

Case Report

무균성 수막염으로 발현한 흔하지 않은 뇌하수체졸종

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A Case of Unusual Pituitary Apoplexy Presented as Aseptic Meningitis

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We encountered a case of pituitary apoplexy who presented with isolated headache and vomiting without visual disturbance or ophthalmoplegia. The cerebrospinal fluid examination was compatible with aseptic meningitis. A computed tomography revealed slightly high density in the pituitary fossa and suprasella area, but the signal change was very faint. Our case suggests that clinicians should take into account the possibility of pituitary apoplexy without visual disturbance or ophthalmoplegia, when aseptic meningitis is suspected.

Key Words: Pituitary apoplexy, Meningitis, Headache

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Pituitary apoplexy occurs when an existing pituitary adenoma undergoes acute hemorrhage or infarcts.¹ It results in severe headache, visual disturbance, ophthalmoplegia, altered consciousness, and hypopituitarism.² Several cases have been reported that pituitary apoplexy can be presented as aseptic meningitis, but all of them showed visual disturbances or ophthalmoplegias within a few days after onset of symptoms.²⁻⁷ We experienced a case who presented with isolated headache and vomiting without visual disturbance or ophthalmoplegia. The clinical diagnosis was

further confounded by the finding of higher cellular cerebrospinal fluid (CSF). Here, we report a case of pituitary apoplexy without visual disturbance or ophthalmoplegia presented as aseptic meningitis.

Case Report

A 49-year-old woman presented with acute onset of severe bifrontal headache for 3 days. She also complained of nausea, vomiting, and febrile sensation. She denied visual disturbances. She had been taking non-steroidal anti-inflammatory drugs (NSAIDs) to release her headache for 3 days, but her headache did not diminish. Her blood pressure was 140/90 mmHg, heart rate 80/min, and body temperature 37.0°C. She was alert and well oriented. The neurological examinations revealed neck stiffness. The cranial nerve examinations including confrontation test for visual field were normal. The range of movement for the

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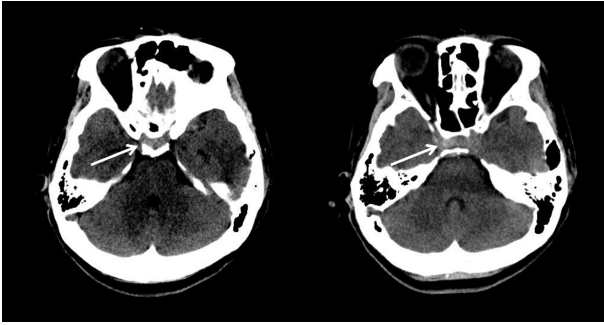


Figure 1. Brain computed tomography (CT) of pituitary apoplexy. CT reveals slightly high density in the pituitary fossa and suprasella area (arrow).

extraocular eye muscles was full. The motor and sensory examinations of limbs revealed no abnormality. The deep tendon reflexes were normal and Babinski's sign was absent. The remainder of the neurological examinations was unremarkable. A computed tomography (CT) without enhancement of the brain revealed so slightly high density in the pituitary fossa and suprasella area (Fig. 1). The complete blood count was normal, although the serum sodium was slightly low at 132 mmol/L. Other laboratory findings including electrolyte profiles, glucose level, urinalysis, renal function test, and liver function test were within normal limits. The analysis of CSF showed leukocyte count of 480/mm³ (55% polymorphs), erythrocyte count of 640/mm³, protein level of 101 mg/dL, and glucose of 46 mg/dL (serum glucose 79 mg/dL). Cultures of the CSF were sterile. The results of antibody studies and polymerase chain reactions for enterovirus, cytomegalovirus, herpes simplex virus and varicella-zoster virus were normal. All of the clinical and laboratory findings led us to diagnose her as aseptic meningitis. The magnetic resonance imaging (MRI) showed acute and subacute hemorrhage within the pituitary gland compatible with pituitary apoplexy (Fig. 2). The hormonal studies revealed that the levels of prolactin, free thyroxine, thyroid stimulating hormone (TSH), adrenocorticotropic hormone (ACTH), cortisol, luteinizing hormone (LH) and follicle-stimulating hormone (FSH) were lower than reference ranges. Eventually, we were able to diagnose her as panhypopituitarism. We started to treat her with levothyroxine and hydrocortisone for hormone replacement. She underwent the ophthalmological examinations such as visual acuity, visual fields, and ocular movements by an ophthalmologist a day before surgery, and the results were absolutely normal. The Humphrey computerized perimetry for visual field test was normal as well.

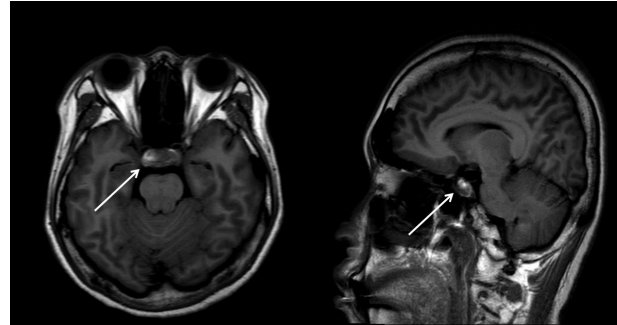


Figure 2. The T1-weighted brain magnetic resonance imaging (MRI) of pituitary apoplexy. MRI shows acute and subacute hemorrhage within the pituitary gland compatible with pituitary apoplexy (arrow).

The removal of the tumor by endoscopic trans-nasal trans-sphenoidal approach improved her headache. The microscopic findings, ghost outlines of the necrotic tumor cells were surrounded by the layers of the hemosiderin laden macrophages and fibrin, were competent to the histology of the total hemorrhagic infarction of pituitary adenoma.

DISCUSSION

The clinical manifestation of pituitary apoplexy can be variable, and reflects tumor expansion and hormone deficiency.² However, in pituitary apoplexy, severe headache and vomiting without other symptoms such as impaired consciousness, decreased visual acuity, visual field defects, or ophthalmoplegia are rare.^{2,8} Isolated headache and vomiting led us to diagnose our patient as aseptic meningitis rather than pituitary apoplexy. It has been known that the sensitivity of brain MRI is higher than that of CT in detecting pituitary apoplexy, and brain CT is not specific for the diagnosis of pituitary apoplexy.⁹ Also, our patient showed very subtle high density in the pituitary fossa and suprasella area. MRI is the choice of radiological investigation in detecting tumor, hemorrhage, or infarction within the pituitary gland.⁹ Visual disturbances and ophthalmoplegias are frequent and classical findings in the diagnosis of pituitary apoplexy. Visual disturbances may result from compressions of the optic nerve or chiasm, and supero-laterally compressions of the cavernous sinus can possibly lead to ophthalmoplegias.¹

The Humphrey computerized perimeter in our patient revealed that there were no visual field defects. This is an extremely unusual case, and has not yet been reported to our knowledge.

However, the previously reported cases of pituitary apoplexy presented as aseptic meningitis had visual disturbances or ophthalmoplegias in the course of the disease.²⁻⁷ The exact mechanism of our patient without visual disturbances or ophthalmoplegias is unknown. However, this could be explained by minimal supero-lateral expansion of the lesion which was not able to compress the optic or oculomotor nerves. Our patient still remained of normal visual acuity, visual fields, and ocular movements even several weeks after onset of symptoms.

If apoplectic tumors bleed into the subarachnoid space, the CSF would exhibit pleocytosis, elevated red blood cell, and elevated protein.⁸ Also, the result of the CSF examination in this case was compatible with aseptic meningitis.

There are several limitations of this case report. First, the previous study suggested that the visual fields by Humphrey perimetry were smaller than those by Goldmann perimetry, but we only used Humphrey perimetry to detect visual field defects.¹⁰ Second, we did not conduct electrophysiological test to detect visual field defects such as visual evoked potentials.

In conclusion, our case suggests that clinicians should take into account the possibility of pituitary apoplexy without visual disturbances or ophthalmoplegias, when aseptic meningitis is suspected. Because the sensitivity of brain MRI is higher than that of CT in detecting pituitary apoplexy, MRI is a useful tool to rule out symptomatic meningitis.

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