Unusual delayed onset of diabetes insipidus following closed head trauma

Case report

MOSHE HADANI, M.D., GIDEON FINDLER, M.D., IZHAK SHAKED, M.D., AND ABRAHAM SAHAR, M.D.

Department of Neurosurgery, The Chaim Sheba Medical Center, and The Sackler School of Medicine, Tel Hashomer, Israel

A case of delayed onset of diabetes insipidus (DI), which developed 27 days after a closed head injury, is reported. The patient sustained only a minor neurological deficit and, except for antidiuretic hormone (ADH) insufficiency, hypothalamic function was intact. This selective damage of posterior pituitary function was total and permanent. Ischemia due to vascular injury may be the most likely etiology. Once the diagnosis of delayed posttraumatic DI is confirmed, the treatment of choice is DDAVP (desmopressin acetate). In contradistinction to DI immediately following minor head injury, most patients with a delayed onset of DI after trauma have permanent ADH deficiency.

KEY WORDS • diabetes insipidus • head injury • antidiuretic hormone

The acute or delayed onset of diabetes insipidus (DI) following closed head trauma is well documented, but is surprisingly rare, except in cases of severe and usually fatal brain injuries. The low incidence can be explained by the fact that most accident victims die soon after the trauma if there is enough hypothalamic damage to lead to DI.

In 1946, Porter and Miller reported a case of DI following head injury, and since then several reports of immediate or delayed DI after head and facial trauma have appeared in the literature. The pathogenesis of the traumatic lesion and its location along the pathway of the production and storage of vasopressin remain obscure. We report a case of DI unique in that onset was 27 days after closed head trauma in an otherwise healthy adolescent.

Case Report

This 15-year-old boy was admitted to our service 1 hour after being involved in a traffic accident.

First Admission. On admission, the patient was unconscious. His Glasgow Coma Scale score was 9, and vital signs were normal. Both pupils were normal in size and shape, and reacted promptly to light. A left spastic hemiparesis was noted. The hematocrit, blood sugar, and electrolyte concentrations were within normal limits. Urine volume and urinalysis were normal. A computerized tomography (CT) scan of the brain revealed a small hemorrhagic contusion in the right temporoparietal region with no mass effect. Treatment with dexamethasone, 24 mg/day, was initiated. During the next 7 days, he gradually regained consciousness. The dexamethasone was tapered and subsequently discontinued.

During his entire hospital course, the patient's daily urine output was 1100 to 1300 cc, with normal urine and blood electrolytes. Plasma and urine osmolarity were within normal limits. Twelve days after the accident, a follow-up CT scan revealed complete resolution of the brain contusion. He was discharged on the 14th day, fully alert with a residual left hemiparesis.

Second Admission. Thirteen days later (27 days after the trauma), the patient started to complain of thirst, polydipsia, and polyuria. On examination, he was fully alert and the neurological examination revealed only residual left hemiparesis. Brain CT scan...
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was normal. Urine output was 450 cc/hr, with specific gravity values of 1.0; urine Na⁺ concentration was 6 mEq/liter, urine glucose was 0, and urine osmolarity 120 mOsm/liter. Blood tests showed the following levels: Na⁺ 147 mEq/liter, K⁺ 4.2 mEq/liter, Ca²⁺ 2.45 mmol/liter, urea 4 mmol/liter, and glucose 5.4 mmol/liter. Plasma osmolarity was 303 mOsm/liter.

Water deprivation test revealed total antidiuretic hormone (ADH) deficiency. Otherwise, hypothalamic pituitary function tests were within normal limits: thyroxine 7.6 mEg/dl, thyroid-stimulating hormone 3 IU/liter, serum cortisol 17.4 μg/dl (morning) and 9 μg/dl (evening), adrenocorticotropic hormone 13.8 pmol/liter, growth hormone 6 μIU/liter, luteinizing hormone 5 IU/liter, follicle-stimulating hormone 3 IU/liter, and prolactin 18 pmol/liter. Visual fields were normal.

He was initially treated by subcutaneous Pitressin in water 5 IU every 6 hours, following which urine output decreased to 90 cc/hr. Blood and urine Na⁺ concentrations and osmolarity returned to normal values. After 3 days, treatment with DDAVP (Minerin: desmopressin acetate) was initiated and the urine output decreased to 2100 cc/day, urine osmolarity was 320 mOsm/liter and blood Na⁺ concentration 140 mEq/liter. With this treatment he was sent home. Six months later the DDAVP was withheld, but the diabetes insipidus promptly recurred.

Discussion

This 27-day interval between trauma and the onset of diabetes insipidus (DI) is the longest we have found in the literature. In previous reports of delayed traumatic DI, the interval ranged from 1 to 14 days.4.6.7.14 The delay suggests an intact posterior pituitary or lower stalk, along with a lesion above the median eminence, caused by either direct cellular damage or retrograde degeneration of the cell bodies in the supraoptic and paraventricular nuclei.2 In the symptom-free interval, water balance may be maintained by stored ADH, and DI appears following the exhaustion of vasopressin in the denervated posterior lobe. Delayed onset is a bad prognostic sign, since a hypothalamic lesion usually leads to permanent DI.

Notman, et al.7 showed that the onset of symptoms was delayed for 2 to 14 days in all of their 10 cases with permanent DI following closed head trauma. In contrast, acute onset of DI following minor to moderate closed head trauma usually indicates a lesion in the posterior pituitary below the median eminence, which causes only transient ADH insufficiency. The six cases reported by Savino, et al.,11 to have acute DI following traumatic chiasmal syndrome were probably of this category.

The exact mechanism involved in the production of DI following blunt head trauma is not yet clear. However, autopsies of head-trauma patients have shown that the major damage in the hypothalamo-hypophyseal complex was located in the lower part of the hypothalamus, in the infundibulum, and in the upper half of the stalk, while the pituitary gland itself did not usually suffer injury.2 The principal mechanism involved seems to be ischemia from vascular injury. Several etiologies of the hypothalamic lesion were suggested to cause DI following head trauma, among which were microhemorrhage, contusion necrosis, vascular injury,2-6 localized edema, and venous engorgement.10 Crompton2 showed that the majority of these vascular lesions in the hypothalamus and stalk following blunt head trauma resulted from shearing of small perforating vessels at the time of impact. It was also demonstrated that there were more temporoparietal and fewer fronto-occipital blows to the head in patients with hypothalamic lesions. Presumably, the forces most likely to shear hypothalamic vessels are those that are directed laterally.

The sparing of the anterior pituitary function in most previously reported cases, as well as in ours, is of interest because the presumably damaged supraoptic and paraventricular nuclei lie in close proximity to the other hypothalamic nuclei. Crompton2 explained the normal anterior pituitary function by the fact that the hypothalamic and stalk lesions are high enough to denervate only the posterior pituitary, with degeneration of the cell bodies of the supraoptic and paraventricular nuclei, while the portal system which supplies the anterior pituitary is not involved. Triep10 assumed that the supraoptic and paraventricular nuclei were the most vulnerable regions in the hypothalamus, hence the only region to be damaged by certain trauma. Another rare example of highly selective hypothalamic damage after head trauma involving only thermoregulation and thyroid function was recently reported, but the mechanism in that case remained obscure.9

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