ACADEMIC: CASE REPORT

From type 2 to type 1 diabetes: A case report on the diagnostic challenges in an older adult

Siew Wei Han, George Tang, John Kok

Abstract

Type 1 diabetes (T1D) is an autoimmune disease that targets the pancreatic beta cells, leading to insulin deficiency. Meanwhile, Type 2 diabetes (T2D) is primarily characterised by insulin resistance leading to decreased insulin production. Although younger individuals are commonly affected by T1D, an increasing number of cases are being reported in older adults. The overlapping features of T1D and T2D can make distinguishing between them difficult. Misdiagnosis of T1D as T2D may cause a delay in insulin administration, increasing the risk of diabetic ketoacidosis (DKA). This case report highlights the diagnostic challenges in identifying T1D in a 72-year-old female, referred to as Mrs Q, who presented with hyperglycaemic symptoms. On assessment in the emergency department, Mrs Q was initially diagnosed with T2D based solely on her blood glucose reading. She was commenced on oral hypoglycaemic agents; however, her blood glucose level remained elevated despite therapy. Given her poor response to the medications, further evaluation was undertaken. An antibody panel revealed that Mrs Q was positive for anti-glutamic acid decarboxylase and islet antigen 2 antibodies, confirming the diagnosis of T1D. Insulin was promptly administered to prevent the onset of diabetic ketoacidosis. This case report highlights the importance of keeping T1D as a differential diagnosis for older adults who present with hyperglycaemia, to ensure the right treatment approach is undertaken and to avoid adverse outcomes such as DKA.

Background

The 2023 Virtual Diabetes Register in New Zealand reported that 323,700 people in the population are diabetic; this figure is projected to increase by 7% each year.[1,2] It is estimated that 10% of people with diabetes in New Zealand have type 1, whilst 90% have type 2; however, there is limited data on the exact proportions.[3] Type 2 diabetes (T2D), accounting for 90% of global diabetic cases, is primarily characterised by insulin resistance, which eventually leads to decreased insulin production.[4] Meanwhile, type 1 diabetes (T1D), accounting for 5% to 10% of the population living with diabetes, is characterised by the presence of antibodies such as anti-glutamic acid decarboxylase (GAD) and islet antigen 2 (IA2) that target the pancreatic beta cells, leading to insulin deficiency.[5] Although T1D most commonly occurs during puberty and early adulthood, its prevalence in older adult is increasing with more men than women affected.[6,7]

Genetic factors strongly associated with T1D include the human leukocyte antigen (HLA) and insulin gene, especially HLA-DR and HLA-DQ.[8] Meanwhile, T2D is a polygenic condition determined by numerous variants and a combination of genetic and environmental factors.[9] Distinguishing between T1D and T2D remains challenging due to overlapping symptoms. Over 40% of the adults who develop T1D after the age of 30 were initially diagnosed with T2D.[5]

The mainstay treatment for T1D is the immediate administration of insulin, while T2D may be managed with oral hypoglycaemic agents.[5,10] The initial diagnosis of T1D as T2D could delay insulin administration, resulting in development of diabetic ketoacidosis (DKA).[11] This case report highlights the challenges in diagnosing T1D in an older adult who was initially diagnosed with T2D.

Case

Mrs Q, a 72-year-old female, attended a general practitioner (GP) appointment on 24th December 2024 for a follow-up on her blood test results from the previous day. She had a month-long history of polydipsia, fatigue, and had lost 5 kg unintentionally over this period. Her current weight and height were 50kg and 1.48m, respectively (body mass index: 22.8 kg/m2). Her blood test results revealed low eGFR, high anion gap, and hyperglycaemia, as highlighted in Table 1. Concerned she might have DKA, the GP referred her to the emergency department (ED).

Mrs Q's past medical history included hypertension, dyslipidaemia, insomnia and osteoporosis, managed by candesartan, atorvastatin, temazepam and denosumab, respectively. She had no prior history of gestational diabetes nor family history of diabetes. Her last fasting blood glucose level was 5.2 mmol/L in June 2024. She lived with her daughter and had an active lifestyle. She did not smoke or drink.

Upon examination in the ED, she appeared well-hydrated, and her vital signs were normal. Venous blood gas (VBG) results did not show severe acidosis, with the readings highlighted in Table 2. The ED physician excluded a diagnosis of DKA, diagnosed Mrs Q with T2D, and prescribed her metformin 500mg twice daily. Mrs Q was discharged with instructions to follow up with her GP within a week.

Mrs Q attended a follow-up appointment with her GP on 28th December 2024. She had another blood test the previous day, which showed an elevated glucose level of 19.1 mmol/L, a borderline normal fasting insulin level of 3.1 mIU/L (reference range: 3.0 – 25.0 mIU/L), and an elevated HbA1c level (NGSP 14.4%, IFCC: 134.0 mmol/mol). Mrs Q's limited response to metformin prompted the GP to refer her

Table 1: Mrs Q's blood test results on 23rd December 2024.

Test	Readings	Reference range*	Low / High
Fasting glucose	22.3 mmol/L	3.0 – 5.5 mmol/L	High
Anion gap	20.0 mmol/L	8.0 – 19.0 mmol/L	High
eGFR	39 mL/min/1.73m^2	>59 mL/min/1.73m^2	Low
Creatinine	120 µmol/L	45 – 90 µmol/L	High
Creatinine kinase	153 U/L	30 – 150 U/L	High
Ferritin	418 µg/L	30 – 300 µg/L	High
Insulin (fasting)	3.1 mIU/L	3.0 – 25.0 mIU/L	Normal

** Reference ranges are those of the NSWHP Hospital Laboratory Clinical Projects Laboratory Manual or, where provided, the patient's own test report.[12] This applies to all reference intervals shown in subsequent tables.*

to an endocrinologist. As Mrs Q reported nausea after taking metformin, the GP prescribed her empagliflozin 10 mg and discontinued metformin.

Mrs Q had a subsequent blood test on the 30th December 2024, which also showed elevated glucose (19.3 mmol/L) and ketone levels (6.2 mmol/L) despite taking empagliflozin. She attended her appointment with the endocrinologist the next day. Based on her limited response to empagliflozin, the endocrinologist gave a provisional diagnosis of T1D. Concerned about DKA, the endocrinologist referred her to the ED.

In the ED, medical staff obtained another VBG and further blood tests. The results were consistent with possible early DKA, as shown in Table 3. This prompted the medical team to admit Mrs Q for further investigations, including an antibody panel, C-peptide level and HbA1c test. The results, shown in Table 4, revealed an elevated HbA1c level and the presence of GAD and IA2 antibodies, consistent with the diagnosis of T1D.

Mrs Q was treated with intravenous fluids and insulin in the ED, and her vital signs were continuously monitored. After her blood glucose levels and VBG results improved, she was discharged home. Empagliflozin was ceased, and Mrs Q was prescribed insulin aspart (rys), 14 units (30% soluble, 70% protamine crystallised), to be administered subcutaneously twice daily.

Mrs Q represented to the ED on 2nd January 2025, due to concern about her blood glucose reading of 4.1 mmol/L following an additional insulin dose before bed. Her vital signs were stable at the ED. The medical team discharged her after counselling on correct insulin administration and instructing her to follow up with her GP within a week (Figure 1).

Follow-up

The GP referred Mrs Q to a local optometrist, podiatrist, and diabetes educator to screen for diabetic retinopathy and neuropathy. She was also provided with education regarding the proper use of insulin. The GP also arranged for Mrs Q to have regular follow-up appointments with the endocrinologist and reminded Mrs Q to present to the ED if she developed any alarming symptoms.

Discussion

The ED physician initially diagnosed Mrs Q with T2D. However, the endocrinologist's assessment favoured a diagnosis of T1D, based on Mrs Q's lack of response to empagliflozin and the acute presentation of hyperglycaemia. Table 5 compares features of T1D to T2D, some of which can overlap and complicate distinction.

Latent autoimmune diabetes in adults (LADA) is another class of diabetes that shares similar phenotypic features with T1D and T2D.[17] Patients with LADA are found to be positive for GAD and IA2 antibodies, resembling T1D.[17] However, these patients have an increased risk of developing metabolic syndrome and insulin resistance.[17] There-

Table 2: VBG and blood chemistry results at ED on 24th December 2024.

Test	Readings	Reference range	Low / High
Random glucose	30.0 mmol/L	3.5 – 5.4 mmol/L	High
pH	7.31	7.30 – 7.40	Normal
pCO2	47.6 mmHg	40.0 – 50.0 mmHg	Normal
HCO_3^-	25.0 mmol/L	22.0 – 32.0 mmol/L	Normal
Ketone	4.9 mmol/L	< 0.6 mmol/L	High
Urea	10.5 mmol/L	3.0 – 10.0 mmol/L	High
Anion gap in blood chemistry	25.7 mmol/L	8 – 19 mmol/L	High
Sodium level in blood chemistry	135.0 mmol/L	135.0 – 145.0 mmol/L	Normal
Potassium level in blood chemistry	5.7 mmol/L	3.5 – 5.2 mmol/L	High
Chloride level in blood chemistry	91.0 mmol/L	95.0 – 110.0 mmol/L	Low
HCO_3^- level in blood chemistry	24.0 mmol/L	22.0 – 32.0 mmol/L	Normal

Table 3. VBG and blood chemistry results at ED on 31st December 2024.

Test	Readings	Reference range	Low / High
Random glucose	25.0 mmol/L	3.5 – 5.4 mmol/L	High
pH	7.29	7.30 – 7.40	Low
pCO2	41.8 mmHg	40.0 – 50.0 mmHg	Normal
HCO_3^-	21.9 mmol/L	22.0 – 32.0 mmol/L	Low
Ketone	4.2 mmol/L	<0.6 mmol/L	High
Urea	10.5 mmol/L	3.0 – 10.0 mmol/L	High
Anion gap in blood chemistry	24.6 mmol/L	8.0 – 19.0 mmol/L	High
Sodium level in blood chemistry	132.0 mmol/L	135.0 – 145.0 mmol/L	Low
Potassium level in blood chemistry	4.6 mmol/L	3.5 – 5.2 mmol/L	Normal
Chloride level in blood chemistry	91.0 mmol/L	95.0 – 110.0 mmol/L	Low
HCO_3^- level in blood chemistry	21.0 mmol/L	22.0 – 32.0 mmol/L	Low

Table 4: Endocrinology panel, C-peptide level and HbA1c test results on 1st January 2025.

Test	Readings	Reference range	Low / High
HbA1c NGSP	15.3 %	4.0 – 6.0 %	High
HbA1c IFCC	143.0 mmol/mol	20.0 – 42.0 mmol/mol	High
GAD antibodies	>2000 U/ml	<5 U/ml	High
IA2 antibodies	58 U/ml	<15 U/ml	High
C peptide	286 pmol/L	370 – 1470 pmol/L	Low

Table 5: Comparison of features of T1D and T2D. [4,5,11,12,13,14]

	T1D	T2D
Age	Most common in puberty and early adulthood[5]	Most common in adults but increasingly common among younger generations due to sedentary lifestyles, high-calorie diets, and obesity[4]
Aetiology	Autoimmune process leading to pancreatic beta cell destruction and insulin deficiency[13]	Insulin resistance or insulin secretory defect with relative insulin deficiency[13]
Body mass index	Traditionally more common in lean people, but increasingly observed in those who are overweight and obese[13]	Most common for those who are overweight, obese, or have central obesity[14]
Family history of diabetes	Uncommon, 5%–10% of cases[15]	High likelihood, 75%–90% of cases[15]
Antibodies	Usually present with GAD and IA2 antibodies[15]	Usually autoantibody negative[15]
Insulin secretion	Absent[16]	Present but decreases over time[16]
Risk of ketosis and DKA	Increased risk[14]	Infrequent, mostly stress-induced[14]

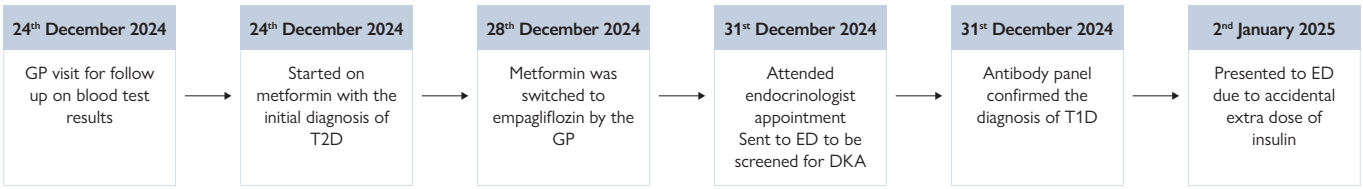

Figure 1: Case progression timeline for Mrs Q.

fore, management of LADA is similar to that of T2D, involving lifestyle modification and oral hypoglycaemic agents.[14] However, patients with LADA tend to deteriorate faster than those with T2D, which warrants the earlier use of insulin.[14] There are no universal guidelines for diagnosing LADA. Diagnosis is made based on the presence of GAD antibodies, the patient being older than 35 years, and the need for insulin therapy is often beyond six months after diagnosis.[14] In Mrs Q's case, her rapid deterioration of insulin level and her lack of response to empagliflozin favoured the diagnosis of T1D over LADA. She required immediate administration of insulin.

T1D occurs in three stages. Patients are usually asymptomatic in the first two stages and present with symptomatic hyperglycaemia in stage three.[18] In stage one, patients typically have normal glucose readings, as this is the initial stage of autoimmune beta-cell destruction and insulitis.[18] In stage two, significant destruction of beta cells causes derangement in glucose and HbA1c measurements.[18] Finally, in stage three, patients experience symptomatic hyperglycaemia with elevated random glucose readings above 11.1 mmol/L.[18] Mrs Q's presentation is consistent with stage three of T1D, as she was symptomatic with elevated glucose readings and HbA1c level, as shown in Tables 3 and 4.

The delay in insulin administration for Mrs Q increased her risk of developing DKA. Her VBG results on 24 December 2024 in the ED showed high ketone and anion gap levels but did not show severe acidosis, with borderline normal pH and bicarbonate levels, as shown in Table 2. The elevated anion gap may have reflected compensated ketosis. DKA is diagnosed based on the triad of hyperglycaemia (glucose greater than 13.9 mmol/L), metabolic acidosis (pH <7.3 or serum HCO3 <18 mmol/L and an anion gap >10 mmol/L), and elevated ketone levels.[19] Since Mrs Q appeared well and had normal vital signs, the ED physician excluded the diagnosis of DKA. However, her VBG results on 31 December 2024 in the ED indicated a possible mild DKA with her low pH, low bicarbonate, and high ketone and anion gap levels, as shown in Table 3. This prompted the medical team to admit Mrs Q for further investigation and administer IV fluids.

The initial misclassification of diabetes as T2D instead of T1D can arise because of an assumption that adults developing diabetes have T2D, and development of T1D is limited to children.[20,21] The AA, BB, and CC method is a mnemonic reminder used to aid clinicians in diagnosing T1D. It reminds clinicians to consider a patients' age, presence of antibodies, body mass index, background, insulin control, and comorbidities when assessing patients with hyperglycaemic symptoms.[22]

Clinical features highly suggestive of T1D instead of T2D include a diagnosis at the age of less than 40 years, a low body mass index (<25 kg/m2), and a rapid need for insulin therapy.[22] For Mrs Q, the presentation of T1D was atypical for her age. This explains why the ED physician initially made a diagnosis of T2D instead of T1D, as T2D is more common for patients of her age and presentation.

The ED physician initially prescribed metformin for Mrs Q, but the GP subsequently switched her to empagliflozin due to nausea and vomiting after taking metformin. Despite commencing on these oral hypoglycaemic agents, Mrs Q's glucose level remains elevated. Further assessment changed her diagnosis from T2D to T1D, and insulin therapy was initiated immediately to lower her blood glucose level. However, controlling the blood glucose level of older adults can be challenging due to their dietary patterns and age-related physiological changes, necessitating tailored diabetic care plans that consider their ability to manage complex insulin regimens.[16,23] In Mrs Q's case, a twice-daily regimen of premixed short and long-acting insulin was prescribed instead of a basal-bolus insulin regimen. This simple regimen was considered more manageable for her.

Mrs Q's case also highlights the need to provide older patients with proper counselling on insulin therapy. Optimal insulin education requires healthcare professionals to use a step-by-step approach to explain the correct methods of insulin administration and perform a follow-up assessment to check the patient's understanding.[24] In most individuals, a 4 mm pen needle inserted at 90° ensures successful insulin injection into the subcutaneous tissue.[24] Other important topics to discuss with patients include the components of the insulin devices and the steps that can be taken to reduce the risk of lipodystrophy.[24] Patients should also be advised to avoid reusing needles or using expired insulin injections.[24]

Older adults with T1D are at risk of developing hypoglycaemia, particularly if they have concurrent cognitive decline.[25] Novel interventions can mitigate this risk, such as automated insulin delivery systems and hybrid closed-loop systems.[25] Automated insulin delivery systems temporarily halt insulin delivery from an insulin pump when patients are close to developing hypoglycaemia.[25] Meanwhile, advanced hybrid closed-loop systems can adjust the insulin doses for patients with hyperglycaemia and suspend insulin delivery when patients are close to being hypoglycaemic.[25]

The case report has limitations. Firstly, a single-patient case report has low generalisability and may not represent all patients presenting with hyperglycaemic symptoms. Furthermore, no formal interview was conducted to gain insight into Mrs Q's experience with T1D and her perspective on the treatment plan. Nevertheless, this report has relevance for GPs, ED physicians, endocrinologists, and medical students, highlighting the need to consider T1D as a differential for older adults with hyperglycaemia, rather than assuming T2D by default. It also emphasises the importance of providing comprehensive insulin education and simpler regimens for older adults with T1D.

Conclusion

Mrs Q, an older adult presenting with hyperglycaemia, was initially diagnosed with T2D and given oral hypoglycaemic agents. Her inadequate response to the agents prompted further assessment, ultimately leading to a diagnosis of T1D. Her case highlights the importance of T1D as a differential for adults presenting with hyperglycaemic symptoms. Misdiagnosis of T1D as T2D can result in prolonged hyperglycaemia, delayed insulin administration, and an increased risk of developing DKA.

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