



Case Report

Pituitary apoplexy associated with cortisol-induced hyperglycemia and acute delirium

Abstract

Pituitary apoplexy indicates pituitary adenoma hemorrhage, which could result in acute pituitary insufficiency and mortality. The typical symptoms are headache, visual disturbance, nausea, vomiting, altered mental status, and panhypopituitarism. However, cortisol-induced hyperglycemia and acute delirium could be an initial presentation of a pituitary adenoma hemorrhage with stormy release of the adrenocorticotrophic hormone. A 28-year-old woman presented with severe vomiting, irritable state, and delusion. She had medical history of irregular menstrual cycles and marked body weight gain after her second childbirth 8 years ago. She was diagnosed of diabetic ketoacidosis 2 days before this visiting at local medical department. On physical examination, Cushing appearance without definite neurological deficit was disclosed. Further blood tests revealed high blood sugar, cortisol, and adrenocorticotrophic hormone levels without evidence of diabetic ketoacidosis. The brain computed tomography and magnetic resonance imaging showed pituitary macroadenoma and pituitary hemorrhage. Cushing disease with pituitary apoplexy was then diagnosed. Conservative management with delayed neurosurgery was applied. The patient became clear with normalized cortisol and blood sugar levels soon after. Follow-up computed tomography scan of the brain revealed no progression of tumor bleeding or mass effect. To our knowledge, pituitary apoplexy associated with cortisol-induced hyperglycemia and acute delirium has never been reported before. This case reminds us of pituitary apoplexy and its rare manifestations.

Pituitary apoplexy is a rare but life-threatening complication of pituitary adenoma. Death may follow with acute pituitary insufficiency due to pituitary ischemia or hemorrhagic infarction. The typical symptoms are headache, nausea, vomiting, visual disturbance, altered mental status, and panhypopituitarism [1-3].

Cushing disease is caused by hypersecretion of adrenocorticotrophic hormone (ACTH) from pituitary adenoma, which further stimulates secretion of cortisone with characteristic presentation of Cushing syndrome [4]. An episode

of pituitary adenoma hemorrhage could result in stormy release of the hormone with cortisol-induced hyperglycemia and acute delirium. In this article, we report a case of pituitary apoplexy with initial presentation of cortisol-induced hyperglycemia and acute delirium.

A 28-year-old obese woman presented with acute deterioration of conscious level to irritable state and severe vomiting since 1 hour ago. She had medical history of irregular menstrual cycles and marked body weight gain for about 75 kg after her second childbirth 8 years ago. According to her husband's statements, she had been admitted to the local medical department due to loss of consciousness and severe vomiting with hyperglycemia. She was discharged under the diagnosis of diabetic ketoacidosis 2 days before this visiting. The patient had no headache, abdominal pain, diarrhea, or fever accompanied. Besides, there was neither head injury nor other oral medication taken after discharge. On the arrival to our emergency department, she was waving her arms and legs vigorously with persecution delusions. On physical examinations, the patient was irritable and could not cooperate. She was afebrile with elevated blood pressure (156/114 mmHg). Moon face, buffalo hump, central obesity, and abdominal striae with skin pigmentations were noticed (Figs. 1 and 2). Neurological examinations showed a supple neck, symmetric pupils, and normal light reflexes. There were neither focal neurological deficits nor visual field defects by confrontation test.

A finger prick blood sugar test was checked immediately and showed 247 mg/dL. Further blood tests revealed a cortisol level of 32.8 μ g/dL, a positive blood ketone body, a sodium level of 140 mEq/L, and a potassium level of 2.8 mEq/L. The blood gas result revealed metabolic alkalosis with a pH: 7.55 and a bicarbonate of 32.2 mmol/L. Complete blood count, liver and kidney function were within normal limit. The diagnosis of diabetes ketoacidosis was excluded after the initial studies, and brain computed tomography (CT) was then arranged for possible brain lesions causing the delusions and the high blood cortisol level. The scan showed a pituitary gland tumor sizing 1.6 \times 2.0 cm (Fig. 3). Emergent magnetic resonance imaging (MRI) of the brain was performed afterward to check for tumor bleeding, and the result was compatible with pituitary macroadenoma and pituitary hemorrhage (Fig. 4). Cushing disease with pituitary apoplexy was then diagnosed. The patient was admitted with



Fig. 1 Moon face and buffalo hump.

intensive follow-up of the cortisol level. Besides, supportive measures, fluid and electrolyte replacement, and close monitoring were applied. Further laboratory tests revealed panhypopituitarism except a peak ACTH level of 123 pg/mL. Follow-up CT scan of the brain revealed no progression of tumor bleeding or mass effect. The patient became clear with normalized cortisol and blood sugar level soon after. Further neurosurgery was considered.

Pituitary apoplexy is often the first indication of a pituitary adenoma. The clinical manifestations are characterized by the sudden onset of headache with visual disturbance or altered mental status with panhypopituitarism [1-3]. However, clinically silent pituitary hemorrhage accounts for 25% of surgically removed pituitary adenoma [4]. Moreover, MRI is more sensitive than CT scan and is the image study of choice [2,3]. The precipitating factors of pituitary apoplexy include trauma [5], hypertension [3], surgical procedures [6], and use of bromocriptine [1], aspirin therapy [3], and pregnancy [7]. It may be fatal when acute pituitary insufficiency with adrenal crisis occurs. Besides,



Fig. 2 Central obesity and abdominal striae with skin pigmentation.



Fig. 3 The CT scan of the brain showing a pituitary gland tumor.

severe mass effect may result in visual compromise [1-3]. The initial management consists of monitoring of fluid and electrolyte balance with replacement of deficient hormones, in particular corticosteroids [1,3]. The timing of decompression surgery remains controversial [8,9].

In our case, pituitary apoplexy is the first manifestation of a pituitary adenoma. Unlike the typical presentations of pituitary apoplexy, our patient presented to our department with hyperglycemia and acute delirium. With high blood ACTH and cortisol levels and a pituitary adenoma, an ACTH-secreting tumor was suspected. Chronic high ACTH level may have resulted in adrenocortical hyperplasia. The hemorrhage of the tumor induces even more excessive

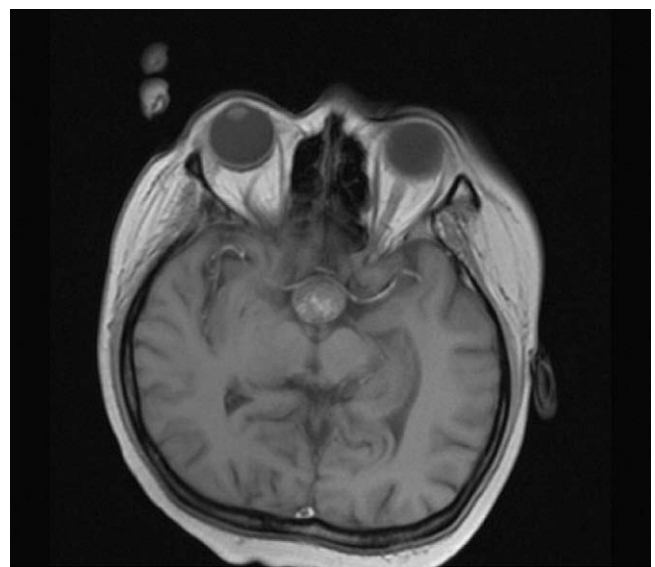


Fig. 4 The MRI of the brain showing pituitary macroadenoma with bleeding.

ACTH release, which causes the adrenal hyperplastic tissue to release a large amount of cortisol; after which, cortisol-induced hyperglycemia and acute delirium developed. After fluid and electrolyte replacement, our patient recovered from these symptoms and signs with normal cortisol and blood sugar levels. The risk of emergent decompression surgery that may lead excessive ACTH secreting status to complete panhypopituitarism was also considered. In addition, no definite signs of acute neuro-ophthalmic deficits were recognized and thus neurosurgery was delayed.

To our knowledge, pituitary apoplexy associated with cortisol-induced hyperglycemia and acute delirium has never been reported before. This case reminds us of pituitary apoplexy and its rare manifestations.

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References

- [1] Cardoso ER, Peterson EW. Pituitary apoplexy: a review. *Neurosurgery* 1984;14:363-73.
- [2] Bills DC, Meyer FB, Laws ER, et al. A retrospective analysis of pituitary apoplexy. *Neurosurgery* 1993;33:602-69.
- [3] Randeve HS, Schoebel J, Byrne J, Esiri M, Adams CB, Wass JA. Classical pituitary apoplexy: clinical features, management and outcome. *Clin Endocrinol* 1999;51:181-8.
- [4] Roelfsema F, Pincus SM, Veldhuis JD. Patients with Cushing's disease secrete adrenocorticotropin and cortisol jointly more asynchronously than healthy subjects. *J Clin Endocrinol Metab* 1998;83:688.
- [5] Wakai S, Fukushima T, Teramoto A, et al. Pituitary apoplexy: its incidence and clinical significance. *J Neurosurgery* 1981;55:187-93.
- [6] Ebersold MJ, Laws Jr ER, Scheithauer BW, et al. Pituitary apoplexy treated by transsphenoidal surgery. A clinicopathological and immunocytochemical study. *J Neurosurg* 1983;58:315-20.
- [7] Wiesmann M, Gliemroth J, Kehler U, et al. Pituitary apoplexy after cardiac surgery presenting as deep coma with dilated pupils. *Acta Anaesthesiol Scand* 1999;43:236-8.
- [8] Onesti ST, Wisniewski T, Post KT. Clinical vs subclinical pituitary apoplexy: presentation, surgical management, and outcome in 21 patients. *Neurosurgery* 1990;26:980-6.
- [9] Gruber A, Clayton J, Kumar S, Robertson I, Howlett TA, Mansell P. Pituitary apoplexy: retrospective review of 30 patients—is surgical intervention always necessary? *Br J Neurosurg* 2006;20:379-85.