Anosmia and isolated ACTH deficiency following a road traffic accident

Case report

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A 42-year-old man suffered a head injury in a road traffic accident and subsequently developed anosmia and isolated adrenocorticotropic hormone (ACTH) deficiency. There was no other evidence of pituitary dysfunction. No previous case of isolated ACTH deficiency following head injury has been reported.

KEY WORDS □ anosmia □ adrenocorticotropic hormone □ head injury

Isolated adrenocorticotropic hormone (ACTH) deficiency is a rare disorder which has not previously been described as a result of a head injury. We report the case of a 42-year-old man who suffered from anosmia and ACTH deficiency following head injury incurred in a road traffic accident.

Case Report

This 42-year-old man was wearing a seat belt when he was involved in a head-on collision with another car. His wife, who was with him, thought that he did not lose consciousness. He did not require admission to a hospital at that time. Following the accident he experienced loss of sense of smell, abnormality in taste sense, general weakness, impairment of memory, and weight loss. He was unable to return to his responsible job, and his wife noticed a change in his personality. He was investigated at a local hospital where low plasma cortisol levels were noted and he was referred to the Royal Free Hospital for further investigation.

Examination. On admission, neurological examination revealed no focal abnormality apart from anosmia. No pigmented areas were found and he had no postural drop in blood pressure. Computerized tomography showed no focal abnormality, but an electroencephalogram revealed a minimal amount of anterior-quadrant intermediate low-frequency activity. The plasma potassium level was 5.2 mmol/liter (normal range < 5 mmol/liter) and the plasma chloride content was 106 mmol/liter (normal range < 105 mmol/liter); however, the bicarbonate and glucose values were within the normal range. Plasma cortisol levels were undetectable and administration of tetracosactin (cosyntropin, 250 µg) intravenously produced only a minimal rise to 69 nmol/liter. At this time, the patient was potent and his serum thyroxine, thyroid-stimulating hormone (TSH), prolactin, follicle-stimulating hormone, and testosterone levels were within the normal range.

Course. Steroid replacement therapy resulted in an increase in weight and an improvement in general well-being, although there was no change in the anosmia. Plasma electrolyte levels were normal on administration of hydrocortisone (30 mg/day), and fluorocortisone was not required. Subsequently, a prolonged course of tetracosactin was given. Urinary free cortisol, plasma cortisol, and ACTH levels were below the detectable range 5 days after withdrawal of cortisol replacement therapy. Urinary free cortisol levels rose to 1573 mmol/24 hrs on the 3rd day of a 2-mg/day course of depot tetracosactin. Infusion of 250 µg tetracosactin intravenously on the 4th day caused a rise in the patient's cortisol level from 682 to 814 nmol/liter. A corticotropin-releasing hormone (CRH) stimulation test was performed using 100 µg rat-human CRH. Although the patient had not received cortisol for 2 days prior to the procedure, the initial ACTH values were below the detectable limit for the assay, and did not rise to a detectable level even at 2 hours after CRH administration.

The patient received hydrocortisone treatment and improved sufficiently to return to full-time employment. Withdrawal of hydrocortisone at the time of the prolonged tetracosactin administration produced a re-
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Discussion

Stacpoole, et al., 7 found 43 previously reported cases of isolated ACTH deficiency, although some of these cases were evaluated prior to the availability of an ACTH assay. None of their cases was associated with head injury. The patient described here had a similar clinical presentation to the reported cases in that he did not exhibit frank addisonian symptoms at any stage. The abrupt onset of his symptoms and anosmia following the car accident suggested that the defect occurred at that time. The maintenance of his other pituitary functions indicates that this may be a hypothalamic defect as observed in a patient described by Lytras, et al. 3 However, the lack of response to CRH is similar to that reported by Tsukada, et al., 9 in two patients with isolated ACTH deficiency who did not respond to CRH stimulation, even though four other patients with hypothalamic hypopituitarism had exaggerated ACTH responses to CRH. 8 This evidence suggests that the defect in our patient may have been located in the pituitary gland even though the presence of anosmia indicates an injury of sufficient severity to cause a hypothalamic lesion.

Hypopituitarism is a rare but well-recognized complication of head trauma. In 1986, Edwards and Clarke 2 described six patients and found 46 other cases reported in the literature. These authors noted that several of the patients described had suffered no loss of consciousness or had been unconscious for a very short period; however, the majority of cases of posttraumatic hypopituitarism do follow unconsciousness which usually lasts for several days. None of the reported cases have exhibited pure ACTH deficiency, although in a number of instances the defect had been in part a hypothalamic rather than a primary pituitary defect. 3,4,6,10-13 Woolf and Schlach 13 reported a case in which cortisol levels were low and did not respond to insulin-induced hypoglycemia but did respond to ACTH therapy, suggesting a central ACTH deficiency. The features supporting a hypothalamic defect in that patient were high basal prolactin levels and a rise in TSH following stimulation with thyroid-releasing hormone. Jambert, et al., 8 described a similar case in which the cortisol value did not rise after insulin hypoglycemia but did rise after lysine vasopressin infusion, suggesting a defect in hypophysial CRH release. Crompton 1 reported that hypothalamic damage was found in 43% of postmortem examinations performed on patients who had died after closed head injury. There is thus evidence that there can be both anatomical and physiological hypothalamic damage following head injury.

References


Manuscript received January 26, 1989. Accepted in final form March 21, 1990. Address reprint requests to: J. E. Scoble, M.D., M.R.C.P., Renal Research Unit, Royal Free Hospital, London NW3 2QG, England.