DOI: 10.1002/ccr3.9301

CASE REPORT

Obesity as a misleading factor in the diagnosis of type 1 diabetes mellitus: A case report

| Olgierd Dróżdż ¹ | Wiktoria Bińczyk ¹ 💿 | Bartosz Siudek ² | Katarzyna Błaszczyszyn ¹ | |
|---------------------------------|---------------------------------|-------------------------------|-------------------------------------|--|
| Filip Jan Grajnert ³ | Michalina Zofia Grz | elka ³ Maciej Ra | bczyński ¹ | |

¹Department and Clinic of Diabetology and Internal Medicine, University Teaching Hospital, Wrocław, Poland

²T. Marciniak Specialist Hospital, Wrocław, Poland

³4th Military Clinical Hospital, Wrocław, Poland

Correspondence

Wiktoria Bińczyk, Department and Clinic of Diabetology and Internal Medicine, University Teaching Hospital, Wrocław 50-556, Poland. Email: wiktoria.binczyk98@gmail.com

Key Clinical Message

A comprehensive evaluation, including symptoms, medical history, C-peptide levels, and anti-GAD antibodies, is essential for distinguishing between diabetes' types, particularly in cases of diagnostic uncertainty. While obesity is often associated with T2DM, BMI should be viewed as a factor rather than a criterion for the exclusion of diabetes type.

K E Y W O R D S

c-peptide, misdiagnosis, obesity, type 1 diabetes mellitus

1 | INTRODUCTION

Diabetes mellitus is a significant global health concern, with an increasing prevalence. While type 2 diabetes mellitus (T2DM) remains the predominant form, the detection of type 1 diabetes mellitus (T1DM) is crucial, as the risk of serious complications is high if not promptly identified and treated.¹ Although the two types of diabetes have different causes, differentiating T1DM from T2DM can be challenging, particularly in adults. While obesity is often associated with T2DM, it does not necessarily exclude the possibility of developing T1DM.² This case describes a 40-year-old man who was initially diagnosed with T2DM and later developed symptoms suggestive of T1DM. The objective of this study is to illustrate the complexities of diabetes diagnosis and to highlight the significance of comprehensive clinical assessment in guiding appropriate treatment decisions, particularly when addressing the diagnostic challenges associated with obesity. Previous research has demonstrated that recognizing the significance of timely diagnosis and

management is crucial for patient outcomes, as evidenced by the implications of diabetes misdiagnosis.³

2 | CASE HISTORY/ EXAMINATION

A 40-year-old male presented to the Emergency Medicine Department with a two-day history of increased thirst, polyuria, and fatigue. The patient appeared dehydrated with dry mucous membranes. No other abnormalities were found during the physical examination. The patient's height was 198 cm, body weight 76 kg, and BMI=19.39 (normal body weight). A confirmed history of type 2 diabetes mellitus (T2DM) was noted, diagnosed 1 year prior. At the time of initial presentation, the patient exhibited symptoms of polyuria, polydipsia, fatigue, and frequent urinary tract infections. Upon diagnosis, the patient's height was 198 cm, body weight 126 kg, and BMI=32.14, indicating first-degree obesity. The patient was initially treated with oral antidiabetic therapy, starting with metformin and

This is an open access article under the terms of the Creative Commons Attribution-NonCommercial License, which permits use, distribution and reproduction in any medium, provided the original work is properly cited and is not used for commercial purposes. © 2024 The Author(s). *Clinical Case Reports* published by John Wiley & Sons Ltd. WILEY_Clinical Case Reports

later adding dapagliflozin. Despite these interventions, adequate glycemic control was not achieved, as evidenced by postprandial glycemia levels consistently above 300 mg/ dL. In addition, the patient experienced an unintentional weight loss of 50 kg within 6 months. The family history of diabetes was negative. At the time of admission, complications of diabetes such as retinopathy, nephropathy, and neuropathy were absent.

3 | METHODS

Laboratory tests performed on the emergency department revealed a markedly elevated capillary blood glucose measurement of 527 mg/dL (see Table 1). Urinalysis was positive for glucosuria (1000 mg/dL) and severe ketonuria. No signs of infection were observed. The patient exhibited a glycated hemoglobin (HbA1c) level of 15.5%. An abdominal ultrasound was also performed, which did not reveal any abnormalities in the pancreas and other organs.

4 | CONCLUSIONS AND RESULTS

Given the patient's significant weight loss and decreased C-peptide levels (0.41 ng/mL), it was suspected that the initial diagnosis of T2DM may have been incorrect. Based on these findings, the patient was diagnosed with latent autoimmune diabetes of adults (LADA)—a subtype of type 1 diabetes mellitus (T1DM). Immediate treatment was initiated with intensive insulin therapy using basal insulin glargine and prandial insulin lispro. Over the course of an eight-day hospital stay, a trend toward normalization of glycemia was observed. A subsequent hospitalization was planned to educate the patient on carbohydrate counting and to introduce the continuous glucose monitoring system.

| TA | BL | Е | 1 | Initial | la | boratory | investigatio | ons. |
|----|----|---|---|---------|----|----------|--------------|------|
|----|----|---|---|---------|----|----------|--------------|------|

| Parameter | Reference | Admission |
|------------------------------------|-----------|-----------|
| Venous pH | 7.32-7.43 | 7.41 |
| pCO ₂ (mmHg) | 35–45 | 49.2 |
| pO ₂ (mmHg) | 25-40 | 34.7 |
| Bicarbonate (mmol/L) | 20-28 | 28.7 |
| Anion Gap (mmol/L) | 8-12 | 6.7 |
| Glucose (mg/dL) | 65–95 | 527 |
| EGFR (mL/min/ 1.73m^2) | >90 | 93 |
| Sodium (mmol/L) | 136–145 | 135 |
| Potassium (mmol/L) | 3.4-4.5 | 3.7 |
| Lactic acid (mmol/L) | 0.5-1.6 | 0.95 |
| Creatinine (mg/dL) | 0.73-1.18 | 0.95 |

The differentiation between the two main forms of diabetes is of crucial importance in initiating a treatment that is both efficient and effective. In order to make an informed decision, it is essential to take into consideration a number of factors, such as the patient's symptoms, recent weight loss, their medical history, their levels of C-peptide, as well as the presence of anti-GAD antibodies. While BMI assessment should be taken into account, it should not be regarded as a criterion that excludes type 1 diabetes. Rather, it should be regarded as a factor that increases the likelihood of type 2 diabetes. The initial misdiagnosis of T2DM in our obese patient, who was later confirmed to have T1DM, highlights the necessity for meticulous evaluation and consideration of alternative diagnoses, even in cases with seemingly typical clinical presentations.

This case illustrates the importance of timely and accurate diagnosis in achieving optimal patient outcomes. Prompt identification of the correct diagnosis enabled the initiation of appropriate intensive insulin therapy, resulting in a positive trend toward glycemic normalization and improved management of the patient's condition.

5 | DISCUSSION

General criteria for diagnosing diabetes are common to all its types and include: fasting blood glucose of at least 126 mg/dL measured twice on different days, or blood glucose at 2h in OGTT of at least 200 mg/dL, or hemoglobin A1c level equal to or above 6.5%, or blood glucose of at least 200 mg/dL with accompanying symptoms of hyperglycemia. However, these criteria do not differentiate between individual types of diabetes. In the clinical picture of type 1 diabetes, sudden onset of symptoms predominates, often in the form of diabetic ketoacidosis, and a young age at diagnosis. Moreover, confirmation of the diagnosis may be aided by evaluating C-peptide secretion and the presence of anti-GAD antibodies. On the other hand, type 2 diabetes is associated with obese patients in adulthood or old age.⁴ This line of thinking is certainly supported by the fact that in reality, obesity and significant accumulation of adipose tissue exacerbate insulin resistance, thereby contributing to the development of type 2 diabetes.⁵

Furthermore, it is imperative to take into account the existence of additional antibodies linked to T1DM. Although anti-GAD antibodies are frequently employed in the diagnosis of T1DM, there are other autoantibodies that can offer further diagnostic insight. Such antibodies include insulin autoantibodies (IAA), insulinomaassociated-2 autoantibodies (IA-2A), and zinc transporter 8 autoantibodies (ZnT8A). Each of these antibodies targets a distinct component of pancreatic beta cells and can

-WILEY

be present in varying combinations in individuals with T1DM. For example, IA-2A antibodies are highly specific for T1DM and are often associated with a more rapid progression to insulin dependency. ZnT8A have been demonstrated to manifest in the initial stages of T1DM and are especially valuable in diagnosing LADA. The detection of these antibodies, in conjunction with an assessment of the clinical presentation and C-peptide levels, can markedly enhance the accuracy of the diagnosis and facilitate the initiation of appropriate treatment.^{6,7}

Latent Autoimmune Diabetes of Adults (LADA) is a form of diabetes that shares characteristics with both type 1 and type 2 diabetes. Often diagnosed in adults over the age of 30, LADA is characterized by the gradual onset of autoimmune destruction of insulin-producing beta cells in the pancreas, similar to type 1 diabetes. However, unlike classic type 1 diabetes, the progression of beta-cell failure in LADA is slower, and patients may initially respond to oral hypoglycemic agents used in type 2 diabetes treatment. Over time, individuals with LADA typically become insulin-dependent as the autoimmune process leads to significant beta-cell loss. A particularly helpful test for diagnosing the LADA subtype is the assessment of anti-GAD antibody levels. The significance of this test is underscored by the development of rapid immunochromatographic tests, which enable the detection of elevated levels of these antibodies in the asymptomatic stage of diabetes. This allows for appropriate prevention of potential decompensation, such as diabetic ketoacidosis.8,9

In its 2023 guidelines, the ADA addresses obesity in type 1 diabetes, noting that it should not preclude the need for further diagnostic investigation into type 1 diabetes.⁴ However, in the case described, the opposite situation occurred. A 40-year-old man was initially diagnosed as having type 2 diabetes. He presented symptoms of hyperglycemia, which can occur in both type 1 and 2. Despite being less common, the onset of type 2 diabetes in the form of ketoacidosis is possible.⁴ Additionally, the patient's BMI, which was over 30 kg/m2, indicating first-degree obesity, favored type 2 diabetes.

Current symptoms could have been interpreted as decompensated type 2 diabetes. However, a detailed medical history, a hemoglobin A1c result indicating severely uncontrolled diabetes, and a decrease of C-peptide secretion led to a decision to change the diagnosis to type 1 diabetes. Intensive insulin therapy was initiated, and a tendency toward normalization of blood glucose was observed.

The presented case is an opportunity to remind about the necessity of verifying the type of diabetes, even with an apparently obvious clinical picture. Despite the frequent occurrence of obesity in type 2 diabetes, it should not be forgotten that a patient with decompensated type 1 diabetes showing a characteristic weight loss can still have a BMI within the range of obesity. A helpful and widely accessible tool in clinical practice for distinguishing between diabetes types can be measuring C-peptide levels. Although it should be routinely performed in the diagnosis of carbohydrate disorders, it can be particularly useful in situations where an obese diabetic patient does not respond to treatment.

AUTHOR CONTRIBUTIONS

Olgierd Dróżdż: Investigation; validation; writing – original draft. Wiktoria Bińczyk: Investigation; validation; writing – original draft. Bartosz Siudek: Investigation; writing – original draft. Katarzyna Błaszczyszyn: Visualization; writing – review and editing. Filip Jan Grajnert: Visualization; writing – review and editing. Michalina Zofia Grzelka: Supervision; validation; visualization. Maciej Rabczyński: Project administration; supervision; validation.

ACKNOWLEDGMENTS

None.

FUNDING INFORMATION

The authors declare that they are not associated with any organizations, nor do they receive funding from them that could be pertinent to the data in this manuscript preparation.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

ORCID

Wiktoria Bińczyk https://orcid. org/0009-0004-6600-9259

REFERENCES

- Rodrigues Oliveira SM, Rebocho A, Ahmadpour E, Nissapatorn V, de Lourdes PM. Type 1 diabetes mellitus: a review on advances and challenges in creating insulin producing devices. *Micromachines (Basel)*. 2023;14(1):151. doi:10.3390/ mi14010151 PMID: 36677212; PMCID: PMC9867263.
- Klein S, Gastaldelli A, Yki-Järvinen H, Scherer PE. Why does obesity cause diabetes? *Cell Metab.* 2022;34(1):11-20.

U.F.Y_Clinical Case Reports ____

doi:10.1016/j.cmet.2021.12.012 PMID: 34986330; PMCID: PMC8740746.

- Bao YK, Ma J, Ganesan VC, McGill JB. Mistaken identity: missed diagnosis of type 1 diabetes in an older adult. *Med Res Arch.* 2019;7(8):1962 Epub 2019 Aug 15. PMID: 31930171; PMCID: PMC6953756.
- ElSayed NA, Aleppo G, Aroda VR, et al. 2. Classification and diagnosis of diabetes: standards of care in diabetes—2023. *Diabetes Care*. 2023;46(Suppl 1):S19-S40.
- Ahmed B, Sultana R, Greene MW. Adipose tissue and insulin resistance in obese. *Biomed Pharmacother*. 2021;137:111315. doi:10.1016/j.biopha.2021.111315 Epub 2021 Feb 6. PMID: 33561645.
- Pihoker C, Gilliam LK, Hampe CS, Lernmark A. Autoantibodies in Diabetes. *Diabetes*. 2005;54(suppl_2):S52-S61. doi:10.2337/ diabetes.54.suppl_2.S52
- Bhatty A, Baig S, Fawwad A, Rubab ZE, Shahid MA, Waris N. Association of Zinc Transporter-8 autoantibody (ZnT8A) with type 1 diabetes mellitus. *Cureus*. 2020;12(3):e7263. doi:10.7759/ cureus.7263 PMID: 32292675.

- Aulanni'am A, Wuragil DK, Susanto H, et al. The early detection of type 1 diabetes mellitus and latent autoimmune diabetes in adults (LADA) through rapid test reverse-flow immunochromatography for glutamic acid decarboxylase 65 kDa (GAD65). *Heliyon.* 2021;8(1):e08695. doi:10.1016/j.heliyon.2021.e08695 PMID: 35028470; PMCID: PMC8741515.
- Rosário PW, Reis JS, Fagundes TA, et al. Latent autoimmune diabetes in adults (LADA): usefulness of anti-GAD antibody titers and benefit of early insulinization. *Arq Bras Endocrinol Metabol.* 2007;51(1):52-58. doi:10.1590/s0004-27302007000100009 PMID: 17435855.

How to cite this article: Dróżdż O, Bińczyk W, Siudek B, et al. Obesity as a misleading factor in the diagnosis of type 1 diabetes mellitus: A case report. *Clin Case Rep.* 2024;12:e9301. doi:10.1002/ccr3.9301