Case Report

Diabetic Ketoacidosis as Presenting Feature of Acromegaly

Ahmed Al Montasir [1], Mahnoor Fatima [2], Karma Dicky Sherpa [3], Md. Azizur Rahman [4], Md. Zakir Hossain [5], Minhaz Uddin [6]

Abstract

A 50-year-old man's initial symptom of acromegaly was diabetic ketoacidosis (DKA). The patient presented with abdominal pain, polyuria, polydipsia, hyperhidrosis, and enlargement of extremities. His acromegalic features also were macroglossia, and jaw prominence. Lab testing showed hyperglycemia, high HbA1c, and elevated IGF-1. Considering hyperglycemia and abdominal pain we checked urine for ketone bodies, arterial blood gas for pH, serum electrolytes and also calculated osmolarity. We found the biochemical results were suggestive of DKA according to American Diabetes Association (ADA) diagnostic criteria for DKA. MRI showed a pituitary macroadenoma. After initial treatment following diabetic ketoacidosis protocol he was referred to neurosurgeon. DKA often the presenting feature of type 1 diabetes. It may also occur in type 1 and type 2 diabetes mellitus (DM) due to severe illness, infections, myocardial infarction, heart failure, pancreatitis, pregnancy, or steroids. In acromegaly, GH induces insulin resistance, poor glucose metabolism. Acromegaly may induce glucose intolerance, however DKA is rare. Some cases were treated with surgery or somatostatin. We present a case of a patient with no past medical history of DM who presented to the hospital for DKA and was found to have acromegaly.

Key words: *Acromegaly, Diabetes, Ketoacidosis, Macroadenoma, Surgery*

Date of received: *08.03.2025*

Date of acceptance: *30.06.2025*

DOI: https://doi.org/10.3329/kyamcj.v16i02.81135

KYAMC Journal. 2025; 16(02): 101-104.

Introduction

Diabetic ketoacidosis (DKA) is an acute metabolic condition seen in persons with diabetes mellitus. The principal metabolic alterations include markedly increased concentrations of ketone bodies, including beta-hydroxybutyrate, acetoacetate, and acetone in the blood, accompanied by concurrent hyperglycemia. DKA may pose a danger to life. Triggers may include the discontinuation of insulin treatment, and the onset of acute infections. This case report details one patient who exhibited DKA as the first manifestation of acromegaly due to growth hormone-secreting pituitary adenomas.

Case report

A 50-year-old male presented to the emergency department with diffuse abdominal pain of two days' duration. He reported a six-month history of increased thirst, appetite, and frequency of urination, accompanied by hyperhidrosis. The patient also noted that his shoes had become tighter more quickly and reported gradual increases in the size of his hands and feet, which began insidiously. These changes led to difficulty in performing daily activities such as sitting, walking, and other normal duties. Additionally, he experienced a weight gain of 10 kg over the past two years. The patient denied changes in temperature sensitivity, cutaneous alterations, galactorrhea, palpitations, vomiting, or seizures. On physical examination, vital parameters were within normal limits. The patient's height was recorded at 5’6”, and his weight was 85 kg. Notable findings included a large head with prominent jaw, gap in the incisor teeth (Fig. 1) enlarged hands (Fig. 2) and feet , and macroglossia. Visual field assessment revealed a decreased (100-degree) lateral gaze, while other systemic examinations appeared normal. Laboratory investigations revealed significantly elevated blood glucose levels: a random blood sugar of 27.05 mmol/L, fasting blood sugar of 26 mmol/L,and postprandial blood sugar of 32 mmol/L. The glycosylated hemoglobin (HbA1C) was found to be 18%. Arterial pH was 7.24, PCO2 25 mmHg, bicarbonate 12 mEq/l (24-30 mEq/L), serum sodium 128 mmol/l (135-145 mEq/L), potassium 5.2 mEq/l (3.5-4.5 mEq/L), chloride 97 mEq/l (95-105 mEq/L), serum creatinine 1.7 mg/dl (0.7-1.2 mg/dl), blood urea 11.4 mmol/l (2.5-10.7 mmol/l), WBC count 18.5 × 10^9/L , urine ketones strongly positive.

1. Resident Physician, Department of Medicine, TMSS Medical College, Bogura, Bangladesh.
2. Fourth Year MBBS Student, Shaheed Ziaur Rahman Medical College, Bogura, Bangladesh.
3. Final Year MBBS Student,TMSS Medical College, Bogura, Bangladesh.
4. Professor of Endocrinology, Department of Medicine, TMSS Medical College, Bogura, Bangladesh.
5. Professor of Medicine, TMSS Medical College, Bogura, Bangladesh.
6. Registrar, Department of Medicine, TMSS Medical College, Bogura, Bangladesh.

Corresponding author: Ahmed Al Montasir, Resident Physician, Department of Medicine, TMSS Medical College, Bogura, Bangladesh.
Cell: +8801711483847 , **E-mail:** a.montasir@gmail.com

Thyroid function tests showed TSH at 3.94 µU/mL (0.35-4.5 µ U/mL), FT4 at 1.22 ng/dl (0.89-1.72 ng/dl). Serum prolactin level was 206.23 ng/ml (4.64 – 15.2 ng/ml). Notably, insulin-like growth factor-1 (IGF-1) was found elevated at 600 µg/ml (n=53–331 µg/ml). X ray skull showed calvarial thickening, particularly of the inner table, enlarged paranasal sinuses (especially frontal sinuses) and an enlarged sella turcica. The mandible enlarged resulting in prognathism and gaps between the teeth (Fig. 3). Magnetic Resonance Imaging (MRI) of the head demonstrated sella enlarged in size. Moderately enhancing mass lesion (1.91.6 cm) in the sella with minimal suprasellar extension most likely pituitary macroadenoma (Fig. 4). Based on the clinical presentation, imaging findings, and laboratory results, the patient was diagnosed with acromegaly secondary to a macroadenoma, concurrent with diabetes mellitus, presenting in a state of diabetic ketoacidosis and hypertriglyceridemia (320 mg/dl) (Normal <150 mg/dl). During admission, the patient's blood glucose levels and diabetic ketoacidosis were managed with insulin administration, intravenous fluids, and careful electrolyte balance. By the time of discharge, the patient was stabilized, reassured and continued on insulin (30/70 regimen). A plan for initiating medical therapy with subcutaneous octreotide was established. The patient was also referred to a neurosurgeon to discuss the need for surgical intervention or radiotherapy.

Figure 1: Face showing prognathism, a wider nose, interdental separation, and increased face skin folds.

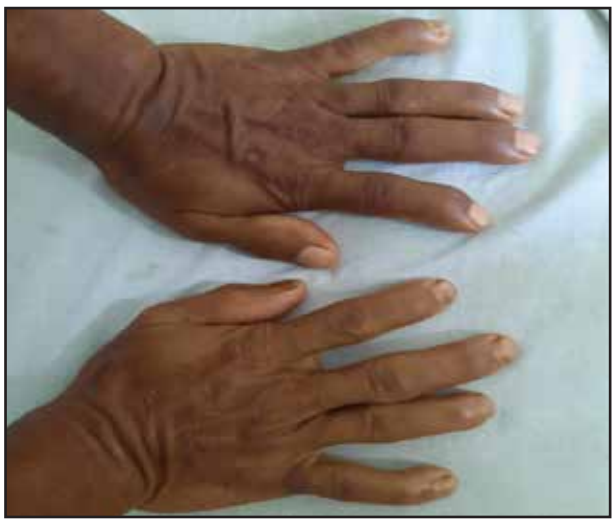

Figure 2: Spade-like hands of the patient.

Figure 3: X ray skull lateral view showing calvarial thickening, enlarged paranasal sinuses, an enlarged sella turcica, prognathism.

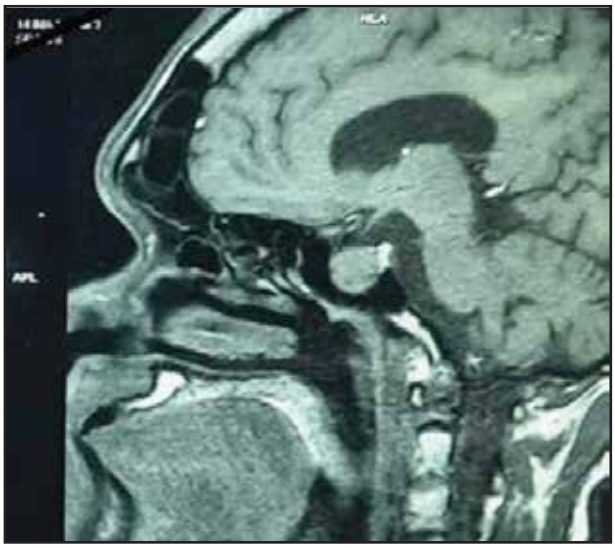

Figure 4: Enlarged sella with enlarged pituitary.

Discussion

DKA often represents the primary manifestation of type 1 diabetes mellitus (T1DM) at the time of diagnosis, especially in pediatric and adolescent population 1. In patients with a prior diagnosis of diabetes, DKA may occur after the cessation or neglect of insulin administration due to psychological, social, or economical influences.[1,2] DKA arises from a relative or absolute deficiency of insulin, frequently intensified by acute illnesses or infections. These conditions initiate a series of events resulting in hyperglycemia, dehydration, and the overproduction of ketones, which leads to metabolic acidosis.[1-6] Glucose intolerance and diabetes are acknowledged consequences linked to acromegaly. Excessive synthesis of growth hormone (GH) may lead to insulin resistance, resulting in impaired glucose tolerance or diabetes in 15% to 38% of acromegaly patients.[6,7] Acromegaly is a disorder caused by excessive secretion of growth hormone (GH) and is marked by distinct clinical features. Key features include enlarged extremities, coarse facial features including a prominent jaw and forehead, along with a significant enlargement of the lips, nose, and tongue. Furthermore, individuals may have hyperhidrosis, seborrhea, voice deepening, cephalalgia, arthralgia, and visual problems.[7]

The primary diagnostic screening test is Insulin-like Growth Factor-1 (IGF-1), followed by confirmatory assessments such as glucose-suppressed GH and MRI of the hypothalamic-pituitary region with dynamic contrast. The predominant etiology is usually a GH-secreting adenoma, with macroadenomas occurring more often than microadenomas; however, ectopic GH-secreting tumors have also been seen. Treatment often involves transsphenoidal surgery, followed by either radiation or pharmaceutical intervention (such as somatostatin analogs or pegvisomant).[8] Growth hormone and insulin-like growth factor 1 influence insulin function via altering the quantity of insulin receptors and the actions subsequent to receptor engagement.[9]

In some acromegaly patients undergoing treatment, the presence of hypogonadism, hypothyroidism, and the management of adrenal insufficiency may exacerbate insulin resistance and persistent diabetes.[10]

Diabetic ketoacidosis (DKA) has been documented as a presenting symptom of Cushing's syndrome in the literature.[11] Kabadi UM reported two acromegaly patients that manifested as moderate to severe diabetic ketoacidosis; both attained full diabetes remission, one after surgery and the other after surgery and an injection of a somatostatin analog.[12] Simmons LR et al. similarly documented a juvenile acromegaly patient who had diabetic ketoacidosis, which fully recovered after transsphenoidal adenohypophysectomy.[13] Quarella M et al. recently reported a case of undiagnosed acromegaly that manifested as diabetic ketoacidosis, induced by a Sodium-Glucose Co-Transporter-2 (SGLT-2) inhibitor.[14]

The patient lacked a documented history of diabetes or any familial predisposition to the ailment. He had the severe complication of diabetic ketoacidosis (DKA). Characteristic manifestations of acromegaly identified throughout the evaluation corroborated the diagnosis. This highlights the need of evaluating acromegaly as a possible diagnosis in individuals exhibiting diabetes problems.

Conclusion

In patients presenting with DKA who are not previously diagnosed with diabetes, it is crucial to explore potential underlying causes. Rare etiologies include type 2 diabetes caused by acromegaly, glucocorticoid excess from Cushing's syndrome, glucagonoma, somatostatinoma, certain antipsychotic medications, alcohol use, and pancreatitis. For those suspected of having acromegaly leading to DKA, measurement of GH and IGF-1 levels and pituitary MRI are important. Although DKA as an initial manifestation of acromegaly is uncommon, the clinical outcome for DM is good, with diabetes potentially resolving following appropriate treatment of the acromegaly.

Acknowledgement

We would like to express our sincere gratitude to Dr. Riyajul Jannat, assistant registrar and Dr. Forhad Hossain, registrar, department of medicine, TMSS Medical College, Bogura, who were actively involved in this patient management. The collaborative efforts and expertise demonstrated by the team are truly commendable.

References

1. Vidal Cortada J, Conget Donlo JI, Navarro Téllez MP, Halperin Rabinovic I, Vilardell Latorre E (1995) Diabetic ketoacidosis as the first manifestation of acromegaly. An Med Interna 12: 76-78.

2. Westphal SA (2000) Concurrent diagnosis of acromegaly and diabetic ketoacidosis. Endocr Pract 6: 450-452. [Cross-ref]

3. Colao A, Ferone D, Marzullo P, et al. Systemic complications of acromegaly: epidemiology, pathogenesis, and management. Endocrine Reviews. 2004;25(1):102-52.

4. Fieffe S, Morange I, Petrossians P, et al. Diabetes in acromegaly, prevalence, risk factors, and evolution: Data from the French Acromegaly Registry. Eur J Endocrinol. 2011;164(6):877-84.

5. Rolla M, Jawiarczyk-Przybyłowska A, Halupczok-Żyła J, et al. Complications and comorbidities of acromegaly - Retrospective study in Polish center. Front Endocrinol. 2021;12:642131. doi: 10.3389/fendo.2021.642131.

6. Melmed S, Casanueva FF, Klibanski A, et al. A consensus on the diagnosis and treatment of acromegaly complications. Pituitary. 2013;16(3):294–302. doi: 10.1007/s11102-012-0420-x.

7. Melmed S. Medical progress: Acromegaly. N Engl J Med. 2006;355(24):2558–2573.

8. Kreze A, Kreze-Spirova E, Mikulecky M. Risk factors for glucose intolerance in active acromegaly. Braz J Med Biol Res. 2001;34(11):1429–1433.

9. Holt RIG, Simpson HL, Sönksen PH. The role of the growth hormone-insulin-like growth factor axis in glucose homeostasis. Diabet Med J Br Diabet Assoc. 2003;20(1):3–15.

10. Ebrahimi F, Christ-Crain M. Metabolic syndrome and hypogonadism–two peas in a pod. Swiss Med. 2016;21:146(1112).

11. Acharya R, Kabadi UM. Case of diabetic ketoacidosis as an initial presentation of Cushing's syndrome. Endocrinol. Diabetes Metab. Case Rep. 2017:16-0123.

12. Kabadi UM. Cases of diabetic ketoacidosis as an initial presentation of acromegaly Integr Obesity Diabetes. 2017;3(2):01-04. Doi: 10.15761/IOD.1000171.

13. Simmons LR, Hughes K, Markovic TP, Chua EL. Diabetic ketoacidosis as a presenting feature of acromegaly: When excess hormone meets hormone deficiency. Clinical Diabetes. 2012;30(4):170-72.

14. Quarella M, Walser D, Brändle M, Fournier JY, Bilz S. Rapid onset of diabetic ketoacidosis after SGLT2 inhibition in a patient with unrecognised acromegaly. J Clin Endocrinol Metab. 2017;102(5):1451-53.