

—Report on Experiments and Clinical Cases—

Pituitary Insufficiency after Penetrating Injury to the Sella Turcica

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We report a 28-year-old male patient with a pituitary insufficiency after a simple fracture of the sella turcica. He was injured by a long nail that punctured the lower jaw. No fracture other than that of the sella turcica was detected. An endocrinological examination revealed both anterior and pituitary dysfunction and diabetes insipidus that continued for about two months. (J Nippon Med Sch 2000; 67: 130—133)

Key words: head injury, pituitary gland, pituitary insufficiency, diabetes insipidus

Introduction

Pituitary insufficiency is a well-known complication of head injuries. However, the incidence of diabetes insipidus in patients with head injuries is actually lower than 1%, and that of anterior pituitary dysfunctions is even lower¹. A pituitary injury following a skull base fracture is sometimes observed, but one following a simple fracture of the sella turcica is rare^{1,2}. We report a case of pituitary insufficiency after a direct pituitary injury by a foreign object.

Case report

A 28-year-old male accidentally pierced his lower jaw with a nail. The patient was a carpenter who used a machine to drive nails into wood. He was admitted to Higashi-Totsuka Memorial Hospital on August 15, 1996. He was alert and well oriented on admission. He had a puncture wound (1 cm) to the right lower jaw and was unable to open his mouth. There was no neurological deficit. A skull X-ray revealed a long nail

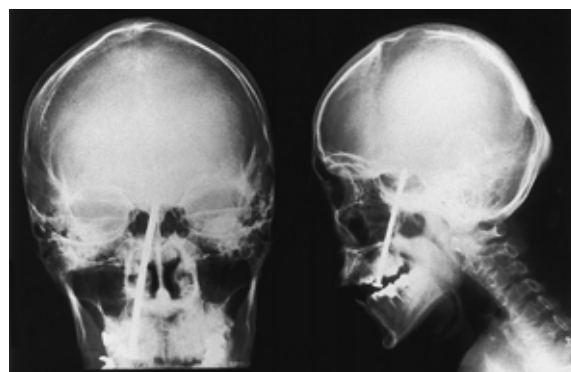


Fig. 1 Skull X-ray revealed a nail penetrating from the lower jaw to the floor of the sella turcica.

about 15 cm long piercing the right lower jaw to the floor of the sella turcica (Fig. 1). The nail did not fracture the mandible or the mandibular teeth. Computed tomography (CT) of the brain showed no intracranial abnormality other than a nail shadow in the sella turcica and the right paranasal cavity. Therefore, we confirmed that the nail fractured the sellar floor and that the dura of the sella floor had been penetrated. However, we could not discern what parts of the sellar contents (anterior or posterior pituitary) were di-

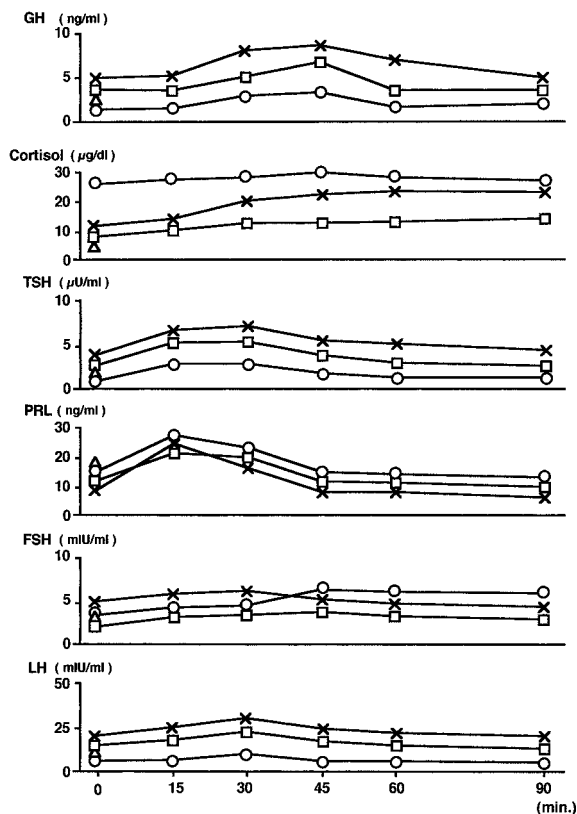


Fig. 2 The endocrinological examination including stimulation tests (regular insulin, 5 international units (IU); thyrotropin releasing hormone, 500 µg; and luteinizing hormone releasing hormone, 100 µg by intravenous injection) of the anterior pituitary hormones on 2, 7, 14 and 30 days after the injury show anterior pituitary dysfunction.
 ○, day 2; △, day 7; □, day 14; ×, day 30.
 The basal values of the hormones are detailed below: growth hormone (GH), under 0.42 ng/mI; thyroid stimulating hormone (TSH), 0.34~3.5 µU/mI; luteinizing hormone (LH), 1.8~5.2 mIU/mI; follicle stimulating hormone (FSH), 2.9~8.2 mIU/mI; prolactin (PRL), 1.5~9.7 ng/mI; Cortisol, 4.0~18.3 µg/dI.

rectly injured. Angiography was immediately performed, showing no vascular injury. Then, the nail was carefully removed. After removal, a brain CT revealed no intracranial abnormality other than a pneumocephalus in the suprasellar cistern and sella turcica. The intracranial air disappeared within a few days. There was no rhinorrhea or otorrhea, and cellular counts of the cerebrospinal fluid obtained by the lumbar tap were normal.

The patient presented with polyuria and polydipsia on the day of admission. The sudden onset of polyuria

and polydipsia strongly suggested the presence of diabetes insipidus (DI). Blood tests on the day of the accident showed that the sodium level in serum was 145 mEq/L; urea nitrogen, 11.5 mg/dI and glucose, 106 mg/dI. Plasma osmolarity (Posm) was 300 mOsm/L. Urine output was 450 ml/hr with a specific gravity value of 1.001. Urine glucose was not detected and urine osmolality (Uosm) was 120 mOsm/L. The patient was diagnosed with DI from the results of the simultaneous Uosm and Posm values³. Therefore, replacement therapy with aqueous vasopressin and water administration began on the admission day. The endocrinological examinations including stimulation tests on hospital days 2, 7, 14 and 30 are shown in Fig. 2. Fig. 2 shows a low level of basal values after stimulation on day 2 of all anterior pituitary hormones. All anterior hormones became normally functional within 1 month. The urinary volume, medication dose, fluid administration, and Posm and Uosm values are shown in Fig. 3.

On magnetic resonance imaging (MRI) obtained at 13 days after admission, the intact pituitary stalk was observed. However, a high signal intensity area was absent in the posterior lobe of the pituitary gland on T1-weighted images (Fig. 4 A, B). He was discharged 1 month after admission without any neurological deficit, but he remained on a regimen of intranasal DDAVP (1-desamino 8-D-arginine vasopressin; 10 µg) every 12 hours, and his urine output decreased to about 2,000 ml/day. Two months later, the DDAVP was stopped without DI recurrence.

Discussion

A fracture of the sella turcica is rare, Occurring in 1.4% of all severe head injuries⁴. Young et al⁵. reported that a fracture of the sella turcica alone, such as with our patient, is extremely rare, and anterior pituitary dysfunction after a head injury is uncommon^{2,5}. In this patient, the nail fractured the floor, of the sella turcica but did not reach the pituitary gland itself. This was confirmed by the skull X-ray and the negative result of the lumbar tap. Therefore, we believe that the anterior hypopituitarism shown in this patient was caused by a secondary injury, the fracture of the sella turcica, not the direct anterior pitui-

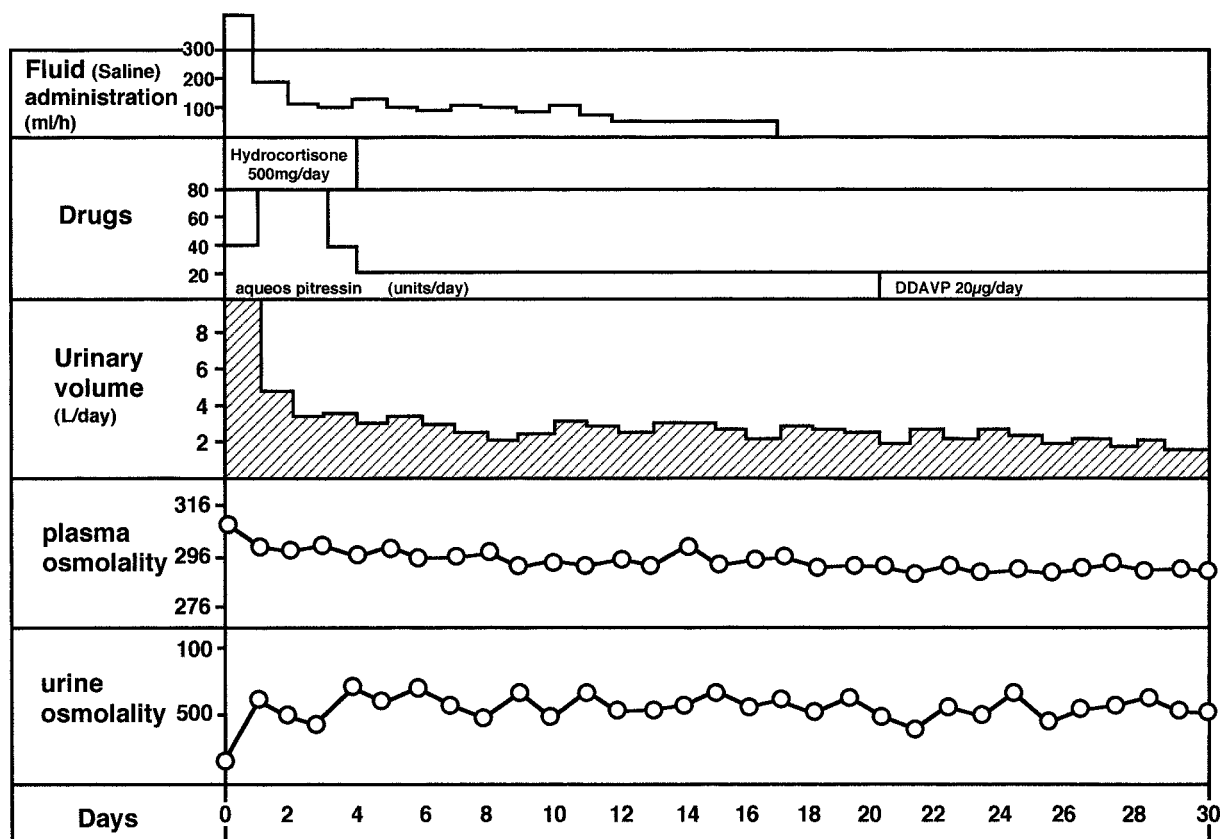


Fig. 3 The clinical course after admission

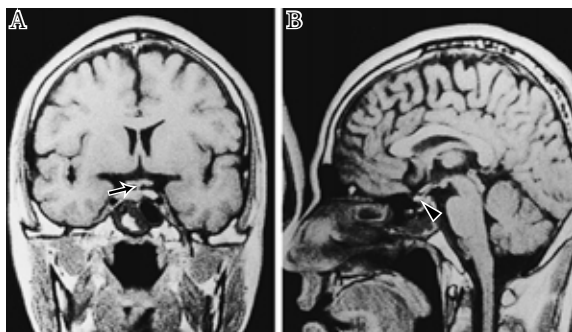


Fig. 4 (A) The magnetic resonance image showed the pituitary stalk was intact (arrow). (B) However, it did not show the high signal intensity area in the posterior lobe of the pituitary gland on T1-weighted images (arrow head).

tary injury by the nail.

Usually DI is a rare complication observed after severe craniofacial injuries due to an abrupt decrease in plasma anti-diuretic hormone (ADH) secretion⁶⁻⁹. It occurs in about 0.17~0.82% of all patients with head injuries and appears within 5 to 10 days after the trauma, although more acute and delayed onsets have been reported^{6,8}. Hyperacute DI within a few hours of

the trauma may be linked to the direct injury of the posterior lobe.

Although CT has been a routine method for evaluating neurohypophysis and non-traumatic DI^{3,10-12}, it has not been very useful for evaluating traumatic lesions of the hypothalamus and the pituitary stalk¹³. However, in some cases, it can show a transection of the stalk in axial CT scans and reconstructed coronal and sagittal images¹⁴. MRI has been proven to be particularly useful in demonstrating lesions around the sella turcica, but there have been only a few reported cases concerning hypopituitarism detected by MRI following a head injury^{13,15}. In these reports, the transection of the pituitary stalk was the only morphological change attributed to the pituitary dysfunction. Fujisawa et al¹⁵ reported a disappearance of the normal hyperintense signal of the posterior lobe in both idiopathic and tumoral DIs. As shown in our patient, this was also observed in posttraumatic DI.

The pathophysiology of the hypothalamo-pituitary injury is controversial, Porter and Miller¹⁴ emphasized a traumatic elongation of the stalk due to shearing stress, while Crompton¹⁵ emphasized that ischemic

and hemorrhagic lesions in the hypothalamus, infundibulum and pituitary stalk may be the cause of the underlying pathologies. These lesions may cause denervation of the posterior lobe with wallerian degeneration progressing upward, or may directly affect the paraventricular and supraoptic nuclei of the hypothalamus, which produces ADH^{6,9}. This probably accounts for the frequent appearance of DI within a period ranging from a few days to 1 month after trauma.

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