Case Report

Pituitary Adenoma Results in the Empty Sella Syndrome

Kaoru NOMURA, Atsushi KONO*, Makoto UJIHARA, Akitsugu MASUDA, Katsuya SAITO** and Kazuo SHIZUME

A 69-year-old female was treated for hyperthyroidism and hypertension. In August 1984, she suddenly began suffering from polyuria and polydipsia. In October, she exhibited fever, headache, vertigo, and poor appetite, probably due to pituitary apoplexy. Her endocrine function was normal, except for partial diabetes insipidus. A contrast-enhanced CT brain scan revealed a pituitary adenoma with a ring-enhanced outer edge and a central low-density area. The MRI scan also indicated cystic adenoma. A CT scan examination repeated 6 months later showed an empty sella with a markedly decreased pituitary adenoma. This case report demonstrates that some empty sella are the final result of pituitary adenoma bleeding or infarction.

Key words: Empty sella syndrome, Pituitary apoplexy, Pituitary adenoma

The empty sella syndrome, initially reported from autopsy findings (1), was diagnosed clinically by the radiological examination. The increased availability of CT and MRI scans now makes it easy to demonstrate empty sellas and, with the help of these techniques, accumulating evidence suggests that pituitary adenomas often cause necrosis (2-5) and may possibly result in empty sellas. This hypothesis, however, remains to be proved. This case report supports, by a follow-up using CT and MRI scans, the hypothesis that empty sellas are caused by previous adenoma infarctions.

CASE REPORT

The patient, 69-year-old female 153 cm tall and weighing 55 kg, first visited a clinic complaining of weight loss, hand tremor, and easy fatigability. She was diagnosed as having hyperthyroidism in 1979 and has been taking propylthiouracil since then. Since 1980, she has been treated at the Mitaka Central Hospital. She was also diagnosed as having essential hypertension and mitral valve prolapse, but responded well to antithyroidal and antihypertensive medication. In August 1984, she suddenly experienced polyuria and polydipsia. In October, she was hospitalized complaining of headache, fever, vertigo, and poor appetite. Her daily urinary volume exceeded 3 liters. Urinary osmolality was 280 mOsm/kg·H₂O even though plasma osmolality was 304 mOsm/kg·H₂O. Her basal plasma levels of adrenal and thyroid hormones were within normal ranges. She recovered well after fluid supplements and was discharged after two weeks. In December, she was admitted to Tokyo Women's Medical College Hospital for further endocrinological and radiological evaluation. Basal and LH-RH-stimulated levels of plasma LH and FSH were low. Other anterior pituitary hormones (ACTH, TSH, prolactin, and growth hormone) were normal as judged by CRF, TRH, and GRF tests. The plasma vasopressin level was relatively low (0.6 pg/ml) when plasma and urinary osmolalities were 294 and 255 mOsm/kg·H₂O, respectively. CT scans with and without contrast enhancement showed pituitary adenoma extending over the sella turcica. The adenoma's center had a low-density area and its surface was ring-enhanced (Fig. 1). The MRI also showed the presence of cystic pituitary adenoma extending over the sella turcica.
Fig. 1. Brain CT (coronal scan) with contrast enhancement, December 1984. The CT scan showed a pituitary adenoma with a central low-density area and peripheral ring-enhancement.

Fig. 2. Brain MRI, December 1984. T2-weighted (SE1200/60) sagittal image. The MRI showed a pituitary adenoma with cystic change.

Fig. 3. Contrast-enhanced brain CT (coronal scan), June 1985. The pituitary adenoma had disappeared and the pituitary fossa was occupied by cerebrospinal fluid.

The patient was diagnosed as having partial diabetes insipidus caused by a nonfunctioning pituitary adenoma. Treatment was started with desmopressin (DDAVP, 0.025 ml, once before sleep), which increased urinary osmolality to 548 mOsm/kg·H2O and reduced nocturnal urination. Reexamination of the pituitary adenoma by CT six months later (May 1985) showed the adenoma to be greatly reduced and an empty sella was observed (Fig. 3). A CT repeated in May 1987 matched that taken in May 1985 (figure not shown), and the patient is doing well.

DISCUSSION

The patient’s nonfunctioning pituitary adenoma extended over the sella turcica and caused partial diabetes insipidus in August 1984. Her first CT scan and an MRI scan showed a pituitary adenoma with central necrosis. CT scans six months, then three years later, showed a collapsed adenoma and an empty sella.

Wakai et al (2) reported that, in 560 cases of pituitary adenomas, 90 (16%) exhibited bleeding in adenoma with or without symptoms of pituitary apoplexy and that, after bleeding, the adenoma showed ring-enhancement on the CT scan. Lindholm et al (3) also reported the high incidence (50%) of symptoms suggesting pituitary apoplexy in 20 cases of empty sella syndromes. The same group also
reported that, in 23 untreated acromegaly patients, 11 had empty sella and 2 had intrasellar cysts. These 13 cases also had symptoms probably due to pituitary apoplexy. Login and Santen reported a case in which the symptoms of acromegaly disappeared without any therapy and found a partial empty sella (5). These reports suggest that the pituitary adenoma often causes necrosis due to bleeding or infarction, with or without symptoms of pituitary apoplexy, and results in empty sella syndrome. Reports (6–10) that the empty sella often accompanied pituitary microadenoma also support this hypothesis. None of these reports showed direct radiographic proof, however.

Recently, Montalban et al. (11) reported three patients with episodes of pituitary apoplexy. CT scans revealed pituitary adenomas. Six to 18 months later, CT scans demonstrated empty sella syndrome in all three cases. Their radiographic follow-ups demonstrated that some cases of empty sella syndrome are the result of pituitary apoplexy in pre-existent adenomas. Our case also demonstrated the same radiographic findings. It had the history which suggested the episode of pituitary apoplexy in October 1984, i.e., headache, fever, vertigo and poor appetite. Although it lacked other symptoms of pituitary apoplexy such as abrupt loss of vision, other cranial nerve deficits, mental obtundity and specific meningeal irritation syndrome, it can occur without any symptoms (2, 5, 12).

In conclusion, our case report, together with those of others, indicates that the pituitary adenoma often causes apoplexy, causing the suprasellar subarachnoid cistern to be lost adequate support, resulting several months later in an empty sella.

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REFERENCES