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Case Report

Pregnancy and Challenging Transient Anti-GAD65 Positivity: A Case Report with Literature Review

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Abstract

Introduction

During pregnancy, women may develop blood glucose abnormalities like gestational diabetes mellitus (GDM) or, rarely, type 1 diabetes (T1D), which can lead to complications. Anti-GAD65 is a key antibody used in diagnosing T1D. This study presents a rare case of T1D developing a week before birth, with transient anti-GAD65 positivity.

Case presentation

A 38-year-old patient who delivered a baby a week earlier and had high blood glucose was admitted to the hospital with shortness of breath, chest tightness, abdominal pain, generalized weakness, nausea, and repeated vomiting. She had elevated anti-GAD65 and was diagnosed with T1D and diabetic ketoacidosis. Insulin injection was prescribed, with lifestyle modifications, later oral hypoglycemic medications added. After a few months, both anti-IA2 and anti-GAD65 antibodies were negative.

Literature review

Seven cases of T1D during or after pregnancy were reviewed. Six reported BMI. The mean HbA1c was >7.63%. Mean anti-GAD65 was 190.74 U/mL, two were borderline and one negative. Six had previously diagnosed GDM. Treatments varied, including insulin and dietary management. All infants were safely delivered, one miscarried in a subsequent pregnancy. Insulin resistance increases during pregnancy due to hormonal changes, raising the risk of GDM, T1D, and type 2 diabetes. Emerging postpartum, often indicated by anti-GAD65 antibodies, though levels can fluctuate. Cases show complications like preeclampsia, DKA, and miscarriage. Early detection, strict glucose control, and monitoring antibody patterns are critical for managing risks and improving maternal and fetal outcomes.

Conclusion

Blood glucose should be monitored during pregnancy, and anti-GAD65 may signal T1D, requiring appropriate management.

1. Introduction

Type 1 diabetes (T1D) is a condition characterized by a lack of the hormone insulin due to an autoimmune response that destroys the pancreas's insulin-producing β cells [1]. During pregnancy, women may develop an abnormal tolerance to glucose, which is not severe enough to be classified as overt diabetes mellitus, known as gestational diabetes mellitus

(GDM). This condition increases the possibility of type 2 diabetes (T2D) after birth, and the risk of T1D, especially if they have autoantibodies targeting the beta cells of the pancreas [2]. Around 2-17% of expectant mothers empierce GDM. While glucose metabolism typically returns to normal after childbirth, there is a high chance, between 34% up to half of the cases, that it will recur in future pregnancies, with around 6% developing into T1D [3]. Throughout an asymptomatic period of variable length, different autoantibodies targeting islet cell autoantigens can be identified, and these play a key role in recognizing individuals at an increased risk of developing clinical disease. Glutamic acid decarboxylase (GAD) is a key enzyme, antibodies against said enzyme or anti-GAD65 are one of the primary antibodies associated with T1D, with two isoforms having molecular weights of 65 kDa and 67 kDa. This antibody is found in most individuals with preclinical or recently diagnosed T1D. [4]. It is well established that pregnancies that involve T1D face a greater risk of complications affecting the mother, fetus, and newborn compared to those with uncomplicated pregnancies [5]. Poorly managed high blood glucose during pregnancy can result in serious complications, including stillbirth, miscarriage, congenital disabilities, and pregnancy-related conditions like hypertension or preeclampsia [1]. The aim of this study is to show a rare case of T1D developing a week before giving birth, with a transient anti-GAD65 positive test result, and a literature review. The references used in this case report have been checked for reliability and compiled with CaReL guidelines [6,7].

2. Case Presentation

2.1. Patient information

A 38-year-old female was admitted to the hospital with shortness of breath, chest tightness, abdominal pain, generalized weakness, nausea, and repeated vomiting that had persisted for two days before admission. She had been pregnant and had delivered a stillborn baby one week earlier at 39 weeks of gestation.

2.2. Clinical findings

On clinical examination, the patient appeared distressed and exhibited tachypnoea. Her heart rate was 110 beats per minute, respiratory rate was 32 cycles per minute, blood pressure was 110/70 mmHg, and her temperature was 38°C. Her BMI was 34 kg/m². She had no history of chronic disease.

2.3. Diagnostic approach

The patient was admitted to the hospital, resuscitated, and diagnosed with diabetic ketoacidosis (DKA), based on the presence of ketone bodies in the urine (3+) and acidosis confirmed by arterial blood gas analysis (pH 7.16, bicarbonate 12 mmol/L), her blood glucose was over 300 mg/dL. Anti-GAD test came back elevated at (65.1 U/mL), leading to a diagnosis of T1D.

2.4. Therapeutic intervention

Several days before her scheduled Cesarean section, she was admitted to the hospital due to elevated blood pressure and blood glucose levels. She was treated with insulin therapy to manage her condition. Following the surgery, the patient was evaluated by an endocrinologist for her hyperglycemic condition. Based on her medical history, the decision was made to discontinue insulin therapy and initiate lifestyle modifications along with oral hypoglycemic medications. However, two days after this change in treatment, she developed DKA. To treat her DKA, she received intravenous insulin infusion and was rehydrated with normal saline and received potassium.

2.5. Follow-up

A few months after starting insulin therapy, she underwent testing for T1D-related autoantibodies, which came back within normal ranges. The results were negative for both anti-IA2 and anti-GAD65 antibodies.

3. Discussion

The sensitivity to insulin declines during pregnancy, primarily due to the influence of placental hormones. Human growth hormone, along with human placental lactogen, plays a key role in regulating maternal metabolism and supporting the growth of the fetus, with levels gradually increasing throughout the first and second trimesters. Pregnancy is also associated with a reduced secretion of human growth hormone in response to low blood glucose, which further contributes to the resistance toward insulin. These effects are compounded by hormonal changes involving progesterone, prolactin, cortisol, adiponectin, and leptin [1]. Seven cases of T1D arising during or after pregnancies have been reviewed (Table 1) [2, 3, 8-10]. Six cases mentioned the patient's body mass index, one of them mentioning the pre-pregnancy body mass index. The mean HbA1c was over 7.63, only one case didn't mention performing a HbA1c test. The mean anti-GAD65 was 190.74 U/mL, with two cases being borderline at around 2 U/mL and one negative at 0.268 U/mL. Six cases were associated with GDM before T1D. Different treatments and therapeutics were used, including insulin therapy and dietary managements. All the infant were delivered safely, but one case had another pregnancy a few months later and the fetus miscarried.

Pregnancies with T1D who have poor blood glucose control face a much higher risk of developing preeclampsia compared to those who do not. Even if T1D is not involved, higher blood glucose levels are consistently linked to increased birth weight and other complications during and after birth [5]. Ikeoka et al. report a case of a 27-year-old pregnant female diagnosed with T1D through a 75-g oral glucose tolerance test conducted during the postpartum monitoring of GDM, which itself was diagnosed earlier after previous plasma glucose measurements prompted a 75-g oral glucose tolerance test. She was advised to follow a nutritional treatment plan and delivered the infant. She had several follow-ups and was diagnosed with established diabetes. She was pregnant again after four months, and intensive insulin therapy was initiated, but sadly, she experienced a miscarriage of the second fetus one week later [2].

Table 1. Review of similar cases of type 1 diabetes arising during or after pregnancies in the literature.

Author and year of publication	Age	Clinical findings	Performed tests	Diagnosis	Treatment	Outcome
Abdelmasih et al. 2024 [8]	22	Not mentioned.	Blood glucose (300 mg/dl), HbA1c (9.4%, prepartum 5.3%), postpartum blood glucose (300-400 mg/dl) C peptide (0.6 ng/dl), & anti-GAD65 (38.8 U/mL).	GDM previously, later postpartum T1D.	Insulin therapy, later stopped and continued on Metformin, Glipizide & Glargine added, later oral antidiabetics were stopped & prandial insulin started.	Infant delivered with macrosomia.
Fujishima et al. 2023 [3]	29	BMI (19.1 kg/m2), TSH receptor autoantibody +ve but low, diagnosed with Graves' disease previously, diabetic retinopathy not observed.	HbA1c (6.9%), anti-GAD65 (1210 U/mL), Fasting C-peptide (1.19 ng/mL), daily urinary C-peptide (120.6 µg/day), blood glucose 2 months after birth≥ 200 mg/dL.	GDM, then postpartum T1D.	Dietary management, insulin aspart, lispro, and detemir administered, & methimazole to control thyroid function.	Infant delivered safely.
Ikeoka et al. 2018 [2]	27	High postprandial blood glucose, diagnosed with GDM beforehand, BMI (16.9 kg/m2).	Frequent postprandial blood glucose (140-180 mg/dL), 6 weeks postpartum 75-g OGTT at 2 h (258 mg/dL), 4 months postpartum 75-g OGTT at 2 h (453 mg/dL) and HbA1c (6.7%), glycated albumin (22.5%), 6 months postpartum anti-GAD65 (57.1 U/mL).	GDM previously, later postpartum T1D.	Dietary management, neutral protamine Hagedorn, and insulin aspart, pregnant again at 4 months postpartum and received intensive insulin therapy.	1st Infant delivered, 2nd fetus miscarried.
Himuro et al. 2014 [9]	32	Fatigue and thirst, Kussmaul breathing with ketotic odor, pBMI (19.9 kg/m2).	plasma glucose (489 mg/dl), anion gap (21.9), arterial pH (7.45), base excess (9.8 mmol/l), anti-GAD65 (25 U/mL), IA-2 (1.5 U/ml).	DKA, with adult-onset T1D.	treatment with saline, intensive insulin therapy, & diet therapy.	Infant delivered safely.
Bonsembiante et al. 2013 [10]	42	Pregnancy BMI (22.5 kg/m2).	Postpartum HbA1c (9.5%), Postpartum OGTT at 2 h (>200 mg/dL), low blood insulin, C-peptide (1.1 ng/mL), anti-GAD65 (>2 U/mL).	GDM & later T1D.	Insulin therapy, & dietary management.	Infant delivered safely.
	39	Pregnancy BMI (20.4 kg/m2).	Blood sugar (170 mg/dL), HbA1c (7.3%), C-peptide (1.2 ng/mL) & anti-GAD65 (2 U/mL).	GDM & T1D in previous pregnancies.	Insulin therapy before, dietary management only now.	Infants delivered safely.
	36	Pregnancy BMI (22.4kg/m2).	anti-GAD65 (0.268 U/mL), fasting plasma C-peptide (1.8 ng/mL), mean HbA1c (6%).	GDM & later T1D.	Insulin therapy, & dietary management, postpartum only dietary restriction.	Infants delivered safely.

T1D = Type 1 diabetes, GDM = Gestational diabetes mellitus, +ve = Positive, pBMI = Pre-pregnancy body mass index, BMI = Body mass index, TSH = Thyroid-stimulating hormone, OGTT = Oral glucose tolerance test, HbA1c = Hemoglobin A1c, DKA = Diabetic ketoacidosis, IA-2 = Islet antigen-2, h = Hours.

Another serious and potentially deadly condition that can result from T1D is DKA, resulting from a lack of insulin, which was also present in this case. High levels of ketones in the blood and elevated blood glucose, although the specific diagnostic criteria can vary. Symptoms typically develop over a few hours and include vomiting, nausea, intense thirst, and frequent urination [11]. Around 1–2% of pregnancies that have glucose tolerance impairment develop DKA. It most commonly affects pregnancies with T1D, but it can also occur, though less frequently, in those with T2D, newly diagnosed T1D, or GDM [9]. Education on diabetes self-management, along with medical and psychological support, can play an essential role in preventing and managing DKA [11]. Himuro et al. report a case of a patient who, although she had normal glucose tolerance in the first trimester, was diagnosed with T1D and went on to develop DKA in her late pregnancy period. The patient was promptly treated with saline and intensive insulin therapy. Following this treatment, her blood glucose levels returned to normal [9].

About 5-10% of all individuals with diabetes have T1D. The autoimmune response is marked by the presence of autoantibodies targeting insulin, tyrosine phosphatase enzyme IA-2, glutamic acid decarboxylase 65, and islet cells [10]. The onset of autoimmune diabetes is closely linked to the presence of specific autoantibodies, with anti-GAD65 being diagnostically the most common. Over 80% of individuals who develop T1D during childhood or adolescence test positive for this antibody. This antibody and similar antibodies are often present years before autoimmune diabetes becomes clinically apparent, suggesting a prolonged phase of autoimmune activity before diabetes actually develops [12]. There can be debate about the exact roles that GAD and anti-GAD65 play in the development of T1D. For example, T1D has been documented in patients with agammaglobulinemia, and anti-GAD65 antibodies can be found in the blood of infants born to anti-GAD65 positive mothers, including those who are diagnosed with T1D during pregnancy, even if the infants themselves do not develop diabetes [13].

A study comparing two health surveys around 11 years apart and selecting for individuals self-reporting as not having diabetes at that time or in the past in both surveys, which found around 4500 individuals, selected 76 people who tested positive for anti-GAD in the first survey. Around 54% of them became negative in the following survey [12]. However, there are not many cases in the literature about anti-GAD65 test results changing from positive to negative during a short time period, especially with T1D. There are several cases of T2D patients who show transient anti-GAD65 positivity following immunoglobulin administrations that later change to negative results [14-16]. This may result from different mechanisms, such as passively transferred antibodies, where the intravenous immunoglobulin made from pooled plasma passes autoantibodies from the donor. Another possibility is transiently triggered antibody production, where the immunoglobulin activates B cells, leading to an increase in plasmablasts and temporary antibody formation that causes a positive test result. These antibodies are likely non-harmful and typically decrease on their own within several weeks [14].

Another case reported temporary anti-GAD positivity in a patient with acute pancreatitis who also had a genetic haplotype associated with susceptibility to T1D. However, the patient's ability to produce insulin naturally was not impaired. The patient was anti-GAD negative a year prior, and levels of elastase-I were increased. Once the elastase-I levels and pancreatic swelling subsided, the anti-GAD levels returned to normal. This may have been triggered by the release of GAD antigen from damaged islet cells caused by pancreatitis, and the immunological response to it [13]. One case reported fulminant T1D with transient anti-GAD65 production. The patient presented with high blood glucose levels, ketonemia, and moderately increased HbA1c and glycoalbumin levels, among other abnormalities. He was initially treated with intravenous fluids and a continuous intravenous infusion of regular insulin, later changed to several insulin injections a day, with anti-GAD65 antibody measurements changing from 111 U/mL at the start, dropping to 22.8 U/mL after two weeks, and becoming negative after a year. The sudden and complete virally induced deterioration of beta cells, possibly causing the temporary rise in anti-GAD antibody levels, particularly in individuals with a genetically susceptible HLA class II haplotype like this patient [17].

Strict blood glucose control before and during pregnancy and careful adjustments to basal and bolus insulin doses help lower the risk of complications [1]. Though this maintenance and control of blood glucose in pregnant women with T1D leads to better health outcomes. However, striving for normal blood glucose levels can also pose risks, especially the increased likelihood of maternal hypoglycemia. Additionally, although achieving normal blood glucose levels is typically possible with treatment in cases of GDM and often in T2D, it is significantly more difficult in pregnant women with T1D and requires continuous management to avoid hypoglycemia [18]. A recent study showed that pregnant women with T1D had the most unstable glycemic profiles and a significantly more frequent hyper- and hypoglycemia records compared to those with T2D and GDM [19]. Females with pre-existing diabetes tend to experience more hyperglycemia complications during pregnancy than those with GDM, especially if high blood sugar isn’t properly managed. However, these risks can be reduced with pre-pregnancy counseling that focuses on keeping HbA1c levels close to normal, checking for existing complications, stopping harmful medications and habits, taking folic acid, and ensuring strict blood glucose control throughout pregnancy [19]. Pregnancies that could develop T1D should be identified and monitored for possible risks, as well as the sudden onset of diabetes after delivery. Those who test positive for anti-GAD should be seen as having a high risk of developing T1D and may be considered for potential immunomodulatory treatments in the future [10].

4. Conclusion

Blood glucose levels should be continuously monitored during pregnancy in anticipation of sudden changes. Autoantibodies like anti-GAD65 indicate the possibility of T1D, which can have

adverse effects on both the mother and fetus, and should be considered and treated appropriately.

Declarations

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Use of AI: ChatGPT-3.5 was used to assist in language editing and improving the clarity of the manuscript. All content was reviewed and verified by the authors. Authors are fully responsible for the entire content of their manuscript.

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References

1. Roeder HA, Moore TR, Ramos GA. Changes in postpartum insulin requirements for patients with well-controlled type 1 diabetes. American journal of perinatology. 2016;33(07):683-7. doi:10.1055/s-0036-1571323
2. Ikeoka T, Sako A, Kuriya G, Yamashita H, Yasuhi I, Horie I, et al. Type 1 diabetes mellitus diagnosed during follow-up of gestational diabetes mellitus in the early postpartum period. Internal Medicine. 2018;57(23):3413-8. doi:10.2169/internalmedicine.1188-18
3. Fujishima A, Onodera Y, Miura H, Terada Y. Anti-glutamic acid decarboxylase antibody-positive gestational diabetes mellitus with autoimmune type 1 diabetes mellitus in the early postpartum period: A case report and literature review. The Tohoku Journal of Experimental Medicine. 2023;259(4):327-33. doi:10.1620/tjem.2023.J013
4. CHILDHOOD DIABETES IN FINLAND STUDY GROUP HOPPU S RONKAINEN MS KULMALA P ÅKerblom HK KNIP M mikael. knip@ hus. fi. GAD65 antibody isotypes and epitope recognition during the prediabetic process in siblings of children with type I diabetes. Clinical & Experimental Immunology. 2004;136(1):120-8. doi:10.1111/j.1365-2249.2004.02416.x
5. Damm P, Mersebach H, Råstam J, Kaaja R, Hod M, McCance DR et al. Poor pregnancy outcome in women with type 1 diabetes is predicted by elevated HbA1c and spikes of high glucose values in the third trimester. The Journal of Maternal-Fetal & Neonatal Medicine. 2014;27(2):149-54. doi:10.3109/14767058.2013.806896
6. Kakamad FH, Abdalla BA, Abdullah HO, Sami S. Omar, Shvan H. Mohammed, Sasan M. Ahmed, et al. Lists of predatory journals and publishers: a review for future refinement. Eur Sci Ed. 2024;50:e118119. doi:10.3897/ese.2024.e118119
7. Prasad S, Nassar M, Azzam AY, García-Muro-San José F, Jamee M, Sliman RK, et al. CaReL guidelines: A consensus-based guideline on case reports and literature review (CaReL). Barw Medical Journal. 2024. doi:10.58742/bmj.v2i2.89
8. Abdelmasih R, Pan W. 6938 An Unusual Case of New-onset autoimmune insulin dependent Diabetes Mellitus Complicating Gestational Diabetes Mellitus in A Pregnant Female Diagnosed Early Postpartum-The Enigma of Autoimmunity and Diabetes During Pregnancy. Journal of the Endocrine Society. 2024;8(Supplement_1): bvae163-661. doi:10.1210/jendso/bvae163.661
9. Himuro H, Sugiyama T, Nishigori H, Saito M, Nagase S, Sugawara J et al. A case of a woman with late-pregnancy-onset DKA who had normal glucose tolerance in the first trimester. Endocrinology, Diabetes & Metabolism Case Reports. 2014;2014(1). doi:10.1530/EDM-13-0085
10. Bonsembiante B, Dalfrà MG, Masin M, Gallo A, Lapolla A. Adult-Onset Type 1 Diabetes and Pregnancy: Three Case Reports. Case Reports in Medicine. 2013;2013(1):920861. doi:10.1155/2013/920861
11. Ehrmann D, Kulzer B, Roos T, Haak T, Al-Khatib M, Hermanns N. Risk factors and prevention strategies for diabetic ketoacidosis in people with established type 1 diabetes. The Lancet Diabetes & Endocrinology. 2020;8(5):436-46. doi:10.1016/S2213-8587(20)30042-5
12. Sørgjerd EP, Thorsby PM, Torjesen PA, Skorpen F, Kvaløy K, Grill V. Presence of anti-GAD in a non-diabetic population of adults; time dynamics and clinical influence: results from the HUNT study. BMJ open diabetes research & care. 2015;3(1). doi:10.1136/bmjdrc-2014-000076
13. Kahara T, Takamura T, Otoda T, Ishikura K, Matsushita E. Transient anti-GAD antibody positivity and acute pancreatitis with pancreas tail swelling in a patient with susceptible haplotype for type 1 diabetes mellitus. Internal medicine. 2009;48(21):1897-9. doi:10.2169/internalmedicine.48.2393
14. Nakashima H, Omura T, Teng X, Kamihara T, Yokoi K, Kawashima S et al. Transient Anti-Glutamic Acid Decarboxylase (GAD) Antibody Positivity and Insulin Secretion Reduction After IVIg in Type 2 Diabetes Mellitus. Cureus. 2025;17(4). DOI: 10.7759/cureus.82038
15. Kawasumi M, Ishii S, Uchida S, Shishido T. A Case of Type 2 Diabetes Mellitus Revealed Transient Positivity of Glutamic Acid Decarboxylase (GAD) Antibodies Following Immunoglobulin Administrations. Cureus. 2024;16(8). doi:10.7759/cureus.66485
16. Takahashi J, Honda H, Hamahara J, Kajitani N, Ando S, Deguchi S et al. Two cases of type 2 diabetes revealed transient positive for anti-GAD antibodies following treatment with immunoglobulin. Journal of the Japan Diabetes Society. 2020;63(11):770-5. doi:10.11213/tonyobyo.63.770
17. Harada N, Nakayama H, Nomura M. A Case of Fulminant Type 1 Diabetes with Transient Production of Anti-Glutamic Acid Decarboxylase Antibody. The American Journal of Case Reports. 2024;25: e943590-1. doi:10.12659/AJCR.943590
18. Maresh MJ, Holmes VA, Patterson CC, Young IS, Pearson DW, Walker JD, et al. Glycemic targets in the second and third trimester of pregnancy for women with type 1 diabetes. Diabetes care. 2015;38(1):34-42. doi:10.2337/dc14-1755
19. Salih JM. Glycemic Profiles and Hypoglycemia Awareness Among Pregnant Women with Gestational and Pre-existing Diabetes Referred to a Tertiary Center in Sulaimaniyah-Iraq in 2024. International Journal of Endocrinology and Metabolism. 2024;22(4):e153529. doi:10.5812/ijem-153529