

A case of acromegaly with diabetic ketoacidosis as initial presentation

Dr.R.Nagamani*, Dr.Sridhar**, Dr.P.Deepika**

* Department of medicine, Osmania medical collage and hospital

** Departmentof medicine, Osmania medical collage

Abstract- Diabetes mellitus can present in 25% of patients with acromegaly, which is usually neither severe nor symptomatic, and can often be controlled with oral hypoglycemic agents. A subgroup of patients with acromegaly exhibit severe hyperglycemia and require insulin. Diabetic ketoacidosis (DKA) is rare.

Index Terms- Acromegaly,diabetesmellitus,diabetic ketoacidosis , pituitary adenoma.

Case Report

- We present here the case of a young man admitted to osmania general hospital with DKA, as his initial presenting feature of acromegaly.
- This case illustrates the importance of considering an underlying cause, other than type 1 diabetes, as the precipitant of DKA, particularly in individuals with severe insulin resistance requiring large amounts of insulin.
- A 23 year old male patient presented to our emergency department with complaints of polyuria,headache and seizures of generalised tonic clonic type.
- There is no previous history of diabetes mellitus, convulsions, hospitalisation and no family history of diabetes.
- There is no history of trauma and no history of blurring of vision,fever,ear discharge
- No significant medication history.
- On examination patient has coarse facial features wide nasal bridge and thick palms and soles



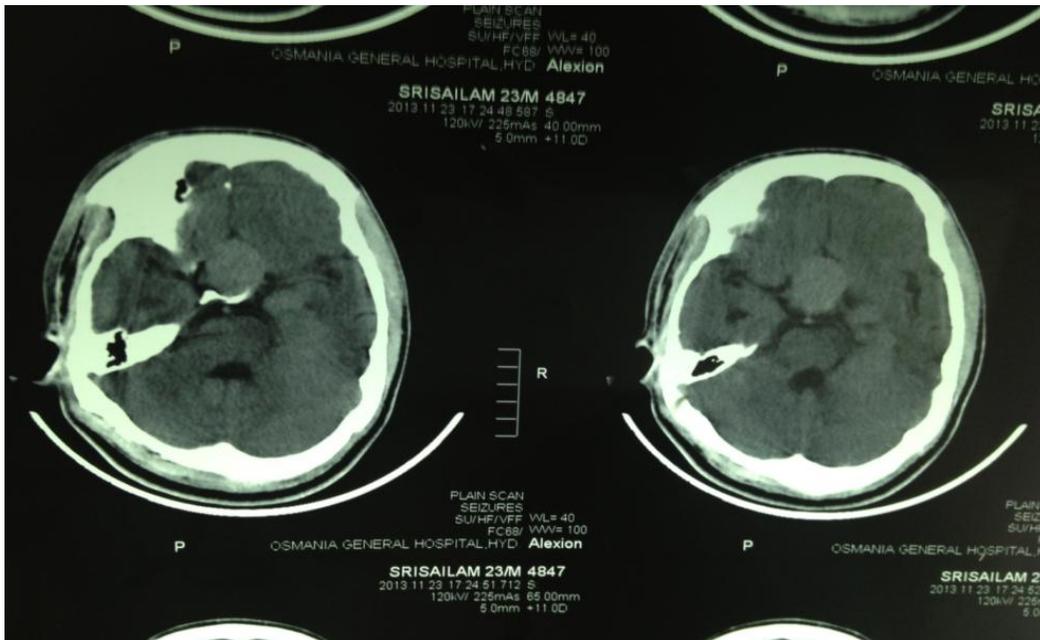
- Patient was in a state of post-ictal confusion and tachycardic with 106 per minute pulse rate. Bp was 100/60 mm of hg
- His GRBS came as high and urine for ketones were positive and ABG was suggestive of metabolic acidosis
- A provisional diagnosis of new-onset diabetes with moderate DKA was made.

- He was admitted to the intensive care unit. Normal saline rehydration and insulin infusion were started as per our institution's DKA protocol.
- His acidosis resolved within 48 hours, and the insulin was changed to a subcutaneous basal-bolus regimen
- His insulin requirements remained unusually high
- His high insulin requirements prompted the addition of metformin and a more extensive evaluation for a cause of his insulin resistance. Physical examination revealed disproportionately large hands and feet with thickening of the soft tissue
- Suspicion of acromegaly was confirmed on the basis of biochemical and imaging findings. Growth hormone (GH), at >40ng/ml (reference range 0-3), and insulin-like growth factor 1 (IGF-1), at 928ng/ml(reference range 116-358), were markedly elevated. Serum TSH,FSH,LH,cortisolare normal.



- Roentgenogram of skull suggestive of widened sella and prognathism
- Magnetic resonance imaging (MRI) brain revealed 3.2*2.5 cm of pituitary macro adenoma. His visual acuity was counting fingers at 3mts and visual field contracted on B/L temporal side and his fundus showing bilateral papilledema.

MRI Brain



- His blood glucose levels normalized postoperatively with complete resolution of diabetes, and insulin was ceased. Hydrocortisone was discontinued before discharge because of normal early morning cortisol levels.

Discussion

- Insulin resistance, glucose intolerance, and diabetes are commonly seen in patients with acromegaly.
- An analysis of the risk factors promoting glucose intolerance in acromegaly revealed that higher GH levels, older age, and longer duration of disease predicted a tendency to develop symptomatic diabetes
- Evidence suggests that both GH and IGF-1 excess can induce insulin resistance directly in the liver, adipose tissue, and muscle, leading to increased endogenous glucose production, decreased muscle glucose uptake, and rising blood glucose levels.
- Elevated levels of these hormones in the presence of relative insulin deficiency are thought to lead to DKA. DKA therefore develops in the presence of an absolute or relative deficiency of insulin together with increased levels of counterregulatory hormones (cortisol, catecholamines, glucagon, or GH).
- high GH levels may inhibit fatty acid metabolism, increasing lipolysis and leading to ketosis. Glucagon has also been considered as a possible contributing factor to DKA and may be increased in acromegaly.
- Excessive glucagon reduces hepatic fructose 2,6biphosphate, a metabolite that inhibits gluconeogenesis in the liver and also induces hepatic ketogenesis. Together with insulin deficiency, glucagon may therefore play a role in the pathogenesis of DKA in acromegaly. Increased levels of GH and glucagon, even in the presence of insulin, may be enough to shift the balance towards ketogenesis and ultimately DKA.

- When acromegaly is treated, diabetes will often resolve with normalization of the patient's OGTT. Patients with a shorter duration of acromegaly and lower GH levels before surgery are more likely to show a reversal of their impaired glucose tolerance.

References

- Waterhouse M, Sabin I, Plowman N, Akker S, Chowdhury T A “growing cause” of diabetic ketoacidosis. BMJ Case Rep. Electronically published 3 April 2009 (bcr11.2008.1226)
- Reddy R, Hope S, Wass J: Acromegaly. BMJ **341**:c4189, 2010
- Chen YL, Wei CP, Lee CC, Chang TC: Diabetic ketoacidosis in a patient with acromegaly. J Formos Med Assoc **106**:788–791, 2007
- Ezzat S, Forster MJ, Berchtold P, Redelmeier DA, Boerlin V, Harris AG: Acromegaly: clinical and biochemical features in 500 patients. Medicine **73**:233–240, 1994
- Lakhotia M, Mathur R, Singh H, Gahlot A, Tiwari V, Gahlot RS: Diabetic ketosis as a presenting feature of acromegaly. J Assoc Physicians India **55**:377–378, 2007
- Nabarro JDN: Acromegaly. Clin Endocrinol **26**:481–512, 1987
- Kreze A, Kreze-Spirova E, Mikulecky M: Risk factors for glucose intolerance in active acromegaly. Braz J Med Biol Res **34**:1429–1433, 2001
- García-Estévez DA, Araújo-Vilar D, Cabezas-Cerrato J: Non-insulin-mediated glucose uptake in several insulin-resistant states in the postabsorptive period. Diabetes Res Clin Pract **39**:107–113, 1998

AUTHORS

First Author – Dr.R.Nagamani, MBBS, MD (GENERAL MEDICINE), Professor of medicine, Osmania medical collage and hospital, Hyderabad, INDIA, Email- ramapantula@yahoo.com

Second Author – Dr.Sridhar, MBBS, MD (GEN MED), Assistant professor of medicine, OMC, Hyderabad

Third Author – Dr.P.Deepika, MBBS, MD (GEN MED), Senior resident OMC, Hyderabad

Correspondence Author – Dr.R.Nagamani (+919848175551)