

CASE REPORT

Diabetic Ketoacidosis as an Initial Sign of Acromegaly: A Case Report

Akromegalinin İlk Bulgusu Olarak Diabetik Ketoasidoz: Bir Olgu Sunumu

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Abstract

Acromegaly is a chronic disease leading to a multisystem and metabolic disease characterized by growth hormone (GH) hypersecretion resulting from adenoma of hypophysis. Acromegaly patients usually have insulin resistance and elevated plasma glucose levels due to increased GH action. But in the literature, diabetic ketoacidosis as a complication of acromegaly is rarely reported. The presented case report about a patient who had acromegaly and diabetic ketoacidosis emphasizes the importance of evaluating every single patient with secondary hyperglycemia resulting from other metabolic diseases.

Keywords: Acromegaly; Diabetic ketoacidosis; Diagnosis; Case report

Acromegaly is a chronic disease due to growth hormone (GH) hypersecretion. GH regulates insulin-like factor 1 (IGF-1) metabolism in the liver and systemic tissues. GH exerts most of its metabolic and systemic effects through IGF-1. GH and IGF-1 hypersecretion leads to comorbidities such as rheumatic, cardiovascular, respiratory, neoplastic, and neurological disorders and physical disfigurements (macroglossia, hand and foot enlargements, coarse facial features such as frontal bossing, prognathism, and enlargement of nose).^[1]

Diagnosis of acromegaly should rely on clinical and biochemical evaluation. Supporting clinical features with elevated GH and IGF-1 measurements must remind ac-

romegaly, and these patients should be evaluated by hypophysis magnetic resonance (MR) imaging to locate adenoma and determine the size and possible tumoral invasions. Because of the disease's slow-progressing nature, acromegaly diagnosis is usually delayed.^[2] Headache is the most frequent reason for hospital visits.^[3] In patients who have obstructive sleep apnea, type 2 diabetes mellitus (DM), debilitating arthritis, carpal tunnel syndrome, hyperhidrosis, and hypertension, and who have typical findings (physical disfigurements) of acromegaly, measuring IGF-1 levels help diagnosis. Rare cases are brought to the hospital with diabetic ketoacidosis coma (DKA). DKA is characterized by the existence of hyperglycemia,

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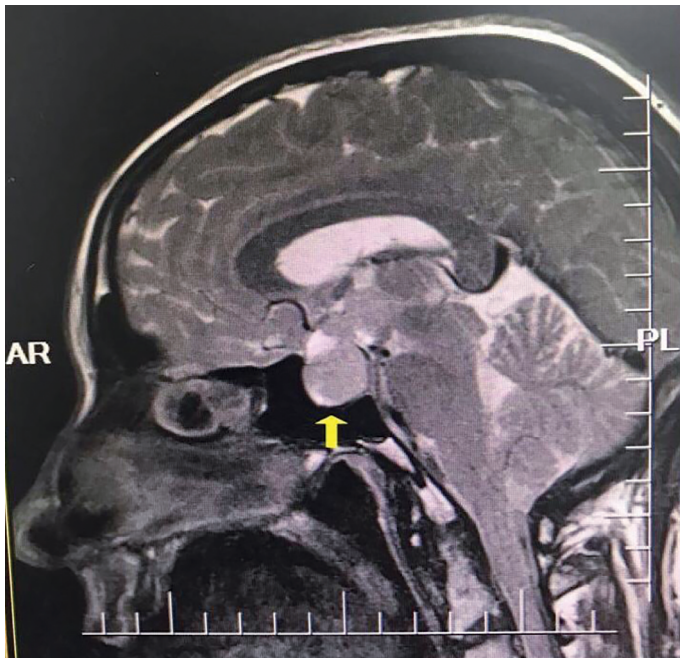


Figure 1. Macroadenoma of the hypophysis in sagittal T2A phase of magnetic resonance imaging.

metabolic acidosis, and ketonemia.^[3] The clinical situation usually exaggerates due to increased levels of counterregulatory hormones impairing the balance in gluconeogenesis, glycogenolysis, and glucose utilization in peripheral tissues. The primary counterregulatory hormone in the process of DKA is glucagon. Other hypersecreted counterregulatory hormones which result in the development of DKA are catecholamines, cortisol, and GH.^[4] DKA cases resulting from hypersecretion of GH are rarely reported in the literature. Here, a case report of a female patient who was admitted to the emergency department with hyperglycemia and who was diagnosed as DKA due to acromegaly is presented.

Case Report

A 43-year-old Caucasian female patient applied to the hospital emergency department with nausea, dizziness, dry mouth, hyperhidrosis, and headache complaints. The patient had no previous history of any chronic diseases. She previously applied to another medical center with similar symptoms and had a new-onset type 2 diabetes mellitus along with DKA diagnosis and took insulin treatment. In follow-up, she was also diagnosed to have secondary amenorrhea with accompanying symptoms of hyperhidrosis and headache when she applied to the endocrinology outpatient clinic and had a detailed hormonal evaluation for the first time, after diagnosis of diabetes mellitus. Physical exam-

Table 1. Laboratory findings of the patient before and 2 months after surgery

Parameter	Before surgery	After surgery	Normal values
Free T3 (pmol/L)	0.04	0.05	0.04–0.06
Free T4 (pmol/L)	9.40	10.68	11.46–22.65
TSH (mIU/L)	1.50	0.11	0.27–4.20
FSH (IU/L)	1.54	1.20	2.50–10.20
LH (IU/L)	0.15	–	1.90–12.50
Estradiol (pmol/L)	34.25	22.39	45.52–196.03
Somatomedin-C (nmol/L)	74.54	67.99	13.23–34.98
GH (ng/mL)	24.10	7.77	0.06–6
Insulin-like growth factor binding protein 3 (nmol/L)	1519.60	–	432.30–864.60
ACTH (pmol/L)	5.81	6.03	0–10.13
Prolactin (µg/L)	15	1.26	2.8–29.2
Sodium (mmol/L)	139	137	135–145
Potassium (mmol/L)	4.3	4.1	3.5–5.5
Cortisol (mmol/L)	35.70	38.59	19.31–80.00

GH: Growth hormone; TSH: Thyroid-stimulating hormone; FSH: Follicle-stimulating hormone; LH: Luteinizing hormone; ACTH: Adrenocorticotropic hormone.

ination revealed enlargement of hand and foot, macroglossia, and frontal bossing. Laboratory results were compatible with hypogonadotropic hypogonadism and secondary hypothyroidism. She had elevated levels of GH (24.10 ng/mL) and somatomedin-c (569 nmol/L). MR imaging revealed a macroadenoma of hypophysis which was compressing optic chiasm and which was 17×28×35 cm in size (Fig. 1). Surgical removal of pituitary adenoma was recommended for the patient. Laboratory findings of the patient before and 2 months after surgery are shown in Table 1. After transphenoidal hypophysis surgery, the patient received 50 µg levothyroxine replacement therapy. Early postoperative hypophysis MR imaging revealed that the majority of the adenoma was excised in operation. Headache complaint was soothed after hypophysis surgery. Because of elevated GH and somatomedin-c levels, the patient was continued to be treated with 120 mg lanreotide injection every 28 days, and this treatment continued. The blood glucose level of the patient was regulated with 18 units of insulin detemir injection daily, and postoperative hypothyroidism was treated with 50 µg of levothyroxine daily.

Discussion

Acromegaly is a chronic disease leading to a multisystem and metabolic disease characterized by GH hypersecretion from adenoma of hypophysis. Even though acro-

megaly leads to insulin resistance and elevated plasma glucose levels, diabetic ketoacidosis complication due to acromegaly is rarely reported in the literature. The prevalence of DM in acromegaly is reported to be 12%–37% as a complication.^[4] Patients with acromegaly apply to hospital with various symptoms and conditions such as acral gigantism, prognathism, vertebral fractures, carpal tunnel syndrome, cardiomyopathy, hypertension, arrhythmia, cardiac valvulopathy, heart failure, insulin resistance, DM, obstructive sleep apnea syndrome, edema due to retention of water, hyperaldosteronism, renal failure, hypogonadism, proximal myopathy, lipolysis, and visceromegaly.^[5] Literature rarely reports diabetic ketoacidosis complications due to acromegaly.^[6] Dosi et al.^[7] reported a 45-year-old male patient hospitalized due to acute complications of diabetes mellitus due to acromegaly. Katz et al.^[8] reported a 40-year-old male patient who was admitted to the hospital with acute complications of diabetes mellitus and who was diagnosed with acromegaly consequently. The cause of DKA in this patient is most likely due to lipotoxicity and glucotoxicity, which results in severe β -cell dysfunction.

The case report of a 43-year-old female patient who had hyperglycemia and DKA as initial findings and hyperhidrosis, secondary amenorrhea, and severe headache complaints in the follow-up period due to acromegaly was presented. It was aimed to emphasize that hypersecretion of GH and IGF-1 might cause ketoacidosis. This reported case reveals the importance of thorough evaluation of any patient with hyperglycemia or DKA that might be secondary to other reasons like acromegaly. Examination for physical disfigurements and consideration of underlying disease or condition may be vital for these patients.

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