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Abstract ID: 5

Turning the T1DE: MDT management of a dangerous condition

van Heeswijk I
Royal Derby Hospital

Background: Described in a 2017 BBC documentary as "The world's most dangerous eating disorder", type 1 diabetes (T1DM) with disordered eating (T1DE, sometimes referred to as diabulimia) is a disorder in which patients intentionally restrict insulin in order to achieve weight loss. It is associated with high morbidity and mortality, due to diabetic ketoacidosis (DKA) and long-term complications of elevated blood glucose levels. Patients require intensive multidisciplinary team (MDT) support and psychological input to manage this often-fatal condition.
Case report: A 29-year-old patient was referred to the diabetes pump clinic. She had a 4-year history of T1DM and an HbA_{1c} of 135mmol/mol. On average she took insulin degludec three times a week but she never took boluses due to her severe anxiety about weight gain. She had had multiple episodes of diabetic ketoacidosis and suffered with lethargy, osmotic symptoms and recurrent infections. Initially diagnosed with gestational diabetes during her first pregnancy, the patient enjoyed the weight loss she experienced when insulin was discontinued after delivery. Five months postpartum, she was re-diagnosed with T1DM due to persistent hyperglycaemia and ketosis. Having come to associate insulin with weight gain, the patient continued to restrict her insulin intake, stating she would be "6lb lighter by the morning" if she missed a dose. Decisions to inject were influenced by her weight on a particular day. This situation was exacerbated by a relationship breakdown and social services involvement with her children, triggering feelings of self-loathing and low self-worth in the patient.

She was initiated on a Medtronic 780g insulin pump and a Freestyle libre 2 glucose monitoring system, with half of her daily basal requirements given as insulin degludec due to safety concerns. The risks of her behaviour were clearly explained in an honest but non-judgmental manner. She received intensive input from the MDT and underwent cognitive behavioural therapy through the eating disorders team.

Three months later, the patient is consistently receiving basal insulin and is inconsistently bolusing. Her HbA_{1c} is 101 mmol/mol and she has had no further episodes of DKA. She unfortunately was hospitalised due to an overdose following loss of custody of her children but is now motivated to improve her health and regain custody.
Conclusion: : T1DE is a difficult and dangerous condition to manage and it necessitates intensive support from both mental health and diabetes teams. Derby does not yet have an integrated T1DE service but pilots at other centres have given positive outcomes, indicating that these services should be made more widespread nationwide.

Abstract ID: 6

Utilising an advanced hybrid closed-loop system to improve glycaemic control in a patient with suboptimally controlled T1DM

Parez N, Jones O, Ali C
University Hospitals Sussex NHS Foundation Trust

Introduction: Hybrid closed-loop insulin delivery systems use an algorithm to adjust continuous subcutaneous insulin infusions according to data from continuous glucose monitoring. The algorithms adjust, maintain or stop insulin delivery according to: glucose readings and their rate of change; previous insulin delivery; and information given by the user about carbohydrate intake, exercise and sleep. NHS England has undertaken a nationwide pilot to assess the efficacy of hybrid closed-loop systems in individuals with type 1 diabetes (T1DM) who are already on a pump with an HbA_{1c} >69mmol/mol.
Case history: A 39-year-old male had T1DM diagnosed in 2011. Prior to this diagnosis he had been physically active, participating in many martial arts. He would fast during Ramadan.

His physical activity and ability to fast were significantly affected due to challenges with glycaemic control. He had two severe hypoglycaemic episodes in a year, which led to a fear of hypoglycaemia and a consequent tendency to hyperglycaemia during work and exercise.
Management: In 2015, he self-funded the Freestyle Libre glucose monitoring system while on a basal-bolus insulin regime. He met NICE criteria for insulin pump therapy and was started on an Omnipod in 2017, six years following diagnosis. By using more tailored insulin delivery he was able to fast for a few days during Ramadan. However, glycaemic control demonstrated little improvement, with his HbA_{1c} remaining in the low 70s (mmol/mol) for many years.

In 2021 he was commenced on a Control IQ hybrid closed-loop system using a Tandem t:slim insulin pump and a Dexcom G6 as part of the NHS England pilot. He reported an improved sense of ability to manage his diabetes, reflected in his glycaemic control (HbA_{1c} 54nmol/mol; time in target glucose range: 73% versus 43% prior to hybrid closed-loop). He was able to fast for the whole month of Ramadan. We are still working on managing post-exercise dysglycaemia (despite using the algorithm's exercise mode), and preventing hyperglycaemia after take-aways.
Discussion: This case illustrates improvement in glycaemic control in a patient with T1DM using a hybrid closed-loop system. The system allowed him to do activities that he had been unable to complete since being diagnosed with T1DM such as fasting during Ramadan.

This case also shows that people on hybrid-closed loop systems still need ongoing advice and support to manage lifestyle activities such as diet and exercise.

Abstract ID: 8

A rare case of pembrolizumab-induced diabetic ketoacidosis

Lolomari SJ, Thayyil S, Kong MF
University Hospitals of Leicester, UK

Introduction: The programmed cell death-1(PD-1) inhibitor, pembrolizumab, is one of the widely used immune checkpoint inhibitors. it is associated with various immune-related adverse events (irAE) including endocrinopathies like thyroiditis and hypophysitis, and with autoimmune diabetes. Pembrolizumab-induced diabetic ketoacidosis (DKA) is a rare but life-threatening irAE, reported in only 0.1% of the patients. Here we present a case of DKA which occurred in a pembrolizumab-treated patient after three cycles of treatment.
Case presentation: A 75-year-old man with malignant melanoma was started on palliative immunotherapy with pembrolizumab in January 2022. He tolerated pembrolizumab therapy during the first three cycles

but developed osmotic symptoms and unintentional weight loss almost three weeks after the third treatment cycle. During the pre-assessment clinic review for the fourth cycle he was diagnosed with DKA, with pH of 7.2, blood glucose of 26 mmol/L and ketones of 4 mmol/L. He responded to treatment for DKA and was started on a basal-bolus insulin regimen. His HbA_{1c} was 9.3% (78 mmol/mol) and the anti-glutamic acid decarboxylase (GAD) antibody came back positive (>2000 IU/ml).
Discussion: DKA is the most common first presentation in checkpoint inhibitor-induced type 1 diabetes (T1DM). Patients treated with checkpoint inhibitors should be informed about the potential risk of T1DM. Risk stratification and monitoring of glycaemic control should be performed before each treatment cycle. IrAE can develop at any time during or after immunotherapy. In contrast to other irAE, immunosuppressive treatment with steroids or infliximab is not effective in these patients.

Abstract ID: 9

Hidden life-threatening complication of Cushing's syndrome identified by blood gas analysis

Zahid H,[1] Whitelaw BC,[2] Mustafa OG,[2] Taylor DR,[2] Iqbal M[1]
1. Maidstone General Hospital, Maidstone and Tunbridge Wells NHS Trust;
2. King's College Hospital NHS Foundation Trust

Background: Cushing's syndrome is caused by excess cortisol, leading to a wide range of clinical features. Associated metabolic consequences include increased insulin resistance, increased hepatic gluconeogenesis and a tendency towards a prothrombotic state.[1]

Diabetic ketoacidosis (DKA) is a life-threatening acute metabolic complication of all forms of diabetes mellitus.[2] DKA as an initial presentation of Cushing's syndrome is rare, with only a handful of cases reported in the literature.[3-4] We describe the clinical lessons learnt from a patient admitted with clinical and biochemical features of Cushing's who developed this acute but insidious life-threatening metabolic complication.
Case report: A 55-year-old woman presented to the emergency department (ED) with symptoms and signs suggestive of Cushing's syndrome. The initial biochemical tests strongly supported this diagnosis (random serum cortisol at 22:00 hours was 1702 nmol/L and her ACTH was elevated at 218 pg/ml). Despite optimal initial supportive management, her clinical condition deteriorated rapidly, leading to re-evaluation of the clinical and biochemical parameters.

A careful analysis of arterial blood gases revealed a complex compensated mixed acid-base disorder (pH 7.42, PaCO2 2.43, PO2 15.0, bicarbonate [HCO3] 12.0, Na+ 138, K+ 2.2, Cl- 98, lactate 2.3, anion gap 30). Cushing's syndrome generally causes metabolic alkalosis but this patient had a predominant severe high anion gap metabolic acidosis, with compensatory respiratory alkalosis and a minor metabolic alkalosis. The serum lactate was not significantly raised. Renal function was normal. Blood glucose was 9.5 mmol/L, and there was no clinical evidence of poisoning or a toxicological cause.

The patient was tested for capillary ketones, which were significantly elevated at 6.6mmol/L. A diagnosis of severe euglycaemic diabetic ketoacidosis was made (euDKA). It was noted that the patient was on SGLT2i therapy (empagliflozin) for type 2 diabetes (t2DM). The patient was treated according to the conventional DKA protocol. Venous blood gas analysis following resolution of the euDKA demonstrated metabolic alkalosis, which was consistent with severe Cushing's syndrome (pH 7.51, pCO2 5.38, pO2 8.90, HCO3 32.2, Na+ 140, K+ 3.6, Cl- 104, lactate 1.2, anion gap 8). Further investigations confirmed the diagnosis of pituitary-dependant Cushing's disease. The patient was commenced on metyrapone to control the hypercortisolemia and she proceeded to pituitary surgery. She is now well and independent.
Conclusion: Patients with Cushing's syndrome generally have either normal acid-base status or a metabolic alkalosis. The presence of severe metabolic acidosis in a patient with Cushing's syndrome indicated an additional pathological process, later identified as severe euglycaemic diabetic ketoacidosis. This only became evident when the blood gas analysis showed a complex mixed acid-base disorder. Uncommonly, patients with DKA present with plasma glucose level below 11mmol/L, which is defined as euDKA.[5]

This is a well-recognised adverse effect of treatment with sodium-glucose co-transporter 2 (SGLT2) inhibitors.[6] It requires identification and treatment as a medical emergency in a similar way to conventional DKA.[7] This case also provides an opportunity to discuss potential mechanisms by which the combination of cortisol excess and SGLT2i therapy facilitates the development of EuDKA.

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Abstract ID: 12

The effects of diabetes technology on diabetes gastroparesis and cost implications of therapeutic options

Virgo E, Guy E, Joseph S
Department of Diabetes and Endocrinology, East Kent Hospitals Universities Foundation Trust, Kent

Abstract: Mrs KW, 42-year-old mother of three, was diagnosed with type 1 diabetes (T1DM) at the age of 20. She had multiple admissions with recurrent intractable vomiting and with a presumptive diagnosis of gastroparesis: a radio-isotope gastric emptying study revealed a gastric delay of 13 hours. In view of her impaired quality of life, referral for a gastric pacemaker insertion was made. The high cost for this procedure (up to £19 000) and its limited availability on the NHS indicated that access to this procedure was not possible within a reasonable time frame..

Initial management of her gastroparesis revolved around optimizing glycaemia control, avoiding foods that might provoke symptoms and consuming smaller, frequent meals. Unfortunately, these measures had limited success. She suffered from severe distress due to poorly controlled glycaemic levels despite her best efforts, compounded by the significant social and domestic pressures of being an only parent. She had numerous contacts and communications with healthcare providers over the years.

She would be admitted to hospital every 2 to 6 weeks with diabetes ketoacidosis and hypoglycaemia brought on by her persistent vomiting. These episodes were often managed by using a combination of fixed- and variable-rate intravenous insulin infusion regimes, antiemetics and pro-motility agents.

She had 67 hospital admissions between 2015 and 2022, with 256

bed days. We were of the view at this stage that the introduction of diabetes technology would help to break the cycle of vomiting-induced metabolic disturbance and hence the need for recurrent admissions. A hybrid closed-loop insulin pump (Medtrum Nano CGM) with a low glucose suspend facility was commenced after reinforcing the patient's carbohydrate counting skills.

The commencement of pump technology led to a dramatic improvement in the patient's quality of life. She had only three documented admissions between April and December 2022 and these were for manageable mild hyperglycaemic episodes requiring a 0-1 day length of stay. Her HbA_{1c} improved from 98 to 72 mmol/mol, with less variability in her glucose profile. She is now planned to have a gastric perioral myotomy (G-POEM), with an estimated cost of £4 000.

This case illustrates the challenges of managing T1DM with complicating diabetic gastropathy. It shows how the potential benefit of early introduction of diabetes technology can be a worthwhile investment to improve metabolic control, quality of life, reduce health care costs and avoid frequent hospital admissions.

Abstract ID: 13

Partial cannula blockage in CSII presenting as severe DKA and OOH cardiac arrest: a case report

Ahmed N

Arrowe Park Hospital, Birkenhead, Wirral

A 49-year-old woman with type 1 diabetes (T1DM) who had been on a Medtronic 640G insulin pump since 2015 was admitted to the Countess of Chester Hospital, Chester with an out-of-hospital (OOH) cardiac arrest in January 2022. The patient was found to be in severe diabetic ketoacidosis (DKA) on admission with a pH of 6.8, ketones high and BMs unrecordably high.

Initial diabetes pump data review on the intensive treatment unit (ITU) revealed that the pump was 14 months old, in good condition, with pump download showing insulin doses as 43% basal and 57% bolus. This is a reasonable percentage split. The bolus dose will be higher on this download as the patient gave multiple correction doses with average glucose 13.3 mmol over 14 days. This value will be elevated due to high readings over the last 36 hours of data.

There were no occlusion alarms registered on the pump and no other alarms apart from a low reservoir alarm on 2nd January 2022 with a set change registered on the pump following this alert. Pump data showed BMs rising since the set change; the patient gave a correction bolus via the pump following every one of these elevated readings but there is no record of any further set changes to rule out a cannula issue as an explanation for the persistently high glucose levels. We do not know whether the patient checked her ketones during this timeframe; the last recorded glucose reading on the pump was 33 mmol/l, and this was the last time a bolus was delivered (on the day before her hospital presentation with an OOH cardiac arrest).

The pump did not have any occlusion alarms throughout this period, but this would usually occur only with a complete blockage or pump malfunction. A partial cannula blockage such as a kinked or dislodged cannula would usually be detected by rising or not reducing levels despite correction doses in the absence of other factors causing hyperglycaemia such as infection or acute illness. The patient had normal infection markers and sepsis screen on presentation.

Immediate action was taken. Medtronic were contacted, and the pump returned to them as requested for interrogation. The pump has been sent to Medtronic US for full analysis. A safety mailshot was sent out to all 241 adult insulin pump users registered at COCH. SBAR was completed as requested for the Trust patient safety lead.

Abstract ID: 14

Fluid management and fluid shifts in diabetic emergencies – an ongoing dilemma

Sankaran L,[1] Mackli A[2]

1. Internal medicine trainee year 3 , Department of Diabetes and Endocrinology, Dorset County Hospital NHS foundation trust, Dorchester; 2. Consultant In Diabetes and Endocrinology , Dorset County Hospital NHS Foundation Trust, Dorchester

Case report: a 47-year-old Romanian national, previously fit and well, presented with a 15- hour history of acute balance issues, vomiting and veering to the right side.

He was hypertensive and vomiting on arrival at hospital, and on neurological examination he had lower limb ataxia. A CT angiogram of the head revealed an acute posteroinferior cerebellar stroke with bilateral vertebral artery occlusion and mass effect with compression of fourth ventricle. Urgent transfer to the regional neurosurgery unit was recommended.

A venous blood sample showed a glucose level of 32 mmol/L, a pH of 7.01 and ketosis (6.8 mmol/L) consistent with diabetic ketoacidosis (DKA). Treatment was started with fixed-rate insulin and a bolus of normal saline, according to national guidelines. The stroke team raised concern about potential worsening cerebral oedema and risk of brain stem herniation, however.

The local diabetic team reviewed him and advised that treatment should continue using the national protocol. DKA was resolved after six hours, and variable-rate insulin was initiated. He was transferred to the local neurosurgery unit, where the patient had posterior fossa craniectomy. He was started on basal bolus insulin post operatively. A test for GAD antibodies came back negative. A CT scan of pancreas was also performed, which did not show an abnormality. The patient was subsequently discharged with a diagnosis of type 1 diabetes (T1DM).

Discussion: This case raises two issues. Which came first, the DKA or the stroke? In DKA there is activation of the coagulation and inflammatory cascades, which could cause brain infarcts. However, it could also be argued that the stress of the stroke precipitated DKA in a susceptible individual.

Secondly, the treatment of DKA – insulin and rapid intravenous fluid administration-- can induce cerebral oedema due to the rapid shift in osmolality. Inadequate fluid and electrolyte replacement also carries a risk of adverse outcomes. In a paediatric population an average drop of effective serum osmolality (2 × plasma sodium [PNa] + plasma glucose concentrations) by 9 +/- 2 mOsm/kg in four hours was associated with cerebral oedema. Some studies suggest that cerebral oedema should be anticipated in adults with a similar drop in serum osmolality, and fluids should be adjusted accordingly. In this case the osmolality dropped by 14 mOsm/kg in six hours. This case report highlights that fluid administration should be considered on a case by case basis in adults. As these cases are rare, it is difficult to accumulate case reports and experience. There are no set guidelines as of yet.

Finally, accepting that cerebral oedema may well be idiosyncratic rather than iatrogenic could have important medicolegal connotations.

Abstract ID: 18

Type 1 disordered eating: a new domain in mental health and diabetes

Naeem A,[1] Kunka D,[1] Ismail K,[2] Turner D,[2] Rosenthal M[1]

1. Royal Free NHS Foundation Trust; 2. T1DE London King's College Hospital

Case presentation: JM was diagnosed with type 1 diabetes (T1DM) at the age of 12 years. At 16 years, she presented with recurrent

diabetes ketoacidosis (DKA). She had poor engagement with the diabetes team at the Royal Free Hospital (RFH). Despite integrated care (diabetes and mental health) she continued to have repeated episodes of DKA and would present herself to different NHS Trusts for treatment. Her body mass index (BMI) become low, and it was realised that she was omitting insulin in order to lose weight. In 2020 she was referred to the T1 disordered eating (T1DE) pilot at King's College Hospital (KCH), where she was treated for 24 months. Her HbA_{1c} improved from 14% to 6.2 % with no episodes of DKA over 10 months, showing compliance and response to the treatment strategies.

Our case highlights two important management issues in this cohort for the general diabetes team.

1. Delay in identification of T1DE. JM had repeated admissions to different Trusts and was not seen at the RFH for many months. This resulted in an obscuring of the clinical picture along with a delay in developing a management strategy and a diagnosis, hence a delay in referral for an effective treatment.
2. Once engaged with the T1DE team, care was taken to lower the HbA1c slowly over time so as not to precipitate insulin oedema and exacerbation of diabetes complications.

Conclusion: type 1 disordered eating (T1DE) refers to a serious mental health disorder in which patients omit their insulin in order to control their weight. T1DE includes a range of other behaviours, including restriction of food, over-exercise, self-induced vomiting and abuse of laxatives and diuretics. It is most common in young teenaged females living with T1DM and can result in life-threatening recurrent DKA and long-term microvascular and macrovascular complications.

The T1DE service at KCH is an intensive multidisciplinary intervention delivered by a liaison psychiatry psychoanalytic psychotherapist, CBT therapist, diabetes nurse specialists, diabetes consultant and a patient navigator as well as local teams in a hub. It is a bespoke model using assertive outreach to engage patients. Service evaluation has demonstrated significant improvements in outcomes.

People with T1DE most commonly come in contact with the acute medical and emergency staff rather than diabetes teams due to repeated presentations with DKA. The authors recommend that there needs to be increased awareness of T1DE in these clinical groups to support early diagnosis and appropriate management of blood glucose together with clear referral pathways.

Abstract ID: 19

Patient choice in the management of diabetic foot ulcer requiring amputation: factors favouring conservative management

Nijith L, Chan S, Banerjee R

Luton and Dunstable University Hospital, Bedfordshire NHS Trusts

A 46-year-old man with a history of type 2 diabetes (T2DM) diagnosed 11 years previously presented with a new-onset right foot ulcer with black discoloration of the dorsum of the foot, involving the fourth toe and 4/5th web space. He was diagnosed with ascending cellulitis and superficial gangrene with a non-viable right fourth toe.

After he was seen by the diabetes foot multidisciplinary team (MDT), the patient underwent surgical debridement of the ulcer and fourth right toe amputation. CT angiography of the right lower limb showed tight stenosis distally of the right popliteal artery with multilevel disease and angioplasty was carried out. The MDT suggested transmetatarsal amputation, which the patient declined. He opted for conservative management. He had maggot therapy followed by Natrox wound oxygen therapy. He was discharged with a vacuum-assisted closure dressing. Follow-up appointments showed significant improvement, with wound healing.

The possible factors that might have caused the wound healing are: the site of the ulcer (in the non-weight-bearing area of the foot), antibiotic coverage of infection, initiation of insulin for better glycaemic control, early involvement of the MDT team comprising a diabetes specialist, vascular surgeons, orthopaedic team and podiatrist, maggot therapy, vacuum- assisted dressing and Natrox oxygen therapy.

This case study also demonstrates a favourable outcome for a diabetic foot ulcer where Natrox was used. This device delivers up to 98% of humidified oxygen directly to the wound bed, stimulating wound healing. Recent NICE guidance states that Natrox therapy is beneficial as adjunct therapy in grade II/III diabetic foot ulcers. Our local centre has limited use of this.

We propose increased usage of Natrox oxygen therapy as an adjunct to other conservative measures to augment wound healing. This will need further education, funding and reports of its usage in multiple centres.

Abstract ID: 20

Pre-hyperosmolar hyperglycemic state, frailty and COVID-19

Sathyanarayan S, Saqib A

Guy's and St Thomas' Hospital

We present the case of an 88-year-old woman who was known to have type 2 diabetes mellitus (T2DM). She was wheelchair-bound, had carers three times a day and had multiple other co-morbidities. Her pre-existing diabetes control was sub-optimal and her HbA_{1c} on admission was found to be 135 mmol/mol. Pre-admission she was treated with metformin and Abasaglar insulin 10 units. Her insulin was administered by her son, who also monitored her blood sugars at home.

She started to feel tired two days prior to admission and her son called the ambulance when her blood sugar readings at home were high. On presentation to the hospital, blood glucose was 36mmol/L, pH was 7.4 and ketones 0.1mmol/L, serum sodium was 127mmol/L and serum urea was 14.4mmol/L. Her calculated serum osmolality was 304.4mOsmol and laboratory serum osmolality was 318mOsmol.

She tested positive for Sars-Cov-2 on admission but had no chest X-ray changes and did not need oxygen.

She received 10 units of Actrapid insulin prior to admission under the medical team and 4 units NovoRapid insulin with a litre of Hartmann's solution in the emergency department. Metformin had to be held due to acute kidney injury. Her blood glucose remained between 15–27mmol/L despite these treatments and intravenous fluid administration, so she was given a variable rate insulin infusion (VRII). Blood ketones remained low throughout. Once the blood glucose normalised the VRII was stopped and she was treated with Abasaglar insulin variable dose during admission.

She was started on dexamethasone 6mg for worsening COVID-19 with increased oxygen demand and COVID pneumonitis which worsened the hyperglycaemia. She was started on Humulin I insulin (13-19 units) units along with Abasaglar insulin. Unfortunately, she continued to deteriorate, and had hypoglycaemic episodes which were deemed secondary to poor oral intake and worsening multi-organ failure. Conversations were held with the family and she was managed as per the local end-of-life pathway until she passed away in hospital.

This case highlights poor outcomes in elderly patients admitted with COVID-19 and with pre-existing sub-optimally controlled diabetes,

how aggressive to be when managing elderly inpatients with COVID, and what targets to use. This case also highlights the need for continued glucose monitoring and insulin titration as the clinical condition changes in frail inpatients.

Abstract ID: 22

A case of deliberate insulin misuse: the complex interplay between physical and mental health

Madan A, Johnston T, Stones A, Donkoh Y, Pender S, Saqib A
Guy's and St Thomas' NHS Foundation Trust

A 21-year-old man with type 1 diabetes (T1DM) was admitted to intensive care in diabetic ketoacidosis (DKA), with a pH of 7.24, glucose 20 mmol/L and ketones 5.5 mmol/L. His DKA was managed with fixed-rate insulin and fluid resuscitation and his basal-bolus regime was adjusted. He is on multiple daily insulin injections and uses a Dexcom for his T1DM. His ability to manage his diabetes deteriorates significantly as his mental health deteriorates.

The reason for this episode of DKA was deliberate insulin omission as a form of self-harm due to severe depression and suicidal ideation. He has a background of emotionally unstable personality disorder, anxiety and depression. He had presented at least four previous times to hospital with self-harm secondary to insulin omission. He had recently had to drop out of his university degree due to a mental health breakdown and was now homeless. He was estranged from his family and extremely socially isolated.

The key issue that needed addressing to avoid readmission was the patient's mental health. The inpatient psychiatry team saw the patient and deemed that he did not reach the threshold for an inpatient admission but required close follow-up with the home treatment team. They determined that his behaviour was in keeping with chronic self-harm behaviour and he was not actively suicidal at present. The homelessness team was involved in re-housing the patient as he had lost his university accommodation.

For cases like this, it is vital for the diabetes team to work closely with the psychiatry team. This case illustrates the importance of a multidisciplinary approach to the patient's care and the critical interplay between mental health and its impact on physical health.

Abstract ID: 25

A challenging case of DKA in a patient with ESRF at high risk of fluid overload

Madan A
Guy's and St Thomas' NHS Foundation Trust

The management of diabetic ketoacidosis (DKA) in patients with end-stage renal failure (ESRF) remains challenging due to the risk of fluid overload and pulmonary oedema with fluid resuscitation. This case describes a 72-year-old man with ESRF secondary to diabetic nephropathy who developed DKA during an inpatient admission on the renal ward. The on-call team were contacted about this patient during the night shift as he had ketones of 5 and a pH of 7.28 on his venous blood gas. He was being treated for a diabetic foot infection and was already in pulmonary oedema. Management and resolution of his DKA were extremely challenging.

Cases like this require particular care when prescribing fluids and insulin, as well as regular assessments for signs of fluid overload. This case illustrates the difficulty in managing DKA in complex renal patients at risk of fluid overload. It also highlights the need for a protocol for management of fluid resuscitation in patients with DKA and ESRF.

Abstract ID: 26

Hyperglycaemia after transplantation

Aung NN, Cranston I
Portsmouth Hospital

Case report: the patient is a 33-year-old woman with a history of type 1 diabetes (T1DM) since the age of 11. She is not compliant with diabetes management and is known to be in self-management of her own diabetes.Sixteen years after her diagnosis of diabetes, she had chronic kidney disease (CKD) stage V secondary to diabetes nephropathy, proliferative diabetic retinopathy with vitreous haemorrhage, peripheral neuropathy and diabetic gastroparesis.

In August 2016, peritoneal dialysis was started while she waited for a simultaneous pancreas and kidney transplant in Oxford. She had a transplant in April 2018 from a DBD donor. Her medication after the transplant included tacrolimus, mycophenolate, co-trimoxazole, valaciclovir and atorvastatin. She had been seen by the diabetes team approximately 15 months after her transplantation while she was planning a pregnancy. Her 7 points of self-monitoring blood glucose were satisfactory, with capillary blood glucose ranging between 4 to 7 mmol/L and HbA_{1c} of 33 mmol/L.

In August 2022, there was a gradual increase of creatinine with a level up to 210 mL/min. Ultrasound of the kidney showed hydronephrosis, which was initially thought to be responsible for the increasing creatinine. However, the second opinion from the radiology team was that long-standing changes were more likely. At the same time, the tacrolimus level was out of range and the plan from the renal team was dose adjustment and repeated tests of kidney function. As her kidney function was still deranged even after the tacrolimus levels were normal, a renal biopsy was arranged.

The diabetes team were asked to see her to address the raised glucose.

A renal biopsy showed stage IV lupus nephritis and she was treated with IV 500 mg methylprednisolone for three days followed by 40 mg of prednisolone.The patient had steroid-induced hyperglycaemia secondary to use of glucocorticoids and the Liber record showed raised glucose in the evening, which is consistent with steroid-induced hyperglycemia.

A Humulin I Kwikpen was started to tackle steroid-induced hyperglycemia.

She is currently on prednisolone, weaning the dose by 5 mg every 2 weeks and dose titration of Humulin I according to her day-to-day glucose profile. We will make a further appointment to review her in three months' time, at the end of her steroid weaning period.

Abstract ID: 27

Double diabetes in a family – getting the diagnosis right

Ahsan M, Dales J, Gregory R, Kong M
The University Hospitals of Leicester NHS Foundation Trust

Introduction: Monogenic diabetes is still frequently misdiagnosed as either type 1 or type 2 diabetes (T1DM or T2DM). Accurate diagnosis of diabetes subtype ensures appropriate treatment, reducing the risk of complications and the treatment burden on the patient. Here we present a family with HNF1A (MODY3) monogenic diabetes and also T1DM with negative HNF1A mutation.

Case report: A 72-year-old woman was diagnosed with diabetes at the age of 9 (in 1964) following osmotic symptoms. She was started on Insulin initially and then gradually weaned off that and started on tolbutamide. It was not previously thought possible to stop insulin in children with diabetes and the success of her case on tolbutamide gen-

erated interest in the medical literature in the early 1960s. She went back onto insulin in 1968 when she started having evening glycosuria.

She has always been managed as a T1DM patient, and she continued her treatment with insulin.

Her other past medical history includes necrobiosis lipoidica, thyrotoxicosis, ischaemic heart disease and coeliac disease.

Family history of diabetes: Her mother was diagnosed with diabetes after childbirth in 1963 and was always treated as a T1DM patient, with insulin. In 1994 her 17-year-old son was having osmotic symptoms and started on insulin for T1DM. In the same year her daughter, aged 15, was noted to have glycosuria after using her brother's test strips. She was started on insulin for T1DM.

Question of monogenic diabetes: The first suspicion of monogenic diabetes was raised in 2006, noting the strong family history of diabetes and the lack of diabetic ketoacidosis on insulin cessation. She was referred to clinical genetics and had positive genetic testing in 2010 for HNF1A. Her urinary C-peptide 3.47nmol/L with a C-peptide:creatinine ratio 0.68 suggested significant endogenous insulin production.

At that point further genetic testing on her family was undertaken. Her mother remained on insulin until her death in 2014 and did not undergo any genetic testing. Her daughter was also found to be positive for HNF1A. Her son underwent genetic testing: the HNF1A mutation was not identified and he remained on Insulin.

Discussion and learning point: This case report demonstrates T1DM and monogenic diabetes co-existing in the same family. If her son been the first to have the genetic test it might have been inferred that the rest of the family were also negative. Therefore, a single negative genetic test in a family should not prevent further genetic testing. Moreover, other co-existing autoimmune conditions more prevalent in people with T1DM may not adequately differentiate between monogenic diabetes and T1DM.

Abstract ID: 31

DKA-induced pancreatitis: a case report

Cox L, Craig S, Douek I

Musgrove Park Hospital, Taunton, UK

A 43-year-old man was brought to the Emergency Department by ambulance after he was found collapsed outside his GP surgery. He had contacted the practice eight days previously complaining of a 3-week history of polyuria, polydipsia, fatigue and weight loss. He declined a face-to-face review, but agreed to a telephone assessment and blood tests. His HbA_{1c} was 108 mmol/mol and he was prescribed metformin, but had not yet collected it from the pharmacy.

The patient's past medical history included psoriasis, raised BMI (38kg/m^2), opiate dependence and previous alcohol excess; he and his partner confirmed that he no longer used alcohol or recreational drugs. He had no fixed abode and was unaware of his family history.

On admission, he had a strong ketotic odour, fever of 38.5^{0}C and was in cardiovascular shock. There were bibasal coarse crepitations on lung auscultation, but abdominal examination was unremarkable. Investigations revealed pH 6.99, glucose 51.3 mmol/L, ketones 7 mmol/L (<1), bicarbonate 4mmol/L (24-32), and potassium 5.2mmol/L (3.5-5.0). CRP was 198mg/L (<10), amylase 366µ/L (22-80) and creatinine acutely raised at 155µmol/L (50-120). Triglycerides were 8.8mmol/L (<1.8), and islet cell autoantibodies were negative. His chest X-ray confirmed bilateral consolidation.

The patient was diagnosed with diabetic ketoacidosis (DKA), new-onset diabetes, moderate hypertriglyceridaemia, bilateral community-acquired pneumonia and acute kidney injury (AKI). He was volume resuscitated with intravenous crystalloid, commenced on intravenous piperacillin/tazobactam and a fixed-rate insulin infusion. The following morning, he became drowsy and complained of new epigastric tenderness. A CT scan of the head excluded cerebral oedema, but CT abdomen showed peripancreatic inflammatory fat stranding with free fluid posteriorly, consistent with acute pancreatitis (AP); there was no pancreatic necrosis, and the gallbladder was collapsed with no stones.

The 'enigmatic triad' of DKA, hypertriglyceridaemia and AP is increasingly reported but, because AP and DKA present similarly, it is often under-recognised (Simones-Linares *et al*., 2019).[1] Research has found that 11% of patients with AP also have hypertriglyceridaemia and DKA, with AP and hypertriglyceridaemia both being a cause or consequence of DKA.[1,2] Although the exact pathophysiologic role of DKA within this triad remains undetermined, evidence suggests that patients have higher rates of hypertriglyceridaemia and complications (e.g. AKI and systemic inflammatory response syndrome), longer and more costly hospital stays, and higher mortality compared to those without DKA,[1,3] thus highlighting the importance of early diagnosis and treatment.

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Abstract ID: 32

Antibiotics or amputation: balancing the risks and benefits in patients with severe osteomyelitis

Vathenen R, Amin A, Todd G, Kong W

Central Middlesex Hospital, Acton Lane, London, NW10 7NS

Introduction: Extended antibiotics are an effective alternative to amputation for diabetes foot osteomyelitis (DFOM).[1,2] However, frequent and prolonged antibiotics may be required, increasing the risk of resistant organisms such as methicillin-resistant Staphylococcus aureus (MRSA). We present a man with DFOM who avoided amputation through prolonged courses of antibiotics but developed teicoplanin-resistant MRSA.

Case report: A 77-year-old man with type 2 diabetes (T2DM) presented in December 2019 with forefoot wet gangrene. Despite successful revascularisation there was minimal improvement with antibiotics. A transmetatarsal amputation was advised but the patient declined.

He slowly improved with multiple extended courses of targeted antibiotics (a total of 15 months of antibiotics, including three months of teicoplanin to treat MRSA).

After six months off antibiotics, his ulcer deteriorated again and bone cultures were positive for teicoplanin-resistant MRSA. This was treated successfully with six weeks of high-dose ciprofloxacin. His foot is now near healed, with radiological resolution, and he remains independently mobile.

Subsequently, two other patients developed genotype-matched teicoplanin-resistant MRSA and non-limb-threatening infection.

Discussion: Managing DFOM with vascular disease is challenging. Amputation carries the risk of non-healing and further progression to major amputation.[3] Whilst conservative management resulted in a

good outcome for this man, it led to the development of teicoplanin-resistant MRSA and cross-contamination of two other people with DFOM. This case illustrates the challenges of antimicrobial stewardship in patients with severe foot disease; and balancing risks and benefits of early amputation compared to conservative treatment.

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Abstract ID: 33

First presentation of HNF1B-MODY5 at age 40 years without a family history of diabetes

Thackray K, Sibal L

Cambridge University Hospitals NHS Foundation Trust, Cambridge

Introduction: People with MODY are often misdiagnosed as having type 1 diabetes (T1DM) or type 2 diabetes (T2DM). The Exeter MODY Probability Calculator shows good discrimination in distinguishing monogenic diabetes from other types in people diagnosed under the age of 35 years but is not validated in people presenting when they are older than 35 years. We present a case of a 40-year-old woman without a history of diabetes in a first- degree relative, in whom genetic testing was undertaken due to her past history of urogenital problems. Testing confirmed a de novo presentation of HNF1B-MODY or MODY5 due to a microdeletion in the 17q12 region.

Case report: A 40-year-old woman presented with a 5-day history of cystitis. Urinalysis demonstrated significant ketonuria and glycosuria. Her blood glucose was 46.6 mmol/L. She was mildly acidotic so was initially treated for diabetic ketoacidosis, although she did not strictly meet the criteria, as a first presentation of T1DM. Body Mass Index was 26.1 kg/m^2 but examination was otherwise unremarkable. She was commenced on a basal-bolus insulin regime. She had a past medical history of endometriosis and a partial laparoscopic hysterectomy for a left uterine horn. A coronary arteriovenous malformation had been identified incidentally as part of pre-operative investigations. She came from a very large family but the only significant family history was a brother who had had three kidney transplants.

Her C-peptide level was 507 pmol/L. GAD-65 and islet cell autoantibodies were negative. Renal function was within normal limits. Liver function tests were raised, with ALP 145 U/L (normal range [NR] 30-130) and ALT 66 U/L [NR 7-40]). The liver screen was unremarkable, with only a mild right hydroureter/nephrosis on liver ultrasound. Subsequent CT demonstrated an extra-renal pelvis in the right kidney, felt to be a normal variant.

Although she was older than 35 years at diagnosis, given her initial presentation of diabetes, negative autoantibodies, a past medical history of genitourinary abnormalities and elevated liver function tests, MODY 5 was considered. Genetics confirmed MODY 5, with a 1.4 megabase deletion within 17q12 which includes the HNF1B gene. This was not seen in either parent, confirming a de novo mutation.

Conclusion: MODY5 has a diverse phenotype, including endocrine and exocrine pancreatic insufficiency, urogenital abnormalities, and renal and liver disease. Clinicians need to maintain a high index of suspicion to reduce the possibility of misdiagnosis of MODY as T1DM or T2DM. This facilitates the delivery of personalised medicine, including optimal treatment and genetic counselling.

Abstract ID: 35

Acute painful neuropathy following rapid glycaemic control

Amer L, Rajbhandari S

Diabetic and Endocrine department, Chorley and South Ribble General Hospital

Background: Rapid metabolic correction of glucose levels can sometimes give rise to untoward complications. Insulin neuritis is an historical term for an acute neuropathy affecting patients with diabetes who achieve rapid re-establishment of previously poor glycaemic control. It presents with neuropathic pain, symptoms of autonomic dysfunction or a combination of both, and is usually reversible. If the clinician treating the patient is not aware of the condition, there can be undue stress to patient, as in this case.

Case presentation: A 48-year-old businessman was diagnosed with diabetes in October 2019 with HbA_{1c} 66 mmol/mol and was managed with lifestyle changes and diet. However, he did not have any regular follow-up after that. In January 2022, he presented to his GP with osmotic symptoms (polyuria and polydipsia) and with an HbA_{1c} 131 mmol/mol at that time. He was given lifestyle advice and was started on metformin 2000 mg daily. He also researched diabetes and its management and became very strict with his diet and exercise.

His HbA_{1c} had significantly improved in May 2022, at 52 mmol/mol. However, he started complaining of neuropathic pain in both feet. This was not fully controlled with duloxetine 120 mg and pregabalin 300 mg daily in two divided doses. The patient attributed this pain to a side effect of metformin. His GP changed this medication to gliclazide 80 mg twice a day and the patient was referred to neuropathy clinic by the podiatrist as per local protocol in November 2022.

In the clinic his HbA_{1c} was 50 mmol/mol. He had severe neuropathic pain involving both legs and the right hand. This affected his sleep. On examination his vibration threshold and monofilament sensations were normal. He had good peripheral pulses, and other tests such as vitamin B12 and thyroid function tests were in the normal range. The diagnosis of 'insulin neuritis' was explained to the patient and he was reassured that it would get better with time. His duloxetine was changed to amitriptyline to improve his sleep quality.

Discussion: This case explains the need for physicians managing diabetes to be aware of this uncommon condition. It is unclear whether relaxation of glycaemic control is warranted, and whether this would lead to a more rapid resolution of insulin neuritis, but the current consensus is that good glycaemic control should not be relaxed. Physicians should reassure their patients that this condition is reversible over months after stable diabetes control. This will avoid unnecessary anxiety and suffering to patients.

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Abstract ID: 39

DKA and COVID-19 in pregnancy

Gregori M, Brackenridge A, Banerjee A, Saqib A

Guy's and St Thomas' NHS Foundation Trust

Background: During the recent Covid pandemic, an association was observed between COVID-19 infection and diabetic ketoacidosis (DKA) presentation, with these patients often experiencing adverse

outcomes. In people with diabetes mellitus, pregnancy is an additional risk factor for DKA and is also associated with morbidity and mortality.

Clinical case: A 40-year-old woman with type 1 diabetes (T1DM) and pregnancy-induced hypertension (PIH) presented at 35 weeks' gestation with hypertension and reduced fetal movements.

The patient had a previous pregnancy complicated by PIH. Pre-pregnancy glycated haemoglobin (HbA_{1c}) was 96mmol/mol. She had evidence of background diabetic retinopathy but no evidence of nephropathy. The patient struggled with glycaemic control throughout her pregnancy; during the second half she was managed on a regime of insulin levemir and three-times a day insulin novorapid, with continuous glucose monitoring (CGM). She was taking aspirin 150mg daily for pre-eclampsia (PET) prophylaxis and nifedipine monotherapy for PIH. The patient conceived during the second wave of the COVID-19 pandemic and was unvaccinated.

On assessment at 35 weeks, her blood pressure was 150/90mmHg. Cardiotocography (CTG) did not demonstrate any abnormality. Urinalysis demonstrated proteinuria and the urine protein to creatinine ratio (PCR) was sent to the laboratory for quantification. In view of her diagnosis of PET and reduced fetal movements hospital admission was recommended. However, due to childcare issues the patient was unable to stay in hospital.

The patient presented again two days later, vomiting and hypertensive. The fetal heart could not be auscultated and abdominal ultrasound sadly confirmed intrauterine death. Investigations were consistent with DKA, with hyperglycaemia (glucose 29.1mmol/mol), unrecordable ketonemia and metabolic acidosis (pH 7.2, HCO3 14mmol/L). She had elevated inflammatory markers (white cell count 10.3x109, C-reactive protein 114 mg/L) and she tested positive for SARS-Cov-2. The urinary PCR result from her prior attendance was elevated (105 mg protein/mmol creatinine).

The patient was admitted to the intensive care unit (ICU), where she was commenced on the DKA protocol and received intravenous magnesium and labetalol therapy for PET. She required supplementary oxygen therapy and was treated for COVID-19 with dexamethasone, remdesivir and casirivimab/imdevimab. She underwent a lower segment caesarean section the following day and was stepped down from ICU after three days.

Conclusions: Recent confidential enquiries have highlighted the rise in maternal mortality related to both DKA and PET, and have demonstrated the negative impact of COVID-19 on the care these women received. To promote better maternal and fetal outcomes we must have a low threshold for diagnosing DKA in pregnancy and must manage these patients within a multidisciplinary team.

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Abstract ID: 40

Atypical presentation of a typical problem

Shah V, Hill N
Imperial College Healthcare NHS Trust

A 44-year-old male of south Asian ethnicity self-referred to a private diabetes specialist clinic after a 1-week history of polyuria and nocturia. There was no history of recent weight loss prior to referral and he had no gastro-intestinal symptoms. His weight was 62 Kg (height 1.7m) and body mass index 21.4 Kg/m^2. On initial assessment he had a random blood glucose of 20 mmol/L and ketonuria, with HbA_{1c} 93 mmol/mol. He was immediately commenced on basal bolus insulin with a working diagnosis of type 1 diabetes (T1DM) and he was provided with a flash glucose monitoring device (Freestyle Libre, FSL). He was also referred to an NHS specialist diabetes clinic for ongoing management.

There was no family history of diabetes (including his grandparents). He had been diagnosed with sensorineural deafness 10 years ago; his mother was also diagnosed with deafness in her late 40s. He had no cutaneous manifestation of insulin resistance and he had a lean phenotype. Biochemical investigations revealed negative test results for glutamic acid decarboxylase (GAD), islet antigen 2 and zinc transporter 8 antibodies. His C-peptide level was >1000pmol/L with paired blood glucose 21 mmol/L, suggesting adequate insulin secretion.

The patient made significant changes to his diet, including limiting carbohydrate and lipid intake. These led to him voluntarily discontinuing insulin use based on a stable blood glucose profile on his FSL. At his 3-month clinic review his continuous glucose profile estimated glycated haemoglobin (HbA_{1c}) was 51mmol/mol and this was confirmed with a lab HbA_{1c} of 47mmol/mol. This result was achieved through dietary measures alone and without use of insulin. A possible monogenic cause of diabetes was considered, including mitochondrial diabetes syndrome. Genetic testing for all known diabetes-related genes did not show any mutation, raising the possibility of lean type 2 diabetes (T2DM) or an unknown genetic mutation as the cause of his diabetes.

Important learning points include the complexities in the investigation of diabetes that have a phenotypic appearance of T1DM, and the safe de-prescription of insulin without the risk of developing diabetic ketoacidosis. This case also illustrates the atypical phenotype of lean T2DM and the role of genetic testing to identify affected individuals. It has implications for optimum management for genetic diabetes.

Abstract ID: 41

Recurrent hypoglycaemia in a patient with type 1 diabetes: not always too much insulin

Sadiq SA, Jayakar N, Maltese G(Consultant Endocrinologist and Diabetes), Johri N
Epsom and St Helier University Hospitals NHS FT

Abstract: A 48-year-old man with a known history of type 1 DM (T1DM), ischaemic heart disease and end-stage renal failure presented to the emergency department (ED) with a reduced level on the Glasgow Coma Scale (GCS) (E1 V2 M2-5). Initial assessment revealed low blood pressure due to chest sepsis and the reading on continuous blood glucose (CBG) monitoring was noted to be 1 mmol/L. He was treated with IV dextrose, fluids and antibiotics. Once CBG levels were normal, he was started on a variable rate insulin infusion (VRII) and later switched to his regular insulin.

Although he was recovering from sepsis, he had multiple

episodes of hypoglycaemia while an inpatient, even though his regular insulin was tapered and then completely stopped while monitoring CBG. Further investigation revealed that 9am cortisol were reduced to < 28nmol/L, with raised ACTH of 45ng/L. Further questioning revealed recent weight loss, borderline hyponatraemia and two previous admissions to the ED with a transient episode of confusion (which was diagnosed as a suspected transient ischaemic attack) and non-specific abdominal pain (which was treated as faecal overloading due to gastroparesis). He was started on steroid replacement therapy, and two days later the CBG was on a rising trend which meant that his regular insulin needed to be restarted.

Conclusion: Hypoglycaemia is the most common acute complication of insulin therapy. Our case suggests that the presence of Addison's disease should be taken into consideration in patients with T1DM and frequent episodes of hypoglycaemia. Addison's disease has been described in approximately 0.5% of patients with T1DM, being more frequent in females and occurring in middle-aged patients. Early diagnosis of Addison's disease in diabetic patients would prevent the morbidity and potential mortality of this association.

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Abstract ID: 44

Deterioration of glucose control attributed to new technology

Iqbal Z, Murphy J, Rowles S, Pichaipillai L

Fairfield General Hospital, Rochdale Old Rd, Bury, UK

We present the case of a 19-year-old man with type 1 diabetes (T1DM) since his diagnosis at the age of two years. He takes basal bolus metered-dose insulin: Tresiba 28 units at night and Fiasp 1 unit for every 7g of carbohydrate at all mealtimes. He self-monitors his blood glucose using the Accu-chek glucometer, despite having tried continuous glucose monitoring (CGM) using Libre 2 and Dexcom devices in the past.

He finds T1DM to be "a bit of a nuisance" in his life. He has had two episodes of diabetic ketoacidosis (DKA) in the last two years that occurred after missing insulin on days he was smoking cannabis.

With the CGM devices, he found that there was a deterioration in his glucose control, as these devices did not have an onboard bolus advisor that was present on his glucometer, and which he consistently used. While he was aware of phone apps that he could use for this function in tandem with CGM, he did not find them practical. Since returning to the Accu-chek glucometer, he finds it easier to use and is again making use of the bolus advisor.

The case highlights the fact that while the development of technology for T1DM is a positive step forward, the disparity in the functions available on devices may occasionally prove to be counterproductive. While many traditional glucometers have in-built bolus advisors, the Libre CGM sensors currently lack this function and require the use of an additional phone application to carry this out. This was not commensurate with the lifestyle of this patient. Real-world data from Southampton Clinical Commissioning Group report similar experiences for some of their patients transitioning to CGM.[1]

We are aiming to get this patient started on insulin pump therapy but the lack of bolus advisors on CGM may be seen as a barrier in patients striving to achieve good control on basal-bolus MDI therapy who may not be eligible for pump therapy or simply do not prefer it.

Reference:
Kelly B. FreeStyle Libre use in a real-world population: The Southampton City experience. *Journal Diabetes Nursing* 2019;**23**(3): 71.

Abstract ID: 45

Monogenic diabetes/HNF4A mutation

Memon SS, Fatima A, Dhere A

Royal Devon University Healthcare NHS Foundation Trust

We present a 30-year-old man diagnosed with diabetes at the age of 15 on a routine screen. In the initial years with his lean disposition, he was treated as type 1 diabetes mellitus (T1DM) with insulin. He lived at several places in the UK, with frequent changes to healthcare providers. Although at some point, given his strong family history of early-onset diabetes in his sister, father and grandmother, a genetic test for monogenic forms of diabetes was carried out, this was never firmly acted upon. The patient kept requesting the healthcare providers for tablets instead of insulin, but was not given regular prescriptions.

A year ago, he was referred to us by the ophthalmologist for visual deterioration from maculopathy and vitreal haemorrhage. His lifestyle previously included smoking, and alcohol excess. He weighed 63 kg, with BMI 26 kg/m^2 and HbA_{1c} 83 mmol/mol; he did not like insulin and had stopped taking it.

A re-evaluation revealed negative GAD, IA-2 and ZnT8 antibodies and preserved C-peptide of 1150 pmol/L with a glucose of 8.8 mmol/L. Contact with the national monogenic diabetes lead nurse at Exeter confirmed that our patient was tested in 2013 and that he had a mutation in HNF4A (p.P357fs/N).

A confirmed diagnosis and modified treatment to gliclazide 80 mg twice a day has improved his glycaemic control to HbA_{1c} 51 mmol/mol.

HNF4A diabetes responds well to sulphonylureas and our case highlights how taking the patient perspective into account and corroborating the clinical findings with confirmatory tests and appropriate early treatment modification could have avoided years of patient frustration, poor medication concordance and suboptimal control and complications.

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Abstract ID: 47

Managing gastroparesis in a patient with type 1 diabetes established on a closed-loop system

Aslam A

Royal Bolton Hospital, Manchester Royal Hosipital

According to a 2016 review gastroparesis is prevalent in 13.8 per 100,000 cases. Diabetes is the second most common cause, accounting for 37.5% of cases; idiopathic cases are the most common cause. Gastroparesis is more prevalent in patients with type 1 diabetes (T1DM). The 10-year cumulative incidence of diabetic gastroparesis has been estimated to be 5.2% in patients with T1DM and 1% in those with type 2 diabetes (T2DM).

This case describes a 31-year-old woman known to have had T1DM for 10 years and established on a T:slim pump Control IQ with

Dexcom G6 for 1.5 years. She attended the accident and emergency department with vomiting associated with abdominal pain, unable to eat and drink. On arrival her blood glucose was 10mmol/L but her ketones were not checked. She was diagnosed with gastroparesis earlier in the year when she presented with similar symptoms and was established on metoclopramide 10mg three times a day. She was unable to tolerate anything orally so her insulin pump was switched off and she was initiated on variable rate insulin infusion (VRII) along with long-acting insulin Lantus. Her HbA_{1c} was noted to be 64 mmol/mol. Her investigations suggested no evidence of infection and normal renal function.

She was eviewed by our diabetes specialist nurses the following morning, who advised to start the pump with 0% temp basal until Lantus insulin was due to run out, then to commence manual basal before placing back in Control IQ. A bolus was to be administered via pump for the meal before VRIII was stopped and then the VRIII was to be stopped 30-60 minutes after the meal-time bolus. After 36 hours the patient was able to eat and drink, and the VRII was then stopped. Dietician input was also sought, who advised on diet optimisation. The patient was later discharged with diabetes specialist nurse follow-up.

Gastroparesis, a form of autonomic neuropathy, is most commonly seen in people who have had diabetes for more than 10 years and who have already developed other microvascular complications. The most common symptoms are early satiety, nausea, bloating, abdominal pain and vomiting. Gastroparesis symptom typically persist over 12–25 years even with blood glucose optimisation. With new technologies, particularly with closed-loop systems, we have a better chance of improving diabetes control and preventing the complications but it is important to train staff and patients to manage the pump in the event of an acute episode.

Abstract ID: 48

When pregnancy may, or may not, be good for your health

Batten L,[1,2] Allan B[1]

1. Hull University Teaching Hospitals, Hull; 2. Hull York Medical School, University of Hull

We present the case of a 33-year-old woman, gravida 3 para 2, diagnosed with gestational diabetes mellitus (GDM) early in pregnancy at 12+2 weeks. She was tested for GDM early in the pregnancy since she had had GDM in previous pregnancies. She was routinely seen in the medical obstetrics clinic and was started on metformin and, subsequently, insulin. Her doses had been titrated up to metformin 1g twice a day, Humulin insulin 54 units twice a day and Humalog 24 units three times a day with meals.

At 28 weeks, she had a sudden episode of hypoglycaemia and so all diabetes treatment was stopped. Capillary blood glucose levels before meals were between 1.9mmol/L and 3.7mmol/L. Despite stopping the medication, she still had adrenergic symptoms related to hypoglycaemia. She required large quantities of glucose-containing drinks to bring her blood glucose back into the normal range and to improve symptoms. Fetal growth was in the normal range on a customized growth chart and fetal movements were normal. The patient confirmed that she had had similar symptoms during her previous pregnancies, both of which resulted in an early delivery by emergency Caesarean section.

She continued to have documented hypoglycaemia (2.5 mmol/L) and so was admitted to the obstetrics department for observation. There were concerns at this point that she might have an insulinoma. Blood samples were taken during a hypoglycaemia episode while she was an inpatient (glucose 1.5mmol/L). The results showed an insulin level of 2870pmol/L, C-peptide <94pmol/L, IGF-1 19.4nmol/L and growth hormone (GH) 3.0ug/L. A sulfonylurea screen was negative. IgG insulin antibodies were negative. A PEG precipitation test was performed which resulted in an insulin level of 2540pmol/L, indicating a recovery of 88%. Expert opinion was sought regarding the results. It was confirmed that C-peptide levels are not affected by freezing or a delay in testing and that in cases of insulin autoimmune syndrome, PEG insulin recovery is usually less than 15%.

The obvious concern is that exogenous insulin administration could lead to an emergency caesarean section delivery at 28+5 weeks, with a potentially significant impact on the baby's development and well-being.

Abstract ID: 49

The use of a hybrid closed-loop (HCL) system in a patient with gastroparesis and recurrent diabetic ketoacidosis: a case report

Alomari L, Crabtree TSJ , Redfern A , Kedge C, Langeland L, Redfern Z, Gerrard K, Sugunendran S, Wilmot E, Robinson E, Bakhit M

University Hospitals of Derby and Burton NHS Foundation Trust

Introduction: Hybrid closed-loop insulin delivery systems (HCL) proved to be impactful in the management of type 1 diabetes (T1DM), through improvement in time spent in normoglycaemia and in patients' quality of life. Both high and low glucose levels are known to affect gastric emptying, thus labile glucose levels may exacerbate symptoms in individuals with gastroparesis. The use of HCL systems in these patients is limited due to lack of evidence to support their efficacy and safety.

Case report: We describe a case of a patient with T1DM who had significant gastroparesis and recurrent hospital admissions with diabetic ketoacidosis (DKA). This is a 50-year-old woman who was diagnosed with T1DM 10 years ago. She developed gastroparesis, which led to multiple hospital admissions either due to gastrointestinal (GI) symptoms or DKA, including several admissions to the intensive care unit (ITU). Due to severe nausea and vomiting, she needed to have a percutaneous endoscopic jejunostomy (PEJ) feeding tube inserted. Between December 2021 and July 2022, she had a total of seven hospital admissions with DKA, with a total of 181 days spent in hospital.

Due to the recognised considerable risk of further morbidity and mortality, it was agreed by the multidisciplinary team to start her on an HCL (MiniMed™ 780G system with Guardian™ 4 sensor); this was started in August 2022. Appropriate training was provided during her hospital stay, followed by intensive outpatient remote and face-to-face follow-up. This resulted in significant improvement in her blood glucose levels, achieving a time in range (TIR, 3.9-10mmol/L) of 71% on her most recent data. Time spent in hypoglycaemia (time below range or TBR, <3.9mmol/L) was 0%. She has not had a single hospital admission with DKA since the initiation of HCL (3 months' follow-up). Due to the improvement of her GI symptoms, tube feeding became unnecessary. She also reports significant improvement in her mental health and quality of life.

Conclusion: Patients with gastroparesis have delayed and unpredictable gastric emptying. Labile glucose levels can worsen this, leading to a vicious cycle. HCL systems may have a role in these patients, leading to improvements in glycaemic control, gastroparesis symptoms and quality of life; these may reduce the frequency of hospital attendances and future complications, which significantly reduce the cost of care.

Larger studies, with longer duration of follow-up, are needed to support the routine use of HCL in patients with T1DM and gastroparesis.

Abstract ID: 50

A pitfall in HbA_{1c} interpretation revealed by diabetes technology

Ranasinghe B, Murray M, Kean J, Davie S, Chinnasamy E
Kingston Hospital NHS Foundation Trust, Kingston-upon-Thames

A 37-year-old male Iraqi asylum-seeker with type 1 diabetes (T1DM) and a complex psycho-social background was referred to us. His glycated haemoglobin (HbA_{1c}) was 55 mmol/mol, on Tresiba 46 units nocte and Novorapid 1:10gm. He increased frequency of testing and was commenced on flash glucose monitoring. This revealed his true glycaemic control, with a higher GMI (glucose management indicator). His HbA_{1c} levels were inappropriately low. He is not on any over-the-counter medications. There is no history of blood or iron transfusions. His capillary blood glucose (CBG) correlated with his libre results.

Results (includes 90-day GMI with at least 95% data):
September 2021 HbA_{1c} 51 mmol/mol vs. GMI 72 mmol/mol
January 2022 HbA_{1c} 69 vs. GMI 79
June 2022 HbA_{1c} 52 vs. GMI 88
October 2022 HbA_{1c} 56 vs. GMI 81
Haemoglobinopathy screen was negative
Full blood count (FBC) was normal with marginally raised MCV intermittently (he did not drink alcohol, and vitamin B12, folate and thyroid function tests were normal)
Renal and liver function tests were normal
Fructosamine: 444 umol/L
Reticulocyte count: 125 (30-100), normal bilirubin level and haemoglobin (Hb) was 140gm/L

Discussion: HbA_{1c} has been the cornerstone of monitoring people with diabetes and is increasingly used for diagnosis of diabetes. It is important to understand the factors that may affect HbA_{1c} other than blood glucose. The following can cause inappropriately low HbA_{1c} levels: conditions causing increased red blood cell (RBC) turnover (haemolytic anaemia, acute blood loss, recent blood transfusion, treated iron/vitamin B12/folate deficiency, splenomegaly and reticulocytosis), haemoglobinopathies and methaemoglobinaemia, factors interfering with some assays like uraemia, hyperbilirubinaemia, medication/supplements altering glycation like vitamin C and medication inducing RBC destruction and altering Hb. The rate of glycation of haemoglobin can vary between individuals (low vs. high glycators). Glycation of haemoglobin may be different to glycation of other proteins outside RBC such as albumin, hence measuring fructosamine is helpful. The patient's complex psychosocial background makes it difficult to improve his glycaemic control. We have now commenced him on Novo pen 6; this along with libre would help us to intensify his therapy.

This case highlights the role technology plays in identifying causes where GMI and HbA_{1c} do not correlate and how smart pen technology could be used for clinical benefit. He will be referred to a haematologist for reticulocytosis workup. With widespread access to advanced glucose monitoring technologies, we could easily pick up cases like this.

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Abstract ID: 51

The importance of considering monogenic diabetes at diagnosis—a tale of two cases

Ahmad S, Owen K, Anguelova L
Oxford Centre for Diabetes, Endocrinology and Metabolic Medicine, Churchill Hospital, University of Oxford.

Case 1: A 27-year-old woman was referred to the diabetes clinic. She had been diagnosed with type 1 diabetes (T1DM) at the age of 16, when polydipsia developed. No other osmotic symptoms or weight loss were reported. She commenced basal-bolus insulin regimen at diagnosis. GAD and ICA antibodies were negative, but T1DM was still considered the most likely diagnosis. There was no family history of diabetes (although only limited information was available).

A plan was made to revisit the aetiology later, but she was subsequently lost to follow-up. No diabetes-related hospital admissions were reported in subsequent years.

Ten years after the initial diagnosis, her GP referred her for review. The patient had struggled to engage with diabetes care, but despite not checking her blood glucose levels for 3 years, she had a normal HbA_{1c} of 5.3% and was using small insulin doses with no evidence of diabetes complications or hypoglycaemia.

Paired glucose and C-peptide levels were 5.1mmol/L and 910pmol/L, respectively. Islet antibodies (anti GAD, IA2, anti-islet cell, anti ZT8) were all negative.

Genetic testing for MODY was undertaken. It was positive for pathogenic variant in HNF1A, the commonest cause of MODY. Her insulin was switched to 20mg gliclazide, with an excellent response. The results revealed blood glucose (BG) 7-9mmol/L, HbA_{1c} 5.3% and no hypoglycaemia episodes.

Case 2: A 62-year-old man was referred for investigation. He was incidentally diagnosed with diabetes at the age of 21 when glycosuria was found during a medical check-up. He was lean with no other features of diabetes. A clinical suspicion of MODY was raised, but at the time the genes causing MODY were not known. He was offered insulin treatment, which he refused, and was treated with glibenclamide. He developed hypoglycaemia, however. He managed his diabetes with diet for 10 years, before being switched to gliclazide. Gliclazide was stopped in 2020 due to low HbA_{1c} levels but he quickly developed high glucose up to 26mmol/L and blurred vision so gliclazide was restarted, with good effect.

He had well-controlled HbA_{1c} levels between 5.7 and 6.2%, and no diabetes complications. There was a family history of diabetes in his cousins.

Forty years after his initial diabetes diagnosis, he underwent genetic testing. This revealed a novel ABCC8 mutation (MODY 12). This is a rare cause of MODY that is sensitive to sulphonylureas.

Conclusion: These cases demonstrate how the diagnosis of MODY can be missed for years, with significant impact. Always consider monogenic diabetes in young patients with atypical presentations, as the management is different from that for T1DM or T2DM. All diagnostic tests should be utilised and results followed through. People with rare forms of diabetes may also be under general practitioner care.

Abstract ID: 52

A familiar face: care in a patient with type 1 diabetes complicated by depression and apathy

Boyle LD, Adams C, Aikins-Andoh C, Hall-Wainwright SL, Sankoh F, Theodoraki A

Chelsea & Westminster Hospital NHS Foundation Trust

Case history: A 19-year-old female was referred to the diabetes multidisciplinary team (MDT) in October 2020, having been admitted every 3-6 weeks in diabetic ketoacidosis (DKA), with blood glucose levels typically >40mmol/L and ketones >6 mmol/L.

She was diagnosed with type 1 diabetes mellitus (T1DM) at the age of 5 in 2007, and her condition is now complicated by lipohypertrophy and gastroparesis. Possible DKA precipitants include migraines, coryzal and urinary symptoms. Her current HbA_{1c} is 116 mmol/mol, using an MDI (multiple doses of insulin) regime of Tresiba 28 units OD and prandial Fiasp 14/14/7. Nominally her insulin sensitivity factor (ISF) is 1 unit: 3 mmol/L, and insulin to carbohydrate ratio (ICR) 1 unit: 10g CHO.

A pattern has developed whereby she does not attend appointments or answer the phone. She uses the Freestyle Libre 2, but cites sensor issues and error messages for lack of readings; at best, time in range is 43%, and at other times it is less than 10% with scans 0-4 times daily. A DAFNE graduate, she has hitherto declined the offer of a refresher, and has features of Mauriac syndrome (glucogenic hepatopathy).

Mental health: The patient lives at home with her mother and six siblings, with no safeguarding concerns raised. Her background was mainstream schooling, and she is currently on a gap year. The MDT resolved to offer urgent follow-up in the transition clinic, and to involve psychology and liaison psychiatry colleagues. Mental health assessments when she was an inpatient recovering from ketoacidosis did take place; she was signposted to self-refer for talking therapies (IAPT) and a crisis card was provided. There was no history of psychiatric admissions or body dysmorphic symptoms, but she had exhibited previous self-harming behaviours (duloxetine overdose and bleach consumption). She had been referred to CAMHS by the paediatric team, but did not attend.

The impression was one of untreated depression, and accordingly she was trialled on mirtazapine 15mg whilst under the care of the Home Treatment Team (HTT). Her glycaemic control was better during this period, but mirtazapine was subsequently discontinued due to daytime somnolence. Detention under the MHA/MCA was not felt to be in her best interests and she was discharged with support from the HTT for a month. During a subsequent assessment, note was made of her apathy and ambivalence towards diabetes monitoring and self-management, and of the disconnect between her emotions and the seriousness of repeated DKAs, but no intent to self-harm was found. She has attended the specialist diabetes/psychiatry clinic regionally but is no longer agreeable to any input from mental health services and she declines blood tests when she is admitted.

Discussion points:

1. How can the patient be further supported?
2. What is the role of peer support?
3. Moral injury on healthcare staff?
4. Capacity

Abstract ID: 54

Hyperosmolar hyperglycaemic state secondary to steroid use in a neuro-oncology patient

Patel H, Osman N, Gaoatswe G

Newham Hospital, Bart's Health Trust

A 70-year-old man presented to the emergency department with confusion, reduced level of consciousness and increased urinary frequency. He had a background of lung cancer, which had been previously treated with a lobectomy and radiotherapy. He had recently been diagnosed with brain metastases after presenting with loss of proprioception and ataxia. Dexamethasone 8mg twice daily was started shortly thereafter. Other past medical history included type 2 diabetes (T2DM), treated with metformin with a most recent HbA_{1c} of 43 mmol/mol.

On examination he was confused, tachypnoeic and unsteady on his feet. His observations were otherwise stable. Initial blood tests showed a lactate of 4.6mmol/L and a glucose of 44mmol/L. His calculated serum osmolality was 364mmol/kg and ketones were 1.1 mmol/L. Antibiotics were started for urinary tract infection in the context of a positive urine dipstick. A CT brain scan was completed given his confusion in the context of brain metastases; it demonstrated no interval change.

Hyperosmolar hyperglycaemic state (HHS) was confirmed, and fluid replacement was commenced as per local guidance. Fixed rate insulin infusion (FRII) was started at 1 unit/hour due to his serum ketones increasing to 2.7 mmol/L.

Possible triggers of HHS were considered, including dexamethasone. As a result, neurosurgical advice was sought in the context of the clinical picture and investigation results. On review of the CT imaging and stable neurology with minimal cerebral swelling, advice was given for gradual reduction in the dexamethasone dose to 2mg once daily.

Over the next 72 hours, fluid resuscitation and low dose FRII were continued, resulting in a steady improvement in his clinical condition, with improvements in level of consciousness and serum osmolality. Significant improvement was seen at day 5 of the admission; the patient was more alert, serum osmolality was 320mmol/kg and the dexamethasone had been weaned to 4mg twice daily.

Despite the improvement in the clinical picture, the patient developed worsening right-sided neurology symptoms, noting reduced sensation over the right side of the face, arm and leg. Power was reduced in all muscle groups of the right arm. An MRI of the head showed no change compared to previous CT scans. Despite improvements in serum osmolality to 295mmol/kg, right-sided neurology continued to deteriorate.

Neurosurgical advice was sought, and the worsening neurology was attributed to the improvement in serum osmolality. The physiological changes occurring in HHS relating to hyperosmolality were likened to the mechanism of mannitol. With the improvement of HHS, neurosurgical advice was to increase the dexamethasone with careful monitoring of his glycaemic control.

Abstract ID: 55

A case of monogenic diabetes

Yu T, Huda B

The Royal London Hospital, Barts Health NHS Trust

Introduction: Monogenic diabetes, or maturity onset diabetes of the young (MODY), is a rare form of diabetes diagnosed at a young age with autosomal dominant transmission and lack of autoantibodies.[1]

We present a case of monogenic diabetes. A 25-year-old Bangladeshi woman was referred to our diabetes clinic in 2015 following an incidental finding of high glucose on fingerprick testing and an HbA_{1c} of 73mmol/mol. Her father was diagnosed with type 2 diabetes (T2DM) at the age of 48 and treated with metformin. Her mother was diagnosed with type 1 diabetes (T1DM) at the age of 20 and died from a road traffic accident at the age of 32. Her maternal aunt has T1DM, treated with insulin.

The patient was asymptomatic on referral, with a BMI of 27kg/m^2 and no obvious past medical history. Triple antibodies testing was negative, with C-peptide 931pmol/L and insulin 25.2mu/L, indicating that she is not insulin-deficient. She underwent MODY genetic testing, which revealed the HNF4 alpha mutation. She was treated with gliclazide 40mg OD and has had a good response. She has two brothers who are not known to have diabetes and they have declined genetic testing. She works in a nursery and does not smoke or drink. She has recently married and is planning for pregnancy. Her glycaemic control is currently satisfactory, with a recent HbA_{1c} of 41 mmol/mol and no complications of diabetes. She has been on a statin for high cholesterol but has no other risk factors. She has received standard preconception advice for women with diabetes, but there was a more nuanced discussion around therapy during any future pregnancy. At present we have elected to continue with gliclazide but may need to consider insulin therapy later in pregnancy.

Learning points: Genetic testing should be performed for an atypical form of diabetes, with a high index of suspicion.[2]

Optimal treatment and risk of diabetic complications varies with the underlying genetic defect.[3] In this case, glycaemic control is currently well managed with sulphonylurea monotherapy. Recent guidelines have suggested that insulin therapy may be preferable in pregnancy but there is an argument for continuing sulphonylurea through the early part of pregnancy.

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Abstract ID: 56

Case of management of HNF1A-MODY before, during and after pregnancy

Smout V, Bawlchhim Z
Royal Surrey County Hospital

A female patient was diagnosed with HNF1A-MODY as a teenager due to family history. Gliclazide was started and gradually titrated up to 80mg in the morning and 160mg in the evening. By age 30 the patient's HbA_{1c} was rising again so Lantus was initiated and titrated to 7 units daily.

The HbA_{1c} improved to 45 mmol/mol and she started planning a pregnancy. Folic acid was started and a change of gliclazide to glibenclamide was recommended. The change in medication did not occur, however, due to difficulties with prescribing it at the general practice.

The patient soon became pregnant and gliclazide was stopped. In the first few weeks she noted that pre-meal glucose readings were raised, so the Lantus dose was increased to 9 units and NovoRapid was added with meals at 1unit:8g carbohydrate. She remained stable on these doses during the rest of the pregnancy. Ultrasound scans showed normal foetal growth, with an abdominal circumference in the 50th centile.

At 36 weeks the patient reported increasing frequency of hypoglycaemia. Her doses of insulin were reduced but a few days later she was still experiencing hypoglycaemia of 3.1 mmol/L despite completely stopping her insulin. At 37+2 weeks she had a Category 2 Caesarean section, delivering a boy weighing 7lb 9oz. She and the baby made an uncomplicated recovery and were discharged home. The patient did not continue any diabetes medication at this point.

While breastfeeding, she was advised to restart Novorapid only, at 5-6 units with meals, as she noticed glucose readings rising to 11-12 mmol/L after eating but normal fasting levels. Once she had stopped breastfeeding, Lantus was restarted at 2 units daily as readings were higher overnight. Sulfonylurea may be restarted at a later stage, depending on her plans for further pregnancies.

Management of monogenic diabetes pre-conception, during pregnancy and in the postnatal period presents various challenges, depending on the type of MODY. HNF1A-MODY is sensitive to treatment with sulfonylurea medication and there is some evidence that glibenclamide may be used in the first two trimesters but it carries risk of macrosomia if continued later in the pregnancy. There are few specific guidelines on the management of different forms of MODY in pregnancy, including the timing of sulfonylurea and insulin use, and it is important these cases are managed in a multidisciplinary setting with regular review before, during and after pregnancy.

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Abstract ID: 57

Acute Charcot neuroarthropathy: a case of rapid progression with unusual presentation of lymphoedema and hypercalcemia

Thayyil S, Prouten J, Rippin J
Department of Diabetes and Endocrinology, Northampton General Hospital NHS Trust, NN1 5BD

A 61-year-old man with a seven-year history of type 2 diabetes (T2DM) was admitted to the hospital with septicaemia of unknown origin in October 2021. His right lower leg and foot were swollen and red without any ulceration and the temperature difference between the right and left side was 6 degrees Celsius. His glycated haemoglobin level (HbA_{1c}) was 55 mmol/mol on linagliptin 5mg alone. He had peripheral neuropathy, drank excess alcohol, and had chronic kidney disease (CKD) 3 and left eye maculopathy. MRI foot showed features of osteomyelitis and Charcot neuroarthropathy of the right midfoot. He developed massive lymphoedema of the right leg, which made offloading a huge challenge; a total contact cast was not an option for him. Further investigations confirmed neuropathy of the feet and extensive calcification of all lower limb arteries with triphasic flow of distal arteries. He was on a Scotchcast boot for offloading, along with a compression dressing, which helped to reduce the lymphoedema. His bone culture was negative, and the antibiotics were stopped.

Interestingly, his bone profile showed a high corrected calcium level of 3.3 mmol/L with suppressed parathyroid hormone (PTH). Although this was extensively investigated, no secondary causes were detected. Subsequent X-ray two weeks later showed osteolytic changes in the midfoot involving the navicular, cuboid and all the cuneiform bones, resulting in reversal of the arch. A diagnosis of

hypercalcemia secondary to rapid bone resorption was made. IV bisphosphonate helped to stabilize his calcium levels.

Four months later, he developed a plantar ulcer of the right foot which required another course of antibiotics based on culture results. Sepsis along with the long course of antibiotics caused extensive liver damage. Moreover, he was deconditioned by ongoing sepsis with progressive osteolytic changes on imaging. The foot multidisciplinary team (MDT) advised him to have a below-knee amputation eight months into his diagnosis of Charcot's joint to avoid life-threatening septicaemia.

Learning points:

- Acute Charcot neuroarthropathy is characterised by progressive degeneration of weight-bearing joints, resulting in acute fractures, dislocations and joint destruction[1]
- A temperature difference of 2°C from the contralateral foot is an indicator of active Charcot neuroarthropathy[1,2]
- Increased blood flow in the presence of autonomic neuropathy accelerates osteoclast activity and bone demineralization[3]
- Repeated microtrauma of the foot affected with peripheral neuropathy precipitates the development of Charcot foot[4]
- An ulcer larger than 2 cm^2 and where bone can be visualised increases the risk of osteomyelitis[3,5]
- High calcium with suppressed PTH in diabetes patients could be due to increased bone resorption and should raise suspicion of Charcot foot[3]
- Offloading through total contact casts or irremovable or removable casts is the mainstay treatment of Charcot neuroarthropathy[1,6]

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