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Diabetic Ketosis As An Initial Presenting Symptom Of Acromegaly

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ABSTRACT

Case report: *A 45 year old female was admitted with complains of chronic headache, breathlessness on exertion, increased frequency of micturation and giddiness.*

Findings: *Clinical and laboratory findings suggestive of diabetic ketosis and features of acromegaly. A pituitary macroadenoma was detected on contrast enhanced magnetic resonance imaging of brain and pituitary gland.*

Conclusion: *Diabetic ketosis is an uncommon initial presentation of acromegaly.*

Key Words: Acromegaly, diabetic ketosis, pituitary macroadenoma.

INTRODUCTION

Acromegaly is characterized by autonomous secretion of GH, which is usually due to a pituitary somatotropic adenoma. Growth Hormone (GH) stimulates synthesis and secretion of Insulin like Growth Factor (IGF1), especially in the liver[1]. The clinical manifestations of acromegaly are unique and seen on physical examination with a wide range of endocrine, musculoskeletal, cardiovascular, respiratory, and metabolic morbidities[2]. Acromegaly occurs with a prevalence of 50 to 70 cases per million and an incidence of 3 cases per million per year[3]. Diabetes mellitus (DM) is more prevalent in patients with acromegaly, a higher rate attributed to increased insulin resistance caused by GH excess[4]. The prevalence of diabetes among patients with acromegaly has been reported to range from 19 to 56%[5]. Diabetic ketosis as an initial presenting feature of acromegaly is rare. We present here a case of 45 year old female that presented to us in ketosis with hyperglycemia, requiring high dose of insulin for blood sugar level control and later on was diagnosed to have acromegaly.

CASE REPORT

A 45 year-old female was admitted with history of complains of breathlessness on exertion, increased frequency of micturation and giddiness. The complains 1st began with breathlessness on exertion since 15 days and increased frequency of micturition since 15 days, both gradually progressive in nature, and also associated with giddiness since 15 days. No complain of chest pain, palpitation, sweating, fever, burning micturition. On further inquiring patient also gives history of chronic headache, dull aching throughout a day since 6 years, not associated with any visual complains.

Past medical history of menorrhagia for which hysterectomy was done 5 years back.

On her General examination the patient was dehydrated, her pulse rate was 94/min. regular, blood pressure was 130/80 mmHg and respiratory rate was 24/min. On investigation, her random blood sugar level was 540 mg/dl and on spot urine analysis she had 4+ ketonuria. On her physical examination patient's height was 158cm , weighing 59kg with a body mass index (BMI) 23.63 kg/m^2, prognathism, enlargement of lower lip, nose, hand and foot, macroglossia, large forehead.
Her rest of the systemic examination were within normal limit.There was no previous history of any chronic disease, neither any of his family members suffered from any DM, growth disorders or other endocrinopathies.

Diagnostic Workup

After hospitalisation, treatment of diabetic ketosis was started including fluid supplementation, intravenous regular insulin and potassium supplementation, with regular blood sugar, urine ketones and vital charting. The patient's ketosis was controlled within 48 hours. The one day requirement of regular Insulin was about 270 units. It required about 480 units of regular insulin to control her ketosis.

Her laboratory investigation on admission were as follows

PARAMETER	RESULT	REFERENCE RANGE
Hemoglobin	11 gm/dl	12-16.2 gm/dl
TLC	5450 /cmm	4000-10000 /cmm
Platelets	325000 /cmm	165000 – 415000/ cmm
Urea	13 mg/dl	7-20 mg/dl
Serum Creatinine	0.4 mg /dl	0.5 -1.2 mg/ dl
Serum Sodium	135 meq/L	135-145 meq/L
Serum Potassium	2.7 meq/L	3.5-5.5 meq/L
Serum cholesterol	342mg/dl	150-239mg/dl
Serum triglyceride	576 mg/dl	<150 mg/dl
HDL	29	40-60
Free T3	1.89 pg/ml	2.77-5.77pg/ml
Free T4	0.63 ng/dl	0.78-2.19 ng/dl
TSH	2.95 uIU/ml	0.4-4.6 uIU/ml
Serum cortisol 8(AM)	16.75 ug/dl	5-23 ug/dl
IGF-1 (somatomedin C)	604 ng/ml	101-267 ng/ml
Prolactin	13.10 ng/ml	3-18.6 ng/ml
Serum LH	0.216 miu/ml	11.3-39.8 miu/ml
Serum FSH	0.92 miu/ml	21.7-153 miu/ml
Growth Hormone	31.30 ng/ml	0.05-8 ng/ml
HbA1c	>14%	4.3-6.0%
C peptide	1.3 ng/ml	0.8-3.1 ng/ml

Patient's Arterial Blood Gas (ABG) report was within normal limit. Ophthalmologic examination with formal visual field testing was unremarkable.

Contrast Enhanced Magnetic Resonance Imaging of Brain and Pituitary Gland was suggestive of Homogeneously enhancing sellar and suprasellar solid lobulated mass lesion of measuring 16x24x17 mm in size is seen. Superiorly the lesion is seen indenting optic chiasma and displacing supraclinoid portion of bilateral internal carotid artery with encasement on right side of ICA. Inferiorly the lesion is causing bony thinning and expansion of sella with minimal extension to sphenoid sinus, suggestive of Pituitary macroadenoma.

With this, she was diagnosed to be a case of acromegaly secondary to pituitary macroadenoma with secondary diabetes mellitus presented in diabetic ketosis and dyslipidemia with seconadary hypothyroidism with secondary hypogonadism. During the hospital stay, patient's blood sugar and ketosis was controlled with insulin, oral hypoglycemic agent, fluids and electrolyte balance. Patient was later shifted under neurosurgery for surgical resection of macroadenoma. Patient was operated for macroadenoma by transphenoidal approach and resection of pituitary macroadenoma was done. Post- surgery patient was vitally stable and after 1 week of surgery the insulin requirement drastically got reduced to 12 units of long acting insulin per day.

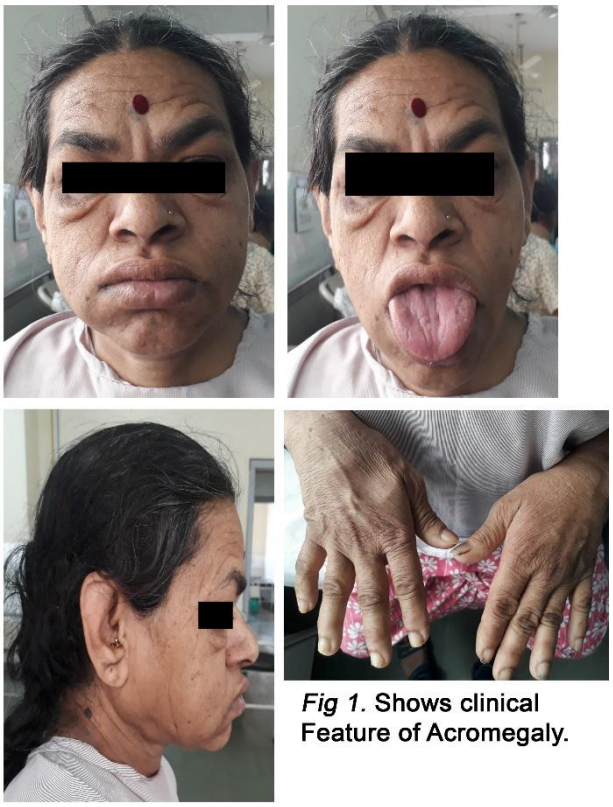

Fig 1. Shows clinical Feature of Acromegaly.

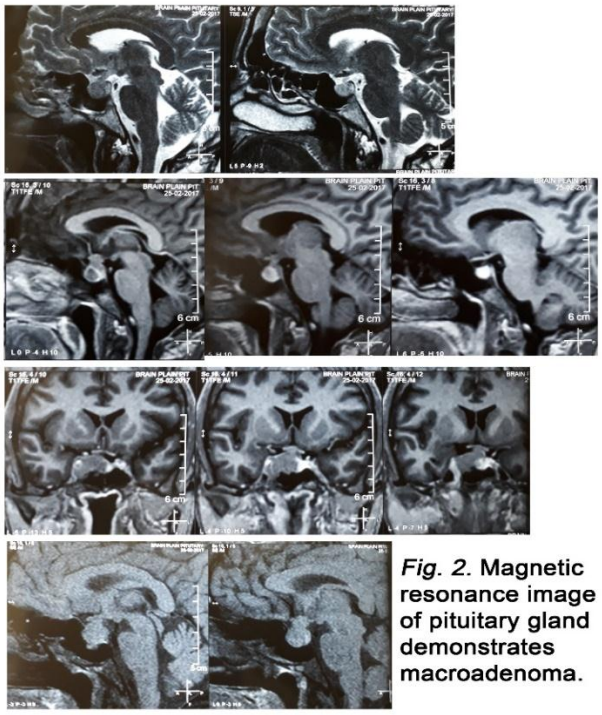

Fig. 2. Magnetic resonance image of pituitary gland demonstrates macroadenoma.

DISCUSSION

Impaired glucose tolerance (IGT) and overt diabetes mellitus (DM) are frequently associated with acromegaly with a prevalence of DM approximately 20-25%[4, 6]. But only some of the patients who have diabetes are symptomatic and will require oral hypoglycemic agents but around 25% of the patients will require Insulin. The diabetes mellitus in acromegaly is due to the excess of the growth hormone (which itself counteracts the action of insulin) and increase in IGF-l levels (which also increases insulin resistance). So, it is the elevated growth hormone and IGF-1 levels that causes excess hepatic glucose production and defective utilization at periphery.[7,8] Treatment of acromegaly with transphenoidal resection of the tumor, significantly reduced insulin and oral anti-hyperglycemic medication treatment, which were completely discontinued within 1 to 20 weeks postsurgery[9]. Diabetic ketoacidosis as an initial presentation is further rare in acromegalics.[7,8]. Diabetic ketosis requires absolute deficiency of Insulin and relative excess of glucagon for its development. However in most of the cases reported in the past, as well as in our case, there is no absolute deficiency of insulin. The normal C-Peptide level speaks for the normal level of insulin. It is assumed that this normal level of insulin is inappropriate to the level of hyperglycemia and in this situation the presence of high levels of counter regulatory hormones leads to diabetic ketosis.[10,11].

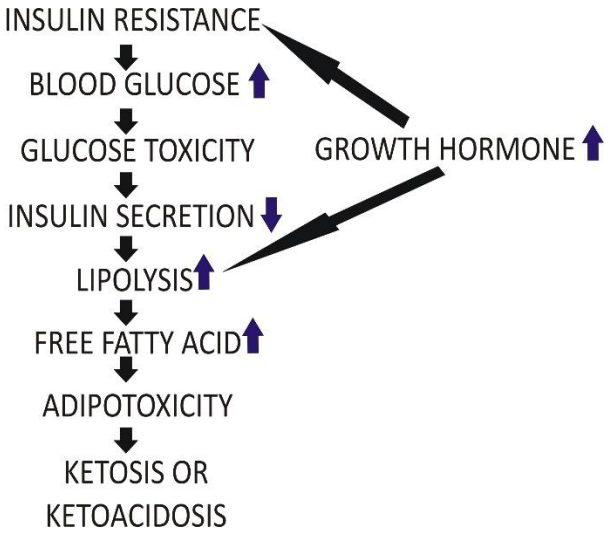

Fig. 3[12]. Schematic of the possible mechanisms by which metabolic derangement in acromegaly promotes development of ketosis or ketoacidosis.

CONCLUSION

Acromegaly is a rare disease caused due to excessive secretion of growth hormone and insulin like growth factor type 1 due to pituitary adenoma. Early recognition and treatment of disease helped in prognosis of disease and arrested the complications like hypertension, cardiomyopathy, diabetes mellitus, visual disturbances etc. Hence early recognition and management is a key to success in better prognosis and improved quality of life.

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