Two cases of traumatic isolated ACTH deficiency

Abstract

Case 1: A 65-year-old man was accidentally injured by wooden hammer on his top of head on 34 years before. He was suffered from vomiting, diarrhea and hypotension, and the laboratory examination revealed increased CRP level, hyponatremia and decreased plasma cortisol and ACTH levels, suggesting isolated ACTH deficiency and Crohn disease diagnosed by colonoscopic biopsy, and finally transferred to University Hospital. LH-RH, TRH, CRH and GHRP stimulation tests showed normal response of plasma pituitary hormones except for no response of plasma ACTH and cortisol levels by CRH stimulation. ACTH stimulation test showed no response of plasma cortisol levels although hydrocortisone replacement therapy had already been started. MRI imaging showed bottom of anterior lobe was crushed and pituitary gland was atrophied, which suggested brain might be injured by any strong trauma.

Case 2: An 83-years old man was injured on brain contusion by staff's violence in nursing home, and introduce to our hospital to remove brain hematoma on 6 months before. He presented transient loss of consciousness because of hypoglycemia. Laboratory examinations revealed hyponatremia, and low levels of plasma ACTH and cortisol. Endocrinological examination showed normal LH-RH and TRH stimulations tests, basal GH and IGF-1 levels, and no response of plasma ACTH and cortisol levels by CRH stimulation, showing traumatic isolated ACTH deficiency. MRI imaging showed atrophic pituitary gland. These results suggest that traumatic isolated ACTH deficiency may be able to appear for short and long period after brain injury.

Introduction

Isolated ACTH deficiency is a rare disease characterized by secondary adrenal insufficiency with low or absent cortisol production and normal secretion of pituitary hormones other than ACTH [1]. Isolated ACTH deficiency has been caused by traumatic injury [2], lymphocytic hypophysitis due to autoimmune etiology [3,4], genetic origin in neonatal or childhood [1], and unknown origin. Previous reports have demonstrated that traumatic brain injury—mediated hypopituitarism could be more frequently occurred [5-7]. High prevalence of neuroendocrine dysfunction in patients with traumatic brain injury has been reported [8].

In this study we have shown two cases of traumatic isolated ACTH deficiency.

Case Presentation

Case 1

A 65-year-old man was accidentally injured by wooden hammer on his top of head on 34 years before. He was suffered from vomiting, diarrhea and hypotension, and the laboratory examination revealed increased CRP level, hyponatremia and decreased plasma cortisol and ACTH levels, suggesting isolated ACTH deficiency and Crohn disease diagnosed by colonoscopic biopsy, and finally transferred to University Hospital. LH-RH, TRH, CRH and GHRP stimulation tests showed normal response of plasma pituitary hormones except for no response of plasma ACTH and cortisol levels by CRH stimulation. ACTH stimulation test showed no response of plasma cortisol levels although hydrocortisone replacement therapy had already been started. MRI imaging showed bottom of anterior lobe was crushed and pituitary gland was atrophied, which suggested brain might be injured by any strong trauma.
Case 2

An 83-years old man was injured on brain contusion by staff’s violence in nursing home, and introduce to Gifu Municipal hospital to remove brain hematoma on 6 months before. He presented transient loss of consciousness because of hypoglycemia (37 mg/dl), and transferred to our hospital.

Laboratory examinations revealed hyponatremia (126 mEq/l), normal HbAtc (5.1 %) level (Figures 4-a, 4-b), and low levels of plasma ACTH (6.8 pg/ml), cortisol (15.2 μg/dl) and suppression of PRA (less than 0.1 ng/ml/hr) and aldosterone (less than 10.0 pg/ml) levels during saline infusion (Figure 4-a, Figure 4-b).

Endocrinological examination showed normal LH–RH and TRH stimulation test, normal plasma basal GH (3.73 ng/ml) and IGF-1 (37 ng/ml) and suppression of PRA (less than 0.1 ng/ml/hr) and aldosterone (less than 10.0 pg/ml) levels during saline infusion (Figure 4-c, Figure 5-a).

Discussion

It has been reported that the percentage of probability in the appearance of endocrinological abnormality was 15–68%, especially in hypotuitarism of anterior lobe was 27.5% and insufficiency of growth hormone secretion occurred in highest rate, and in the next rate hypoadrenocortism and hypoglycemia (37 mg/dl), and transferred to our hospital.

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Discussion

It has been reported that the percentage of probability in the appearance of endocrinological abnormality was 15–68%, especially in hypotuitarism of anterior lobe was 27.5% and that hypofunction in hypophysico–pituitary axis occurs within half year after brain trauma. Gonadal hypofunction and insufficiency of growth hormone secretion occurred in highest rate, and in the next rate hypoadrenocorticot and
hypothyroidism occurred. Schneider et al. [2] also reported that grade of brain damage evaluated in Glasgow Coma Scale (GCS) was correlated with occurrence rate in hypothalamo-pituitary hypofunction as follows: Severe damage (GCS 9-12 points), moderate damage (9-12 points) and slight damage (13-15 points) was 35.3%, 10.9% and 16.8%, respectively. However, even if in the case of slight brain damage, occurrences of endocrinological abnormality should be taken care. They conclude that hypopituitarism is a common complication of both traumatic brain injury and aneurysmal subarachnoid hemorrhage. This systematic review showd ACTH deficiency was occurred in 0-19.2% after traumatic brain injury. Tanriverdi et al. [9], also reported that some 5.8% of the traumatic brain injury patients had TSH deficiency, 41.6% had gonadotropin deficiency, 9.8% had ACTH deficiency, and 20.4% had GH deficiency, and that pituitary function may improve or worsen in a considerable number of patients over 12 months. A patient presented in case 1 occurred clinical symptoms of hypopituitarism for 34 years after severe traumatic injury. Recent report indicated 3 cases of isolated ACTH or TSH deficiency following mild traumatic brain injury with long-term follow (10 days-20 years) [10], which was similar to our case 1. Sixty-five years-old man occurred both isolated ACTH deficiency and Chrohn's disease at the same time in case 1. Kalambokis et al. [11], had been reported that isolated ACTH deficiency associated with Chrohn's disease without traumatic brain injury which might be associated with immune reactions. Therefore, etiology of our case 1 might be a little relevance for complication of Chrohn's disease. Recently, old men and women received violent brain traumas in old people's home have been happened. Clinical symptoms and results of laboratory examinations such as nausea vomiting, hypotension and increased CRP levels should be paid attention.

Conclusion
Two cases of these disorders were treated with 15-20 mg of hydrocortisone and continued to live in good health. These results suggest that traumatic isolated ACTH deficiency may be able to appear for short and long period after brain injury.

Disclosure
None of the authors have any potential conflict of interest associated with this research.

The ethical committee in the Gifu Municipal Hospital have been approved in this study.

References
