the age of 40, was treated with a basal and bolus insulin regime before commencing insulin pump therapy using the Medtronic 780G system five years previously. On review in clinic, she was noted to have a BMI of 29 kg/m^2 and she confirmed that she was treated with oral medication for one year after her diagnosis before commencing insulin therapy. Diabetes-related antibodies returned negative, and plasma C-peptide level was 558 pmol/L, consistent with adequate endogenous insulin production. She was re-classified as having T2DM and this was explained to her. The next step was to broach the subject of insulin pump withdrawal, which was handled sensitively. Based on her basal insulin requirements, she was commenced on insulin Toujeo and metformin, with a plan to add an SGLT2i, one week later. Initially, she was somewhat reluctant about the idea but it went ahead as planned. She was closely supported by the diabetes specialist nurse team in making this transition. On follow-up, she was very pleased with having clarity of diagnosis. Her basal insulin requirement had fallen by two-thirds and she had lost two stones in weight over six months. Her HbA_{1c} levels were satisfactory.

Discussion: Youth-onset T2DM is becoming more prevalent, while autoimmune diabetes may present later in life with a gradual course, as seen in latent autoimmune diabetes in adults (LADA), challenging traditional distinctions based on age and body habitus. Additionally, ketone-prone diabetes can initially mimic T1DM. However, many of these patients are autoantibody-negative, retain β-cell function and later follow a clinical course consistent with T2DM. Current guidelines emphasize the importance of combining clinical assessment with measurement of islet autoantibodies and C-peptide levels to improve diagnostic accuracy and to re-evaluate the diagnosis when the clinical course is atypical.

Conclusion: Insulin pump therapy is the gold standard treatment for people living with T1DM. For people commencing insulin pump therapy, re-visiting the diagnosis in the right clinical context may well be rewarding, as in this case. Precise aetiological classification of diabetes has direct clinical implications as it allows clinicians to frame the content of structured patient education, informs risk stratification, and determines the selection of glucose-lowering therapies and surveillance for complications.

Category: Diabetic emergencies

The enigma of a normal HbA_{1c} in hyperosmolar hyperglycaemic state: unmasking dapsone interference

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Background: Hyperosmolar Hyperglycaemic State (HHS) is a rare but serious metabolic emergency defined by extreme hyperglycaemia, hyperosmolarity and dehydration, usually occurring in patients with type 2 diabetes mellitus (T2DM). Diagnosis typically relies on markedly elevated plasma glucose levels in conjunction with a raised glycated haemoglobin (HbA_{1c}), which reflects chronic hyperglycaemia. However, certain medications and haematological conditions can distort HbA_{1c} interpretation, leading to diagnostic uncertainty. Dapsone, a sulfone antibiotic used for dermatological conditions such as bullous pemphigoid, can cause haemolysis and shorten erythrocyte lifespan, producing spuriously low HbA_{1c} results that underestimate glycaemic exposure.

Case presentation: A 55-year-old male with no prior diagnosis of diabetes was admitted with confusion, polyuria and dehydration following a lower respiratory tract infection. On arrival, his capillary blood glucose was 35 mmol/L, and laboratory findings confirmed HHS. Surprisingly, his HbA_{1c} was only 44 mmol/mol (6.2%), inconsistent with the severity of hyperglycaemia observed. He was treated with the standard HHS protocol involving intravenous fluids, insulin infusion and careful electrolyte correction, leading to gradual metabolic recovery.

Review of his medical history revealed a background of bullous pemphigoid managed with dapsone 100 mg daily for two years. There was no evidence of haemoglobinopathy or chronic anaemia. The discordance between his acute presentation and relatively normal HbA_{1c} was attributed to dapsone-induced haemolysis causing falsely low HbA_{1c} values. After stabilization, he was discharged on oral hypoglycaemic therapy with metformin and gliclazide. He was also provided with a continuous glucose monitoring (CGM) sensor to allow accurate assessment of glycaemic trends, as HbA_{1c} was deemed unreliable for follow-up.

Discussion: This case underscores the importance of recognising drug-related interference with HbA_{1c} interpretation. Dapsone causes oxidative stress and low-grade haemolysis, leading to premature red-cell destruction and reduced HbA_{1c} independent of glycaemic status. In such cases, clinicians should interpret HbA_{1c} with caution and use alternative monitoring tools. CGM provides a reliable and dynamic evaluation of glucose levels and an estimated HbA_{1c} (Glucose Management Indicator) unaffected by erythrocyte lifespan, offering an effective solution for ongoing monitoring when standard HbA_{1c} is misleading.

Conclusion: A normal HbA_{1c} does not exclude diabetes in patients treated with dapsone. Awareness of this interference is crucial to avoid diagnostic pitfalls and prevent life-threatening complications such as HHS. Incorporating CGM in such patients ensures accurate assessment of glycaemic control and supports safer diabetes management.

Category: Diabetes technology in people with T1DM

Hypoglycaemia associated with air travel in people using hybrid closed-loop insulin delivery systems

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A 55-year-old woman using Omnipod 5 (OP5) with Dexcom G6 since April 2024 because of frequent episodes of disabling hypoglycaemia was seen in clinic. These episodes stopped with OP5 but she reported two episodes of unexpected hypoglycaemia during two separate flights.

In February 2025, whilst on board a flight to Italy, she developed hypoglycaemia within an hour of take-off. Blood glucose did not increase with standard treatment and the plane was diverted to land. She had not given any bolus prior to the flight. Continuous glucose monitoring (CGM) data showed persistent glucose less than 3mmol/l for 30 minutes.

Two months earlier, she had a similar episode whilst in flight and was met at the plane door with a wheelchair. Her CGM data for the flight period again showed persistent glucose below 3mmol/l despite having the pump in activity mode and no insulin bolus within several hours of the flight. She started avoiding flying after these episodes.

Following discussion in the MDT, a further five similar cases were identified:

Case 2 was a 51-year-old woman on OP5 who reported recurrent hypoglycaemia when travelling to/from New York for work, usually on take-off. One episode required multiple treatments, and emergency landing was narrowly avoided. Again, diabetes data from the event show prolonged hypoglycaemia (<4mmol/l for 45 minutes) despite pump suspension and the last bolus four hours before. Other cases reporting similar episodes were all using OP5.

Discussion: The effects of ambient pressure changes on insulin pump delivery have been described by Garden et al. In vitro, in a hypobaric chamber, in flight simulation conditions, full cartridges over-delivered 0.60 units of insulin during a 20 minute ascent and under-delivered by 0.51 units during descent, compared with ground level performance. During emergency landing simulation, 5.6 u of excess insulin were delivered.

The Omnipod website states that Omnipod is safe to use at atmospheric pressures typically found in aeroplane cabins during a flight but that changes in atmospheric pressure can affect the pod's insulin delivery leading to possible hypoglycaemia. Since these cases, this possibility is routinely discussed at OP5 starts at our Trust.

It remains to be seen whether tubeless pumps are more susceptible to changes in atmospheric pressure than tubed systems, and whether other factors like low HbA_{1c} or low insulin total dose make certain people more susceptible to hypoglycaemia.

Category: Diabetic emergencies

A fragile balance: linagliptin-associated bullous pemphigoid in an elderly diabetic patient

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Background:Bullous Pemphigoid (BP) is a rare autoimmune blistering disorder of the skin characterised by subepidermal blisters and erythematous plaques. Increasing evidence links Dipeptidyl Peptidase-4 (DPP-4) inhibitors—commonly used in the management of type 2 diabetes mellitus (T2DM)—to drug-induced BP. Among gliptins, linagliptin has been implicated in several reports, particularly affecting elderly patients with multiple co-morbidities. Early recognition of this association is crucial, as delayed diagnosis can lead to significant morbidity.

Case presentation: An 80-year-old woman with T2DM, advanced dementia, chronic kidney disease (stage 3), hypertension and asthma presented with widespread superficial skin ulcers under both breasts, and on the lateral hips and right thigh. Lesions initially developed after a respite care stay in March 2025 and were managed unsuccessfully as infected ulcers due to Staphylococcus aureus. Despite multiple antibiotic courses, erosions worsened, prompting dermatology referral. At that time, her medications included linagliptin 5 mg once daily (initiated recently prior to the skin lesions), metformin 500 mg nightly and porcine isophane insulin 14 units each morning.

Skin biopsy and direct immunofluorescence demonstrated subepidermal blistering with linear IgG and C3 deposition along the basement membrane—findings consistent with bullous pemphigoid. Linagliptin was immediately discontinued due to the strong temporal association.

Management and outcome: The patient was started on oral prednisolone 40 mg daily (tapered by 5 mg weekly) and doxycycline 100 mg twice daily for its anti-inflammatory benefits. Topical Dermovate ointment, emollients and non-adherent dressings were used for local care. Diabetes management was optimised through close collaboration with the diabetes specialist nurse, increasing the insulin dose to 18 units to offset steroid-induced hyperglycaemia. Regular monitoring and supportive care led to significant improvement, with no new blisters forming and gradual healing of existing lesions.

Discussion: This case highlights the potential for DPP-4 inhibitors to trigger bullous pemphigoid, a complication that may be under-recognised in clinical practice. Elderly patients with T2DM and multiple co-morbidities are particularly vulnerable. Awareness of drug-induced causes, prompt withdrawal of the suspected agent and multidisciplinary management—including dermatology, diabetes and geriatric teams—are key to positive outcomes.

Conclusion: Clinicians should consider linagliptin-induced bullous pemphigoid in elderly diabetic patients who present with unexplained erosive or blistering rashes. Early recognition and drug discontinuation are essential to reduce morbidity and optimise both dermatological and glycaemic outcomes.

Category: Obesity management in diabetes

Insulin withdrawal and weight loss: a case for phenotypic reassessment in patients with multiple/ambiguous causes of diabetes

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Background: Patients with transfusion-dependent β-thalassaemia (TDT) require lifelong transfusions; cumulative iron overload causes secondary haemosiderosis, promoting progressive β-cell dysfunction and insulin resistance. We report a case in which phenotypic reassessment after two decades on basal–bolus insulin regimen allowed safe withdrawal of basal insulin and initiation of weight-targeted therapies, resulting in substantial weight loss and improved glycaemic control.

Case report: A woman with TDT developed diabetes at the age of 13 years and was commenced on basal-bolus insulin. At age 29, she was first seen in our department. On presumption of pancreatic siderosis and ongoing insulin deficiency, she was provided with structured education around insulin dose adjustment. She continued daily insulin glargine 30–36 units, and NovoRapid 6-16 units with meals. Fructosamine was 313 µmol/L (NR 205 – 285 µmol/L). At the age of 35, her basal insulin was changed to insulin degludec. Fructosamine levels subsequently ranged from 294 to 420 µmol/L. At the age of 43, she reported missing three doses of insulin degludec while travelling without major hyperglycaemia and had gained weight (BMI 28.6 kg/m^2). These factors, and a strong family history of type 2 diabetes (T2DM), prompted phenotypic reassessment. Stimulated C-peptide measured 554 pmol/L, consistent with endogenous insulin secretion. Basal insulin was de-escalated, and dapagliflozin started. She was instructed to resume basal insulin only if glucose levels rose.

By the age of 44 years, CGM off basal insulin showed time-in-range (TIR) 70% with no hypoglycaemia and a 4.6 kg weight loss (BMI 26.9 kg/m^2). Nocturnal hyperglycaemia persisted, attributed to marked evening appetite related to quetiapine. Tirzepatide 2.5mg weekly was commenced and mealtime NovoRapid ratios progressively reduced. By 45 years, she had been off regular basal insulin for more than12 months; therapy comprised dapagliflozin 10 mg daily, tirzepatide 2.5 mg weekly and occasional low-dose mealtime NovoRapid. Her peak reported weight loss was ~15 kg, with a BMI of 24.1kg/m^2. CGM TIR was 88%, with an estimated HbA_{1c} 51 mmol/mol. There was no severe hypoglycaemia or DKA. Recent fructosamine was normal (284 µmol/L).

Discussion: Phenotyping with stimulated C-peptide revealed substantial residual β-cell function and justified safe basal insulin de-escalation. This, in addition to a staged, obesity-centred approach combining SGLT2 inhibitors and tirzepatide, produced appetite suppression, progressive and clinically meaningful weight loss and improved CGM metrics while markedly reducing insulin exposure. This highlights consideration of phenotypic reassessment in patients with multiple or ambiguous causes of diabetes mellitus, such as those with diabetes presumed secondary to iron overload.

Category: Diabetic emergencies

Severe diabetic bone disease in a young adult with type 1 diabetes mellitus and hypoglycaemia unawareness

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A 34-year-old man with poorly controlled type 1 diabetes mellitus (T1DM) was admitted with acute behavioural disturbance and suspected tonic-clonic seizures. CT imaging revealed multiple pathological fractures including comminuted fractures of the left acetabulum and greater trochanter, right iliac wing, right inferior pubic ramus, S1 vertebral body and L5 transverse process.

Review of his FreeStyle Libre data showed a significant time-below-range of 20% (over the preceding 90 days), with recurrent postprandial hypoglycaemia, overcorrection and hypoglycaemic unawareness. His latest HbA_{1c} was 34 mmol/mol, previously 133 mmol/mol in 2022. Despite low glucose alarms being set at 5.1 mmol/L, he failed to notice his hypoglycaemia alarms prior to admission, likely due to headphone use and silencing the alarms overnight.

His medical history included T1DM treated with insulin glargine and aspart, complicated by gustatory sweating, diabetic retinopathy, grade A3 albuminuria, hypertension and hypercholesterolaemia. One year earlier, he sustained a left tibial plateau fracture following a road traffic accident, requiring surgical fixation and a prolonged hospital admission.

A dual-energy X-ray absorptiometry (DEXA) scan was performed, revealing significantly reduced bone mineral density (BMD) with Z-scores of −4.8 in the lumbar spine (L2–L4) and −2.3 in the total mean neck of femur. Evaluation for secondary causes of osteoporosis identified low dietary calcium intake, vitamin D insufficiency (38.2 nmol/L), five months of immobility following a previous fracture and mildly reduced free testosterone (0.204 nmol/L) in the context of the acute inpatient admission and opiate analgesia (to be repeated once well). However, poorly controlled T1DM was considered the predominant risk factor.

Diabetic bone disease is likely mediated through impaired osteoblast differentiation and function, altered osteocyte signalling, and the effects of advanced glycation end products on the bone matrix. Chronic complications of diabetes, particularly cardiovascular and renal disease, can further increase fracture risk, while recurrent hypoglycaemia elevates the risk of falls.

Management was multidisciplinary. He underwent surgical fixation of his fractures and received calcium and vitamin D supplementation, with plans for subsequent anabolic therapy with teriparatide given his extremely low BMD, fracture history and young age. From a diabetes perspective, his insulin-to-carbohydrate ratios and sensitivity factors were adjusted, education on insulin stacking was carried out, and his FreeStyle Libre was upgraded to Dexcom G7.

This case highlights the profound impact of poorly controlled diabetes and related hypoglycaemia-induced traumatic seizure activity on bone health. Clinicians should remain vigilant for osteoporosis and consider early bone health assessment in patients with long-standing or poorly controlled T1DM.

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 occurs, 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; fluids 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 ensures 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 present two cases of hypertriglyceridemia which presented at the same time but with different clinical features. There is no national guideline on the emergency management of clinically significant hypertriglyceridemia and we managed these with the local guidelines and input from the 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 (ED) 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 the elevated amylase. The likely precipitating factor appeared to be either drug-induced or 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 for triglyceride (TG) lowering, which came down to 18.8 mmol/L by the next day.

Case 2: A 29-year-old male patient, a non-smoker and not an alcoholic, presented to his GP with osmotic symptoms and feeling lethargic and generally unwell. The GP did blood tests and found that the patient had TG levels of 58 mmol/L. He called the hospital on-call and was asked to send the patient to the ED. He was started on an insulin infusion and his TG came down to 17 mmol/L the following day. The patient had no family history of cardiovascular or cerebrovascular disease. His HbA_{1c} was 56 (he was a newly diagnosed diabetic on this admission), cholesterol 10.6 and LDL 2.4.

Given the improved triglyceride levels in both cases, insulin infusion was weaned off 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 to below 5 mmol/L for ongoing cardiovascular risk reduction. After hospital discharge advice was given on measuring fasting lipid profile and they were referred to the lipid clinic.

Lipoprotein lipase is the primary enzyme of TG lipolysis. It 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 results 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 increase 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 (T1DM from T2DM), particularly in adults who present with hyperglycaemia and overlapping clinical features. Traditional biomarkers such as islet autoantibodies have limitations, including false positives/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 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 in 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 T2DM or MODY).

Results: Of 17 patients reviewed over a 6-week period who were initially treated as T1DM, 6 (35%) were confirmed as having T1DM, 10 (58%) as T2DM, and 1 (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 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 asymmetric 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 glycaemic control and rehabilitation.

Case presentation: A 65-year-old male, diagnosed with type 2 diabetes mellitus (T2DM) in early 2023, presented with bilateral lower-limb neuropathic pain and weakness, more pronounced on the left side. 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 HbA1c 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 further to 37 mmol/mol by 2025. As his sugars stabilised, 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 glycaemic correction, highlights the complexity of managing diabetic neuropathies. A multidisciplinary approach including glycaemic optimisation, 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 got 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 to 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.

T1DM 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 ageism: is this a problem?

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NICE TA943 (2023) recommends hybrid closed-loop (HCL) therapy in type 1 diabetes (T1DM), irrespective of the patient's age. Older adults are often underrepresented in clinical trials and there are scant data in this group. Possible barriers include frailty, cognition and ability to use new technology. This audit assessed whether adults 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 $\geq$4, CGM time <4 mmol/L >7% or time <3 mmol/L >1%) and HbA_{1c} threshold $\geq$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 were eligible but none were 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, and absence of formal pump offer/ discussion for pump eligibility despite a clear qualifying hypoglycaemia history.
- HbA_{1c} $\geq$69 mmol/mol: 33 eligible, none 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 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

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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 hypoglycaemia, 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, ultrasound evidence of NAFLD. The thyroid profile was consistent with hypothyroidism

adequately treated. 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 her 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. This resulted in significant weight loss and improved glycaemic control (HbA_{1c} 53 mmol/mol), and permitted insulin discontinuation. Persistent postprandial hyperglycaemia prompted a gastric bypass in 2024, following which she achieved a total weight loss of 5.5 stone (≈35 kg) and normalisation of glycaemia (HbA_{1c} 37 mmol/mol) without pharmacotherapy.

By late 2024, the patient reported nocturnal and exertional hypoglycaemia, 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 hypoglycaemia reflects heightened post-prandial insulin responsiveness in the remission phase. Long-term monitoring and nutritional guidance remain crucial to maintain stability and prevent hypoglycaemia.

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 diabetes clinic. He was diagnosed at the age of 20 in Pakistan after presenting with weight loss, polydipsia, polyuria and HbA_{1c} of 64mM/M. Treatment was initiated with gliclazide 60mg OD but subsequently stopped due to hypoglycaemia. In the UK metformin was started and gliclazide 80mg restarted but again 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.

Bloods taken in clinic showed 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 glucose levels had been in the teens but he had not felt unwell.

His 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 the 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.