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CASE REPORT

Pituitary Apoplexy Presenting as Unilateral Oculomotor Palsy in a Patient with Diabetes Mellitus

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Abstract

Pituitary apoplexy is a clinical syndrome caused by hemorrhage and/or infarction of the pituitary gland. This study presents a case of a 53-year-old man with a seven-day history of severe headache and left-sided oculomotor palsy, who had pupil-sparing oculomotor palsy with a previous history of diabetes mellitus. Diabetes-related microvascular ischemia was initially considered due to the pupil sparing, before imaging studies. However, pituitary hemorrhage was detected adjacent to the ICA cavernous segment in cranial and pituitary MRI, identified as the cause of the oculomotor nerve palsy. This case is significant for highlighting pituitary apoplexy as a rare cause of oculomotor palsy without pupil involvement in patients with diabetes mellitus.

Keywords: Diabetes mellitus; oculomotor palsy; pituitary apoplexy; pupil sparing.

The most common and urgent cause of oculomotor palsy is a posterior communicating artery aneurysm[1]. Isolated oculomotor palsy rarely occurs due to pituitary adenomas[1]. Pituitary apoplexy is a rare clinical emergency secondary to infarction or hemorrhage of the pituitary gland or adenoma[2]. Presentation of pituitary apoplexy as an isolated oculomotor palsy is very rare[3]. Pituitary apoplexy develops mostly in the presence of undiagnosed pituitary adenomas but may also occur in a normal gland[4]. The most common presenting symptoms are headache, ophthalmoplegia, visual field deficits, anisocoria, neurological deficits, coma, and hemiparesis[4,5]. The most common cause of isolated pupil-sparing third nerve palsies is believed to be microvascular ischemia, frequently associated with diabetes mellitus or systemic hypertension[6]. We present a diabetic patient with unilateral pupil-sparing oculomotor nerve palsy diagnosed with pituitary apoplexy.

Case Report

A 53-year-old man presented with a seven-day history of severe headache and a two-day history of diplopia and left-sided ptosis. He had a medical history of hypertension, operated lung cancer, and type II diabetes mellitus.

Physical examination showed that fever, pulse, and blood pressure values were within normal limits. On neurological

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examination, adduction, elevation, and depression of the left eye were impaired. Pupils were isochoric and reacted to light. No other deficits were identified. These findings were localized to the left oculomotor nerve.

Computed tomographic angiography (CTA) performed for the differential diagnosis of posterior communicating artery aneurysm was normal. The patient received an MRI scan with special sella views. In the left half of the pituitary gland, a 12x7x9 mm sized hyperintensity in the T1 and T2 series and a central isointense-peripheral hypointense area with right-sided diffusion restriction in the diffusion series were observed (Figs. 1-3). The lesion was 30-45 degrees adjacent to the left internal carotid artery cavernous segment. These findings were consistent with subacute bleeding in the left half of the pituitary gland. Preoperative and postoperative pre-contrast and post-contrast pituitary MRI scan images are shown in Figures 4 a-f.

The patient was referred to neurosurgery and underwent transsphenoidal surgery. Pathological investigation revealed a pituitary adenoma with intense granular ACTH expression.

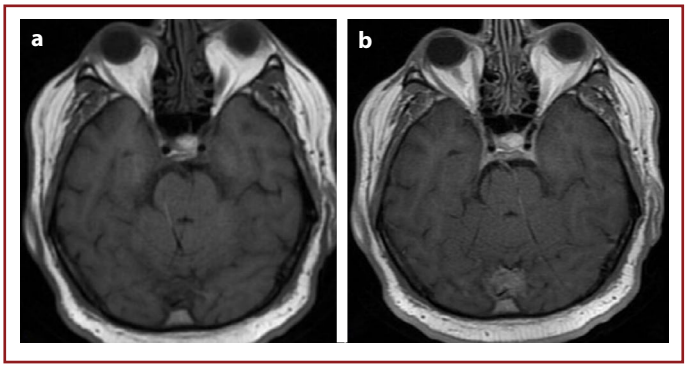

Figure 1. A 53-year-old patient suffered from sudden headache and left oculomotor palsy. **(a)** Pre-contrast Axial T1W and **(b)** Post-contrast Axial T1W images showing a hyperintense heterogeneous pituitary mass.

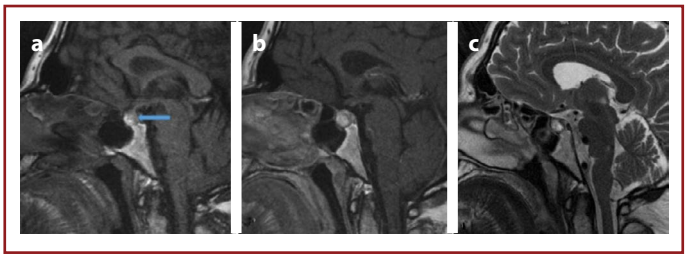

Figure 2. (a) Pre-contrast Sagittal T1W image showing a hyperintense heterogeneous pituitary mass (arrow). **(b)** Post-contrast Sagittal T1W image showing adjacent hypophysis tissue with homogeneous enhancement. **(c)** Lesion appearing as a heterogeneous hyperintense on Sagittal T2W image.

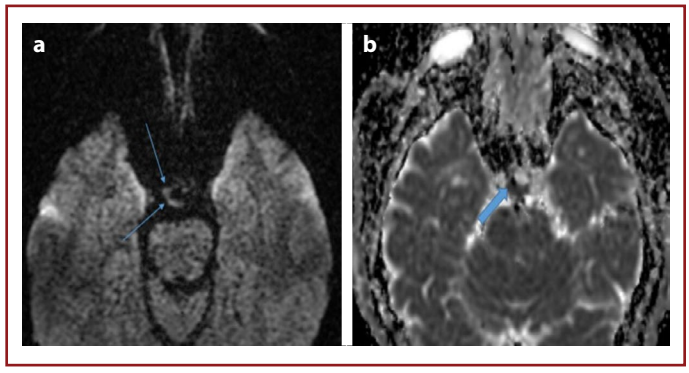

Figure 3. (a) Images showing peripheral diffusion restriction on the same lesion on DWI- B1000 (Thin arrows). **(b)** ADC (Apparent Diffusion Coefficient) (Thick arrow).

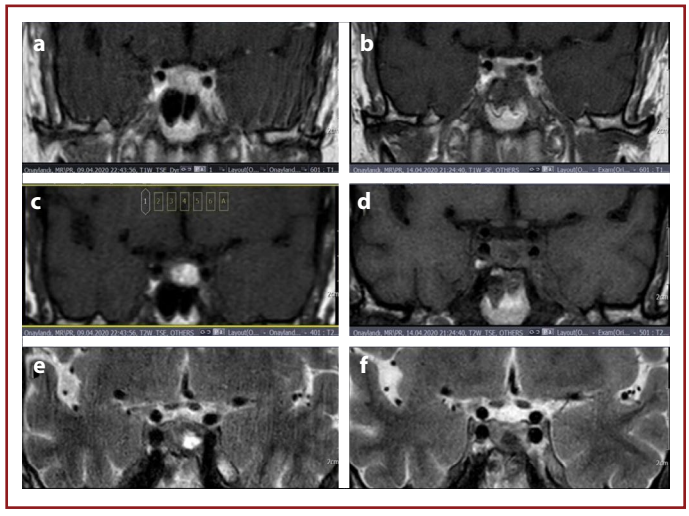

Figure 4. Preoperative (left column); (post-contrast coronal T1 weighted **(a)**; pre-contrast coronal T1 weighted **(c)**; coronal T2 weighted images **(e)** and postoperative (right column; post-contrast coronal T1 weighted **(b)**, pre-contrast coronal T1 weighted **(d)**, coronal T2 weighted images **(f)** pituitary MRI of the patient revealed pituitary apoplexy (pituitary adenoma with bleeding). Postoperative pituitary MRI images showed tumor and blood evacuation and decompression of surrounding neurovascular structures.

Discussion

Pituitary apoplexy is rare and has variable clinical presentations, which can be confused with other conditions, including intracranial hemorrhage, carotid aneurysm rupture, subarachnoid hemorrhage, meningitis, migraine, and transtentorial herniation[7]. It is a clinical syndrome caused by hemorrhage and/or infarction of the pituitary gland, resulting in expansion, edema, and necrosis[8]. Pituitary apoplexy can be associated with certain medications (bromocriptine, anticoagulants, surgery, or radiation), dynamic pituitary function tests, diabetes mellitus, intracranial pressure changes, cerebral angiography, hypertension, pregnancy, thrombolytic

therapy, and head trauma[9,10]. Our patient did not have these risk factors. However, he presented with pupil-sparing oculomotor nerve palsy, which is most commonly caused by microvascular ischemia of the nerve, particularly observed in elderly patients with vascular risk factors such as diabetes mellitus and hypertension. Spontaneous recovery is seen approximately within three months[1,11]. Initially, diabetes-related microvascular ischemia was considered in our patient due to the pupil sparing; before imaging studies. The most common and urgent cause of oculomotor nerve palsy with pupil involvement is aneurysms, primarily in the posterior communicating artery[1,12,13]. In the presence of a compressive lesion, such as an aneurysm or mass, that presses the nerve externally, the parasympathetic (pupillomotor) fibers in the outer part of the oculomotor nerve are primarily affected, resulting in mydriasis and unresponsiveness to light in 95% of the cases. The inner part of the nerve is affected in microvascular ischemia, so the parasympathetic fibers and pupil are not affected in 80% of diabetes-related microvascular ischemia cases[12,13]. CTA angiography was performed to exclude an aneurysm. Pituitary hemorrhage adjacent to the left ICA cavernous segment was detected in cranial and pituitary MRI, identified as the cause of the oculomotor nerve palsy. An isolated sudden oculomotor nerve palsy is a frequent manifestation of pituitary apoplexy[3,13]. The mechanism of oculomotor nerve palsy in pituitary apoplexy is unclear, with several mechanisms proposed. The oculomotor nerve is the most and first affected nerve, being closest to the pituitary gland in the cavernous sinus wall. There may also be multiple cranial nerve involvements (nerves III, IV, V, and VI). Late-onset oculomotor nerve palsy occurs due to the compressive effect of the cavernous sinus by the pituitary mass. Sudden onset oculomotor nerve palsy occurs due to compression of the vasa nervorum of the nerve[3,13]. As seen in our case, the development of acute oculomotor nerve palsy due to apoplexy is attributed to the impairment of the vascular supply of the nerve due to the compression of the vasa nervosa[13].

Pituitary apoplexy is a rare but life-threatening condition. The majority of cases occur in the fifth and sixth decades with male predominance[14]. The demographic characteristics of our case were consistent with these data. Pituitary apoplexy develops mostly in the presence of undiagnosed pituitary adenomas but may also occur in a normal gland[4]. Approximately 80% of cases do not have a prior diagnosis of adenoma[15]. Our case had no previously known diagnosis or complaints of pituitary adenoma.

The most common initial symptom of pituitary apoplexy is sudden onset and severe headache, which was also the initial symptom of our patient. Various degrees of pituitary hormone deficiency are observed in most cases of pituitary apoplexy. If ACTH and TSH deficiencies are not recognized early and precautions are not taken, the development of acute secondary adrenal insufficiency and hypothyroidism may cause serious morbidity and mortality[15]. While free T3/T4 levels were normal in our patient, TSH was found to be low. Hyponatremia is seen in 40% of cases[15]. The serum sodium level was within normal limits in our patient.

Pituitary apoplexy is diagnosed with clinical and hormonal examinations, and pituitary MRI[14,15]. Monitoring of fluid-electrolyte balance and the replacement of deficient hormones are the medical treatments for pituitary apoplexy[15]. Surgical decompression should be applied in cases of severe vision loss, increased intracranial pressure, and altered consciousness[14,15]. Transsphenoidal hypophysectomy was performed on our patient.

Pituitary apoplexy should be considered a rare cause of oculomotor palsy and headache, even in the presence of diabetes mellitus.

Informed Consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

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Conflict of Interest: None declared.

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