

Case Report

A YOUNG FEMALE CASE OF ACROMEGALY PRESENTING WITH DIABETIC KETOACIDOSIS

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ABSTRACT

Diabetes mellitus is reported to be common in cases with acromegaly. However, diabetic ketoacidosis (DKA) is rarely reported in this disease. Here is a 17 year old female case with acromegaly who presented to our hospital with the first presentation was DKA.

Keywords: Acromegaly, diabetic ketoacidosis, pituitary macroadenoma.

INTRODUCTION

Diabetic ketoacidosis is an acute complication of diabetes mellitus (DM), especially type 1. The presence of excess of growth hormone in acromegaly impairs the glucose metabolism,^{1,2} but most of the patients will not require the treatment to control blood sugar levels.³ Diabetic ketosis as a presenting feature of acromegaly is rare.

We present here a case that presented to us in ketosis with hyperglycemia and later on was diagnosed to have acromegaly. Acromegaly is not common among Nepalese population and, its first manifestation as DKA is even rarer. Psychological implication of her physical appearance was other consideration in this case who had periods of increasing distress, sadness, weakness, disturbed sleep and death wishes leading to suicide attempts.

CLINICAL CASE

A 17 years old female, resident of Saptari, Nepal, came to medical out-patient clinic and was admitted in medical ward with:
Clinical features of progressive and excessive increase in height since six years which was insidious in onset. She was remarkably taller than

her peers. She gave history of her shoes getting tighter quickly; also her hands and feet growing excessively larger. This was associated with difficulty in sitting, walking or doing normal things. This was associated with hyperhidrosis, mild headache and progressive blurring of vision. During the course, since six months she also started having osmotic symptoms like increase in thirst, increased appetite and increased frequency of urine. She did not have menstrual irregularities, hot or cold intolerance, skin changes, galactorrhoea, palpitations, vomiting, or seizures.

On Physical examination, her vital parameters were within normal range: Height: 220 cm (7'3"), Weight: 95 kg, BMI: 19.63 kg/m², large head with frontal bossing, large hands and feet, prominent jaws macroglossia, gap in lower incisor teeth (Figure-1). Visual field showed decreased (100 deg) lateral gaze. Other systemic examinations were normal.

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Fig 1: shows linear height as compared to normal individual (left) and gap in lower incisor teeth (right) and large feet (below).

Laboratory Finding showed: Random blood sugar (21.05 mmol/L, $n < 11.11$ mmol/L), Fasting blood sugar (24.33 mmol/L, $n < 7.0$ mmol/L), Post Prandial blood sugar (28 mmol/L, $n < 11.11$ mmol/L), Glycosylated Hemoglobin (HbA1C) (15%, $n < 7\%$) T.S.H (1.04 micro u/l, $n = 0.39-6.16$), fT3 (2.94 pg/ml, $n = 1.40-4.20$), fT4 (1.02 ng/dl, $n = 0.8-2.2$), L.H (15.21 miu/ml, $n = 0.5 - 10.5$), F.S.H (25.98 miu/ml, $n = 3.0 - 12.0$), Serum Prolactin (9.32 ng/ml, $n = 1.2 - 19.5$) Insulin like growth factor-1 (IGF-1) levels and Growth hormone (GH) facility not available in our hospital so were not done. Magnetic Resonance Imaging (MRI) head showed homogeneously enlarged pituitary gland with minimal suprasellar extension along with widening of pituitary fossa suggestive of macroadenoma (Figure 2).



Figure 2. Magnetic resonance image of pituitary gland demonstrates macroadenoma



She was found sad most of the time during ward stay. Upon further enquiry, her sleep was disturbed and there were periods when she used to be persistently more distressed about her physical appearances. She had attempted suicide twice due to social reasons and embarrassment working with peer groups, difficulty getting enough food for her body requirement, poor socioeconomic status and difficulty getting medicines, according to her. After Psychiatry consultation; Fluoxetine, an SSRI was given with the impression of 'moderate depression' in recurrent depressive disorder.

With this, she was diagnosed to be a case of acromegaly secondary to macroadenoma with secondary diabetes mellitus presented in Diabetic ketoacidosis and dyslipidemia (hypertriglyceridemia) with moderate depression. During the hospital stay, patient blood sugar and DKA was controlled with insulin, fluids and electrolyte balance.

At discharge patient was reassured, continued on insulin 30/70, anti depressants advised for follow up with the report of TFT, LH, FSH, Prolactin and plan for starting medical therapy (Octeotride subcutaneous). She was also advised to consult a neurosurgeon regarding need of surgery/radiotherapy.

Discussion:

Acromegaly occurs with a prevalence of 50 to 70 cases per million and an incidence of 3 cases per million per year.⁴ The clinical features of acromegaly are due to local effects of an expanding sellar mass and the direct as well as indirect effects of excess GH and its principle mediator, IGF-I on peripheral tissues.⁵ The characteristic features are macrognathia, macroglossia, and enlargement of the hands and feet.⁶ Neurologic manifestations of acromegaly include headache, carpal tunnel syndrome, proximal muscle weakness, and a mixed sensorimotor neuropathy.⁷ In our case, all the clinical features were consistent with acromegaly, and the MRI showed a pituitary micro adenoma also.

The growth hormone (GH) levels are greater than 5 ng/mL on fasting basal samples but in healthy subjects minimal exercise may elevate the GH level significantly⁸ confounding the interpretation of the test results. For these reasons, dynamic GH testing using the OGTT is the most specific test for establishing the diagnosis.⁹ In one of the case series of 216 patients with acromegaly, seven had basal GH levels lower than 5 ng/mL. However Zimmermann et al, 2011 still quote failure of an oral glucose load to suppress GH secretion to levels less than 2 ng/mL as the diagnostic criterion.¹⁰ In the present case, it could not be done because of the unavailability of the facility in our local set up.

Acromegalics are found to have impaired glucose tolerance and 30% to 56% of them will develop frank diabetes mellitus.^{11,12} 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-I 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.^{13,14} Since some degree of insulin resistance is present in most patients with DKA,¹⁵ an elevated plasma GH is not necessary for maintaining the insulin resistance during DKA.¹⁶ These findings suggest that GH has a small role in the pathogenesis of DKA, which explains the rarity of DKA in patients with acromegaly.

Our patient also had high blood sugar and needed higher doses of insulin for treatment of DKA, which was due to the insulin resistance caused by excess amounts of GH. Although, it is very rare, but DKA could be the primary manifestation of acromegaly.

In 1951 Bleuler et al¹⁷ concluded that acromegaly was not associated with psychotic disorders, whilst others have reported marked psychomotor retardation and depression. However, in a study of

51 patients with acromegaly, reported by Lishman et al¹⁸, there was no increase in psychiatric morbidity in general or in depression in particular. In patients with well established physical features of acromegaly, the associated stress may manifest as psychological symptoms similar to our case.

CONCLUSION

In conclusion, this case shows the importance of insulin resistance due to growth hormone excess in the presence of relative insulin deficiency as a cause of diabetic ketoacidosis.

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