



## CASE REPORT

# A case of acromegaly complicated with diabetic ketoacidosis, pituitary apoplexy, and lymphoma



He-Jiun Jiang, Wei-Wen Hung, Pi-Jung Hsiao\*

*Division of Endocrinology and Metabolism, Department of Internal Medicine,  
Kaohsiung Medical University Hospital, Kaohsiung Medical University, Kaohsiung, Taiwan*

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### KEYWORDS

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**Abstract** Acromegaly is always complicated with comorbidities and increased mortality. The disease activity and mortality outcomes are highly correlated to the level of growth hormone and insulin-like growth factor 1. A variety of clinical manifestations of acromegaly have been reported. We present a unique case where a 49-year-old male was diagnosed with acromegaly with a first manifestation as an episode of diabetic ketoacidosis. Because he refused any suggestion of treatment, a recurrent episode of diabetic ketoacidosis with pituitary apoplexy occurred. A huge B-cell lymphoma displaying as a huge facial mass followed within 1 year of the diagnosis of acromegaly. Death from advanced cancer ensued 3 years later. This clinical experience strongly reinforces the urgency of controlling growth hormone and insulin-like growth factor 1 as soon as possible once acromegaly is diagnosed.

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## Introduction

The clinical characteristics of acromegaly are mainly attributed to a sustained hypersecretion of growth hormone (GH) by pituitary adenoma. The population incidence

of acromegaly is 3–4 cases per million, and its prevalence is about 60 cases per million [1,2]. Complications of acromegaly broadly involve rheumatologic arthropathies, neuropathies, cardiomyopathy, metabolic abnormalities, respiratory compromise, and neoplasia. The prognosis and outcome of acromegaly depends on whether or not it is treated [1,3].

Pituitary apoplexy is defined as pituitary hemorrhage occurring spontaneously in a preexisting nonfunctional or functional pituitary macroadenoma. Its reported incidence is very low (0.6–10.5%) [4,5]. This life-threatening consequence is a clinical challenge owing to complications

\* Corresponding author. Division of Endocrinology and Metabolism, Department of Internal Medicine, Kaohsiung Medical University Hospital, Kaohsiung Medical University, 100 Tzyou 1st Road, Kaohsiung 807, Taiwan.

E-mail address: [pjhsiao@cc.kmu.edu.tw](mailto:pjhsiao@cc.kmu.edu.tw) (P.-J. Hsiao).

including severe headache, risk of vision loss, cardiovascular collapse, and endocrine crisis [6].

Overall, mortality from acromegaly has been estimated to increase 2–3-fold, and death is likely to occur 10 years earlier than in comparably healthy individuals [7]. As reported in series studies, 60% of patients died of cardiovascular diseases, 25% of respiratory diseases, and 15% succumbed to cancer [2]. The comorbidity and mortality of acromegaly are correlated to the levels of GH prior to and after treatment, the insulin-like growth factor I (IGF-1) level, and the patient's age, tumor size, degree of tumor invasion, and duration prior to diagnosis [1].

Here, we describe a patient with acromegaly first presenting with an episode of diabetic ketoacidosis (DKA). Recurrent DKA ensued concurrently with pituitary apoplexy. A malignant lymphoma grew rapidly within 1 year.

## Case report

In August 2008, a 49-year-old male was sent to the emergency unit because of polyuria, polydipsia, and shortness of breath for 2 weeks. He also had a rapid body weight loss of 17 kg within 1 year. He denied any previous illness or contributory family history. DKA was diagnosed according to blood glucose of 592 mg/dL, blood ketone 4.3 mmol/L, and metabolic acidemia (pH 7.30,  $p\text{CO}_2$  28.5 mmHg, bicarbonate 14.7 mmol/L). Newly diagnosed diabetes mellitus was established by hemoglobin A<sub>1c</sub> (HbA<sub>1c</sub>) 17.8%, and screening for negative glutamic acid decarboxylase autoantibody.

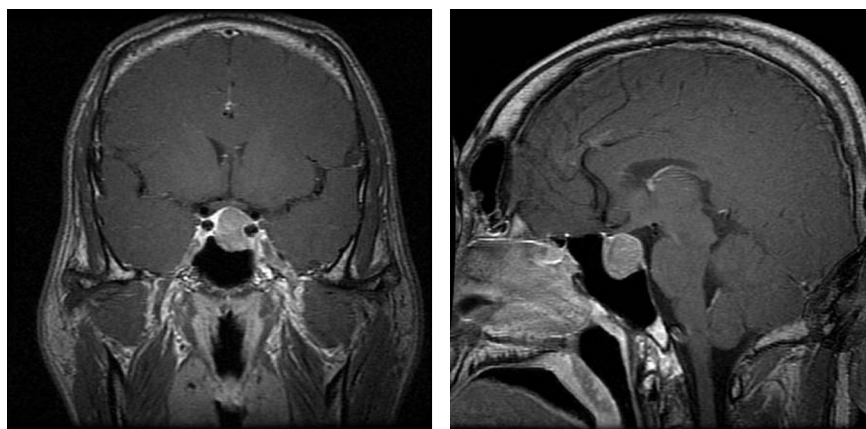
Acromegaly was suspected because typical characteristics were noted including body height of 185 cm, body weight of 87 kg, coarse facial features, large fleshy nose, frontal bossing, jaw prognathism, acral overgrowth, and thickening skin. Acromegaly was confirmed by elevated GH 180.1  $\mu\text{IU/mL}$  (normal  $<15 \mu\text{IU/mL}$ , equal to 5  $\mu\text{g/L}$ ), IGF-1 839.5 ng/mL (normal 41–272 ng/mL for males 40–50 years of age), and prolactin 293.4 ng/mL (normal 1–18 ng/mL). Imaging studies revealed sellar enlargement with a double floor contour of the sella turcica by skull X-ray and pituitary macroadenoma (1.88 cm  $\times$  1.80 cm  $\times$  2.11 cm) by magnetic resonance imaging (MRI; Fig. 1). Diabetes secondary to acromegaly was diagnosed and insulin therapy was used to

control blood glucose. Transphenoidal surgical intervention was highly recommended by an experienced neurosurgeon. However, this patient refused treatment and was lost to follow-up after discharge.

Unfortunately, the patient returned to our hospital due to a sudden onset of severe headache and a recurrent DKA episode 2 months later. Pituitary apoplexy was documented by sella MRI (Fig. 2) showing a marked hemorrhage density over the pituitary macroadenoma with interval enlargement (2.20 cm  $\times$  1.96 cm  $\times$  2.00 cm). Because there was no evidence of ophthalmoplegia or pituitary failure (cortisol 9.2  $\mu\text{g/dL}$ , TSH 0.76  $\mu\text{U/mL}$ , Free T4 0.7 ng/dL), his condition was improved by supportive care. Surgical intervention, radiotherapy, and intensive insulin therapy were well indicated and advised during a team conference by several experts, but the patient denied further treatment again without visiting our outpatient clinic.

In June 2009, the patient visited our endocrine clinic due to a progressive bulging mass over the right face within 2 weeks. He presented with complications from hyperglycemia once again (blood glucose 764 mg/dL, blood ketone 3.4 mmol/L, and HbA<sub>1c</sub> 17.5%) owing to interruption of the insulin injection. He also declared that he had sought alternative traditional herb medicine within this period. A computed tomography scan displayed a soft tissue mass 6.4 cm  $\times$  4.9 cm  $\times$  4.4 cm in size over the right maxillary sinus with extensive invasion of the sinus wall, orbital floor, nasal cavity, and cheek (Fig. 3). B-cell lymphoma was diagnosed by biopsy. His endocrine function was assessed as serum GH  $>175 \mu\text{IU/mL}$ , IGF-1 823.10 ng/mL, and prolactin 196.4 ng/mL. Staging and further chemotherapy were suggested by an oncologist, but this patient still insisted on continuing his herbal medicine regimen.

In August 2012, he visited a urologist due to an enlargement of the left scrotum, intolerable dyspnea, and general weakness. Lymphoma with advanced peritoneal and pleural carcinomatosis was identified by a serial examination. The sella MRI showed interval shrinkage of the adenoma (1.46 cm  $\times$  1.80 cm  $\times$  1.80 cm). In the meantime, his GH (78.44  $\mu\text{IU/mL}$ ) and IGF-1 (449.18 ng/mL) declined and hypoglycemia became frequent. As a result of his intractable clinical respiratory failure, he died in September 2012.



**Figure 1.** Pituitary macroadenoma (1.88 cm  $\times$  1.80 cm  $\times$  2.11 cm) shown in T1 phase with contrast (coronal and sagittal view) by magnetic resonance imaging.

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