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Euglycemic Ketoacidosis in Patient with Acute Gastroenteritis with Known Case of Diabetes Mellitus Diagnosed to be Latent Autoimmune Diabetes of Adulthood

ABSTRACT

A 63-year-old female who is a known case of diabetes mellitus presented with a history of vomiting and loose stool for 1 day following a recent history of travel and outside food consumption. The patient's blood sugar levels were within the normal range. The patient was hemodynamically stable. The patient started developing breathlessness in the ward and was shifted to the intensive care unit. She was tested for serum acetone and urine ketone which tested positive and was started on an IV insulin infusion. The patient showed improvement and was diagnosed with latent autoimmune diabetes of adulthood in view of euglycemic ketoacidosis.

Key words: C-peptide levels, Diabetes ketoacidosis, Insulin level, Latent autoimmune diabetes of adulthood

INTRODUCTION

Latent autoimmune diabetes in adults (LADA) displays similar autoantibodies as type 1 diabetes mellitus (T1D) but, in fact, is a milder and slowly progressive disease as compared to T1D. Therefore, LADA patients generally do not require insulin for some time following diagnosis. As compared to type 2 diabetes mellitus (T2D), LADA patients demonstrate less insulin secretion and progress rapidly to insulin dependency. Patients with LADA generally do not manifest diabetic ketoacidosis (DKA) at presentation owing to the slow progression of β-cell dysfunction. Here, we report a case of latent autoimmune diabetes in an adult male who presented with DKA.

CASE REPORT

A 63-year-old female presented with complaints of vomiting and loose stools for 1 day. She was apparently alright 1 day ago when she complained of acute-onset vomiting, which was 4–5 episodes, non-bilious, non-projectile, yellow in color with food content, and no blood and was associated with abdominal pain. She also complained of loose stools of 3–4 episodes, watery with no blood or mucous, and severe abdominal pain. The patient has a recent history of travel and consumption of outside food. The patient also complained of generalized weakness and a decrease in appetite, along with associated body pain. After 4 h of admission, the patient developed severe breathlessness along with abdominal pain. The patient has been a known case of diabetes mellitus and hypertension for 4 years. There is no history of fever, urinary complaints, polyuria, and increased thirst. There is no history of chest pain.

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On general examination, the patient was conscious and well oriented.

Temp: 98 F (afebrile), Pulse: 93/min, regular. Respiratory rate: 16/min, abdominothoracic in nature, SpO_2: 96% on room air, BP: 100/70 mmHg in the right upper limb, in the supine position. There is no pallor, icterus, cyanosis, clubbing, lymphadenopathy, and edema. No evidence of dehydration. On systemic examination, no significant abnormalities were detected in any of the systems. Patient's random blood sugar was 200 mg/dL.

After a few hours, the patient started developing breathlessness, and arterial blood gas (ABG) was done, which showed severe metabolic acidosis with a pH of 6.9 (decreased), pCO_2 of 20.7 mmHg (decreased), and HCO_3 of 4.1 mmol/L (decreased). The patient was shifted to the intensive care unit, was started on 2 L of oxygen (non-invasive), and was given injection calcium gluconate IV (10 mL) and injection sodium bicarbonate IV (50 mL). Her blood sugar was 210 mg%. Serum acetone and urine ketone were done, which came out to be positive, and the patient was also started on IV fluid (1 L saline IV fast followed by another 1 L 0.45 saline) along

Table 1: Investigations

Parameter	June 07, 2023	June 08, 2023	June 09, 2023	June 10, 2023
Hb	11.4 g/dL	9.6 g/dL		9.1 g/dL
WBC	8740 cells/µL	8580 cells/µL		5100 cells/µL
Platelets	359K cells/µL	238K cells/µL		196 cells/µL
Sodium	137 mEq/L	145 mEq/L	140 mEq/L	137 mEq/L
Potassium	4.3 mEq/L	4.2 mEq/L	3.2 mEq/L	3.9 mEq/L
Chloride	99 mEq/L	111 mEq/L	102 mEq/L	102 mEq/L
Serum acetone	+VE			
Urine ketone	+VE			
Creatinine	0.6 mg/dl			
BUN	13.4 mg/dl			
C-peptide				0.7 ng/L
Glutamic acid decarboxylase (GAD65) autoantibodies				120.3 IU/L

with an insulin infusion (HAI). Blood glucose levels were monitored every 1 hour. Urine output was adequate. Other routine investigations were as follows [Table 1]. Later, blood sugar monitoring was done every 2 hours and showed a level of 220 mg/dL in the 1st h, 212 mg/dL after 2 h, 209 mg/dL in the 4 h, and 180 mg/dL after the 5 h period. ABG was also done again after a few h, which showed a pH of 7.2, HCO_3 of 12.4 mmol/L, and pCO_2 of 10 mmHg. The patient felt better with a reduction in breathlessness. Patients vomiting and loose stools also subsided. ABG was done again, which showed a pH of 7.45, pCO_2 of 20.7 mmHg, HCO_3 of 18 mmol/L, and a blood sugar level of 201 mg/dL.

ABG was done again on the 2nd day and showed a pH of 7.4, pCO_2 of 30.7 mmHg, HCO_3 of 23.8 mmol/L, and a blood sugar level of 113 mg/dL. The patient was switched to subcutaneous insulin, and IV fluids were stopped. Urine output was monitored, which was 70–80 mL/h.

The C peptide level showed a value of 0.7 ng/mL (normal 1.1–4.4 ng/mL), and the glutamic acid decarboxylase (GAD 65) antibody level was 120.3 IU/L (normal <13 IU/L). Fasting blood sugar was 130 mg/dl, and there was no episode of hypoglycemia. The patient was discharged from the hospital on an insulin dose and was advised for regular follow-up and maintenance of her blood sugar levels.

DISCUSSION

Our patient, presented with gastroenteritis with DKA with normal blood sugar (RBS–200 MG%) whereas in DKA, typically, blood sugar should be more than 250 mg%. This stimulated us to think about LADA.

Latent autoimmune diabetes antibody (LADA) is a subtype of diabetes type 1. There is beta cell destruction as a result of an autoimmune response directed against pancreatic islet cells. The C-peptide levels have decreased.[1] LADA patients do not immediately lose all beta cell functions that are why they have a clinical presentation that looks more like diabetes type 2. Approximately 10% of the newly diagnosed people with diabetes type 2 have autoantibodies against pancreatic beta cells, which causes insulin insufficiency.[1] Unlike classic childhood-onset Type 1 diabetes, which is "fast and furious" with kids ending up hospitalized seemingly within days of the first onset of symptoms, LADA "ramps up" much more slowly. As a result, it is often mistakenly assumed that adult patients presenting with LADA have type 2 diabetes. This is because many if not most patients early in the course of LADA respond relatively well to oral diabetes medications and other non-insulin medications, which typically would not work in a younger patient with classic Type 1 diabetes onset (where insulin is invariably required for treatment). Clinical criteria for LADA are loosely defined as, adult onset, lack of initial need for insulin therapy, low C peptide levels, and pancreatic antibodies against pancreatic islet cells.[2]

Several islet cell autoantigens are linked to DM1, including the tyrosine phosphatase-like proteins IA-2 and IA-2beta, GAD65 autoantibodies, islet cell cytoplasmic antibodies, islet cell complement-fixing autoantibodies, and insulin antibodies.[3] The autoantibody titer most commonly used to identify LADA is the GAD65 autoantibody.[4] The association of other autoimmune diseases with latent autoimmune diabetic antibodies, especially autoimmune thyroid disease and coeliac disease, is well established.[5] Insulin therapy is effective and safe for LADA patients as it preserves pancreatic β-cell function. In addition to insulin, other therapy options that preserve β-cell function, including dipeptidyl peptidase 4 inhibitors and glucagon-like peptide 1 receptor agonists, could be considered for LADA patients.[6]

CONCLUSION

This patient had a presentation of DM type 2 with diabetes ketoacidosis which was precipitated by acute gastroenteritis

and was later found to have latent autoimmunity of diabetes with adulthood (LADA). Most patients with LADA are not identified at initial presentation. It is only after the symptoms of DKA which occurs in T2D, a diagnosis of LADA can be made. This case highlights the importance of clinical recognition of LADA and the implementation of screening diagnostic tests, emphasizing that all patients who do not fit the typical type 2 DM profile should be further investigated. The early identification of these patients is associated with better glycemic control, thus potentially decreasing the risk of long-term complications. The best therapeutic approach should be individualized and tailored to each patient, considering the importance of C-peptide measurement to begin insulin therapy.

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