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Case Report

A rare cause of refractory hyponatremia after traumatic brain injury: Acute post-traumatic hypopituitarism due to pituitary stalk transection

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Abstract

We report a 1.5-year-old boy with refractory hyponatremia related to pituitary stalk transection, which is a rare cause of hyponatremia after traumatic brain injury. The patient was referred to our hospital with a hyponatremic convulsion 6 days after head trauma. The patient's laboratory findings were compatible with syndrome of inappropriate antidiuretic hormone secretion (SIADH). The hyponatremic convulsion was treated with a hypertonic saline infusion and the SIADH was treated with fluid restriction, but serum levels of sodium did not increase. Acute post-traumatic hypopituitarism was diagnosed based on basal pituitary function tests and imaging. Hypophysis magnetic resonance imaging showed pituitary stalk transection. He was diagnosed with post-traumatic hypopituitarism due to pituitary stalk transection and given L-thyroxine and hydrocortisone. After the treatment, sodium and thyroid hormone levels returned to normal. Acute post-traumatic hypopituitarism is a potentially important cause of hyponatremia after traumatic brain injury, and can be misdiagnosed as SIADH.

Keywords: hyponatremia; pediatric patients; pituitary stalk transection; post-traumatic hypopituitarism; traumatic brain injury

1. Introduction

The most common causes of hyponatremia after traumatic brain injury (TBI) are syndrome of inappropriate antidiuretic hormone secretion (SIADH) and cerebral salt-wasting syndrome, while post-traumatic hypopituitarism is a rare cause of severe hyponatremia.[1] Post-traumatic hypopituitarism is the failure of the hypothalamic–pituitary axis secondary to TBI and is observed in 15–25% of the patients with a history of TBI.[2]

Neuroimaging studies in patients with post-traumatic hypopituitarism show mostly hemorrhage of the hypothalamus and posterior pituitary lobe, and infarction of the pituitary lobe. Pituitary stalk transection is a rare cause of post-traumatic hypopituitarism.[3]

We report a 1.5-year-old boy with severe, persistent hyponatremia caused by pituitary stalk transection after head trauma.

2. Case Report

A 1.5-year-old boy was referred to our hospital with hyponatremia and a hyponatremic convulsion 6 days after trauma. He had fallen four stories. The physical examination, cranial tomography, and magnetic resonance imaging

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(MRI) were normal. The patient's laboratory values were as follows: serum glucose, 74 mg/dL; sodium, 120 mg/dL; serum potassium, 4.4 mg/dL; serum uric acid, 1.2 mg/dL; urine sodium, 190 mg/dL; urine output, 1.2 mL/kg/h; and urine specific gravity, 1.010. These findings suggested SIADH. The hyponatremic convulsion was treated with hypertonic saline and the SIADH was treated with fluid restriction, but his serum levels of sodium did not increase. Basal pituitary function tests 7 days after the trauma were as follows: serum cortisol, 1.09 (normal 6.2–19.4) μg/dL; adrenocorticotropic hormone (ACTH), 14.2 pg/mL (normal < 46 pg/mL); sT_4, 0.33 pg/mL (normal 0.6–2 pg/mL); cortisol, 1.09 μg/dL (normal 6.2–19.4 μg/dL); sT_3, 1.79 ng/dL (normal 2.3–4.8 ng/dL); TSH, 1.03 mIU/L (normal 0.4–5.6 mIU/L); and prolactin, 35.36 ng/mL (normal 2.64–13.13 ng/mL). Hypophysis MRI showed pituitary stalk transection. There were fine septations in the suprasellar cistern in a three-dimensional sagittal FIESTA image (Figure 1). A coronal contrast-enhanced T1-weighted image showed blunt ending at the infundibulum (Figure 2). The high signal intensity normally seen in the posterior lobe was absent, whereas the adenohypophysis was normal (Figure 3). Based on these characteristic endocrinological and MRI findings, the patient was diagnosed with multiple hypophyseal hormone deficiency secondary to pituitary stalk transection. The hyponatremia resulted from adrenal insufficiency secondary to hypothalamo–hypophyseal ACTH deficiency. Treatment with hydrocortisone and levothyroxine was started and the sodium level subsequently returned to normal.

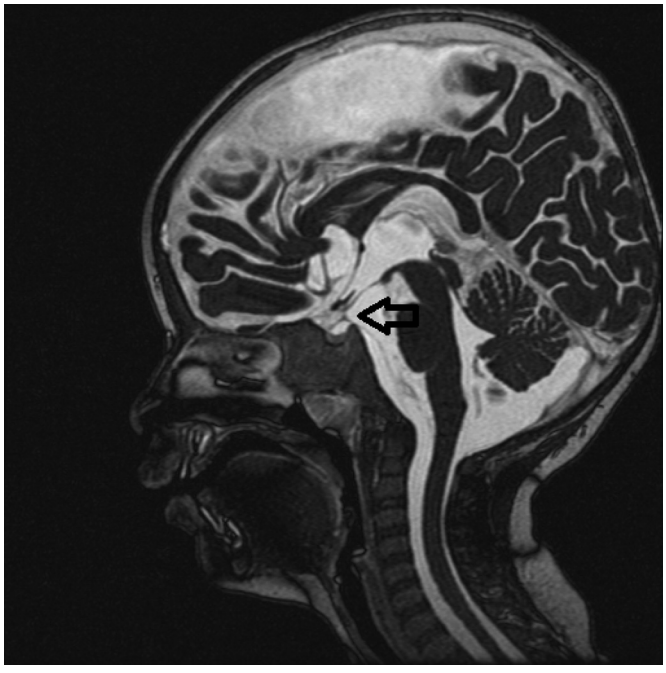

Figure 1. Three-dimensional sagittal FIESTA image. Fine septations in the suprasellar cistern.

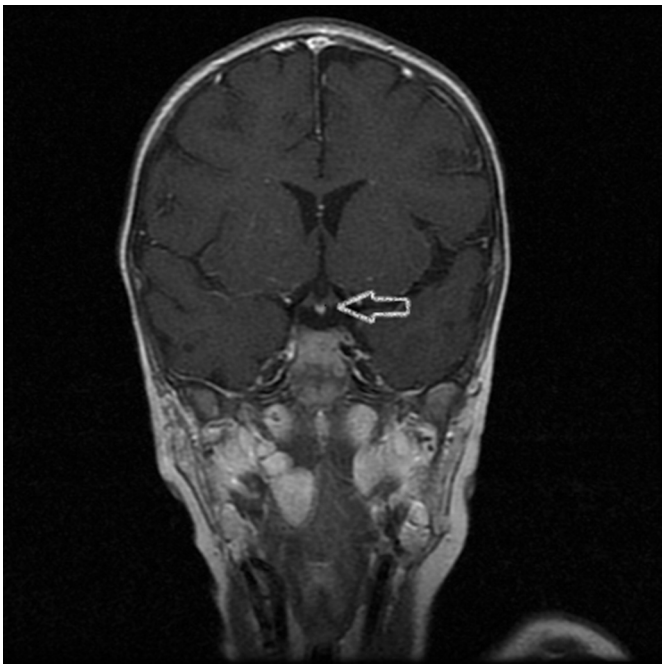

Figure 2. Coronal contrast enhanced T1-weighted image. Blunt ending infundibulum is seen.

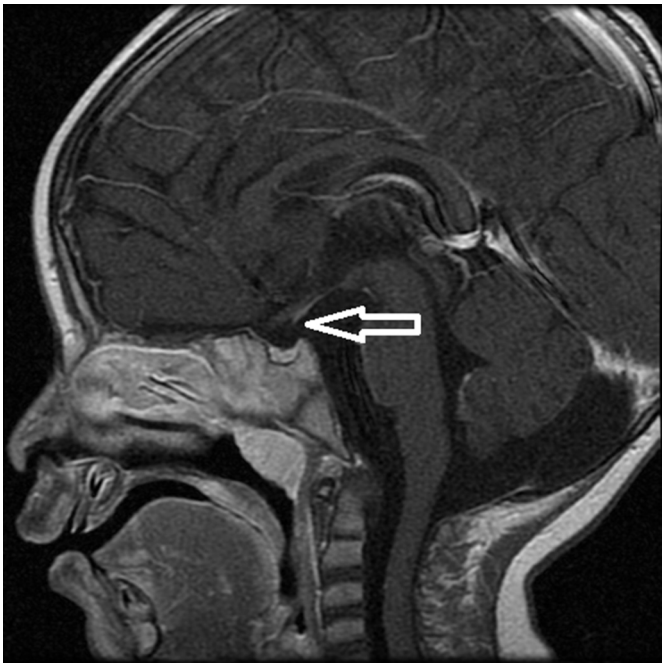

Figure 3. Contrast enhanced T1-weighted sagittal image.

3. Discussion

Hyponatremia is the most common electrolyte abnormality after TBI, occurring in 13% of cases and SIADH is the most common cause of hyponatremia in TBI. Other causes are cerebral salt-wasting syndrome, hypotonic fluid administration, and medications.[1]

Impairment of the pituitary−adrenal axis is linked to hyponatremia. Post-traumatic hypopituitarism is the failure of the hypothalamic−pituitary axis secondary to TBI.[4,5] Pituitary stalk transection syndrome is defined as hypopituitarism with insufficiency of several pituitary hormones caused by transection of the pituitary stalk, accompanied by atrophy of the anterior lobe and formation of a pseudoposterior lobe of the pituitary gland. It occurs mainly during abnormal delivery and rarely in brain trauma.[6] Neuroimaging studies in patients with post-traumatic hypopituitarism rarely show pituitary stalk transection (3.9%).[3]

In our patient, post-traumatic hypopituitarism developed 6 days after the TBI. There were multiple hypophyseal hormone deficiencies, which included central hypothyroidism and hypothalamohypophyseal ACTH deficiency. Agha et al[7] reported that 16% of acute head injury patients show biochemical evidence of ACTH deficiency. Acute hypopituitarism with ACTH deficiency is a potentially important cause of hyponatremia in patients with acute TBI misdiagnosed as SIADH.[7,8]

To illustrate this potential pitfall in diagnosing post-traumatic hyponatremia, we report our case with acute TBI complicated by hyponatremia with all of the biochemical features of SIADH. Our patient had significant head trauma. He had normal blood pressure and plasma levels of glucose, but the plasma levels of sodium did not return to normal with fluid restriction. The patient had low basal serum levels of cortisol and plasma concentrations of ACTH. These findings are very suggestive of acute secondary adrenal (ACTH) deficiency. The patient showed a remarkable, rapid response to the administration of intravenous hydrocortisone, with normalization of the sodium levels. There was evidence of additional pituitary hormone abnormalities. The serum levels of thyroid stimulating hormone and T_4 were low and that of prolactin was high. An elevated prolactin level is associated with acute hypophyseal damage.[5] In our case, diabetes insipidus had not developed in the presence of a pseudoposterior lobe because vasopressin secretion from the transected part of the stalk was preserved.

This case report illustrates that hyponatremia following TBI may be a marker of acute hypopituitarism with secondary adrenal failure. Therefore, pituitary hormones should be assessed in head injury patients with an SIADH-like clinical picture.

4. Conclusion

Patients with TBI with unexplained electrolyte disturbances such as hyponatremia should be screened for hypopituitarism.

Conflicts of interest

All authors declare no conflicts of interest.

Acknowledgments

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