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Acquired growth hormone deficiency due to pituitary stalk transection after head trauma in childhood

C. Yamanaka[1], T. Momoi[1,3], I. Fujisawa[2,4], K. Kikuchi[5], M. Kaji[1], H. Sasaki[1], T. Yorifuji[1], and H. Mikawa[1]

Departments of [1]Pediatrics, [2]Radiology and Nuclear Medicine, Kyoto University Faculty of Medicine, Shogoin Kawahara-cho, Sakyo-ku, Kyoto, 606, Japan
[3]Department of Pediatrics, Wakayama Red Cross Hospital, Wakayama, Japan
[4]Department of Radiology, Kishiwada City Hospital, Kishiwada, Japan
[5]Department of Pediatrics, Shimane Medical University, Izumo, Japan

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Abstract. Two patients are reported with growth hormone deficiency due to head trauma in childhood. Although their injuries were outwardly only slight and there was no loss of consciousness and no subsequent neurological deficits, they exhibited gradual growth retardation from the time of the trauma. Provocative endocrinological tests showed growth hormone deficiency and MRI showed transection of the pituitary stalk. These findings suggest that ordinary head trauma, as well as perinatal insult and congenital abnormalities, could be a cause of growth hormone deficiency.

Key words: Magnetic resonance imaging – Growth hormone deficiency – Head trauma

Introduction

Transection of the pituitary stalk caused by perinatal injury or congenital hypoplasia of the pituitary structure has been demonstrated in many patients with idiopathic growth hormone (GH) deficiency [5, 9]. Likewise many patients with hypopituitarism due to head injury have also been reported [3, 4]. In most of the latter patients however, the trauma was severe and there was loss of consciousness or the subsequent occurrence of neurological deficits [3, 4].

In this paper we report on two boys who developed GH deficiency after slight head injury; there was no loss of consciousness and there were no subsequent neurological deficits in either patient. MRI in both patients showed a transected pituitary stalk and a newly formed ectopic posterior lobe.

Correspondence to: C. Yamanaka

Abbreviation: GH = growth hormone

Case reports

Case 1

This male patient, who has been briefly mentioned previously [9], was born at term by cephalic presentation with no perinatal complications. He had shown normal physical and mental development until the age of 3 years and 3 months when he fell down some stairs, knocking his head on the ground. He cried immediately. He did not show any neurological deficits after this injury. We were unable to obtain a clear picture of his urinary pattern immediately after the injury, despite our careful questioning of his parents. Since the injury, his growth rate has gradually slowed down to less than 2 SD (Fig. 1). At the time he was first brought to our hospital at the age of 9 years and 10 months due to his short stature his height was 117.5 cm (−2.5 SD) and his bone age [6] was 7 years. Basal levels of serum thyroxine, thyroid stimulating hormone and prolactin were normal. Plasma somatomedin-C was reduced (0.26 units/ml). Provocative endocrinological tests were performed when the child was 10 years old. The maximal GH responses to insulin, arginine, and L-dopa were less than 1.5, less than 1.5 and 2.0 μg/l, respectively. The peak GH level after growth hormone releasing hormone injection was 13.8 μg/l. The results of thyrotrophin and gonadotrophin releasing hormone tests were normal. T1-weighted 1.5 tesla MR images (General Electric, Milwaukee, USA), obtained at the age of 10 years, demonstrated transection of the pituitary stalk, loss of the high intensity posterior pituitary lobe signal, and formation of an ectopic posterior lobe at the proximal stump of the transected pituitary stalk (Fig. 2).

Case 2

This male patient was delivered at term by cephalic presentation with no perinatal complications. His physical and mental development had been normal for 11 years. At the age of 11 years he fell down some stairs and knocked his head against a wall. He did not lose consciousness and he showed no subsequent neurological deficits. In this case also, we were unable to get a clear picture of the urinary pattern immediately after the injury, despite our careful questioning of his parents. After the injury his rate of growth gradually slowed down to less than 2 SD. When he was brought to our hospital due to his short stature at the age of 13 years and 11 months, his height was 141.8 cm (−2.3 SD) and his bone age [6] was 10 years. Basal levels of serum thyroxine, thyroid stimulating hormone and prolactin were normal. Plasma somatomedin-C level

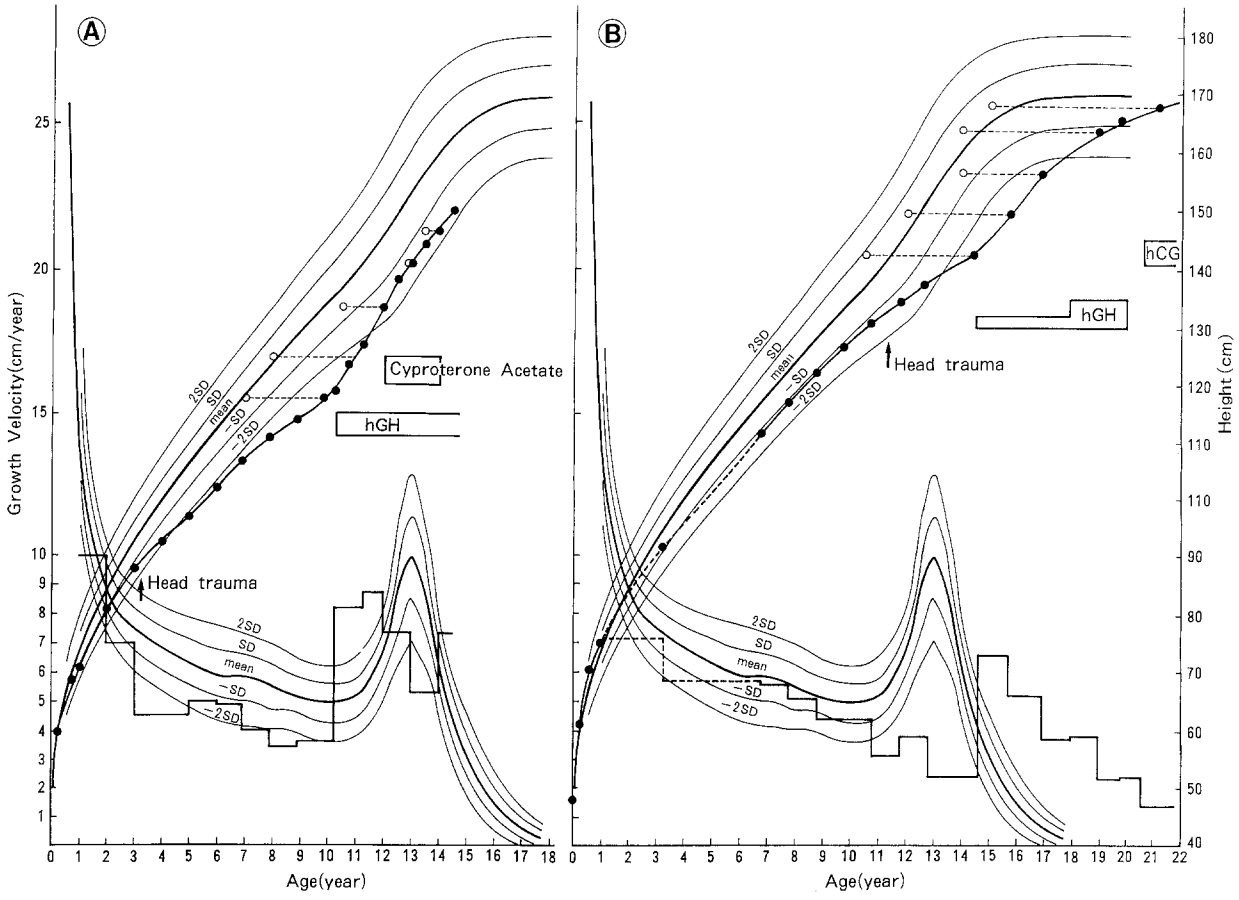

Fig. 1. Height (●) and bone age (○) of two patients with head trauma. Case 1 (**panel A**) had a slight head injury at the age of 3 years and 3 months; hGH therapy was initiated at the age of 10 years and 3 months. He was given cyproterone acetate as gonadal suppression therapy [7]. Case 2 (**panel B**) had a slight head injury at the age of 11 years, and he received hGH from the age of 14 years and 6 months to the age of 20 years. He is now receiving gonadotrophin therapy for hypogonadotropic hypogonadism, with normozoospermia having been achieved. Note that the growth rate had decreased after the head trauma in both patients

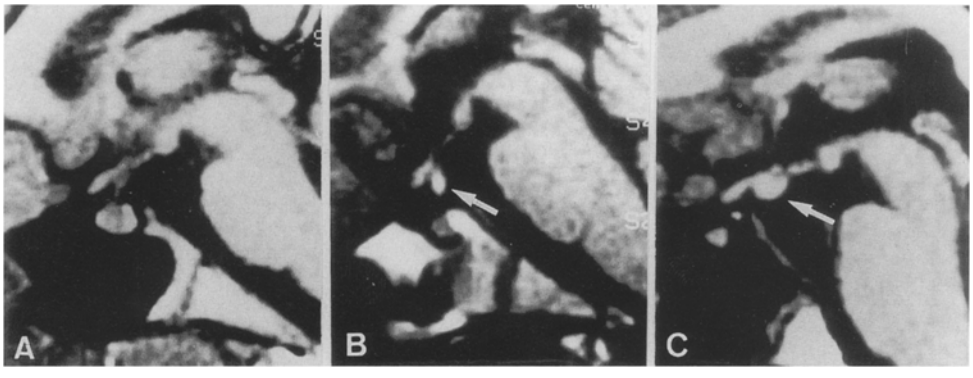

Fig. 2. Sagittal section of hypothalamo-pituitary region in 1.5 tesla MR images (T1-weighted; repetition time 400 ms, echo time 25 ms). In the normal child (**A**), the pituitary stalk is clearly visible and the posterior pituitary lobe is demonstrated by its characteristic high signal intensity in the sella turcica. In cases 1 (**B**) and 2 (**C**), transection of the pituitary stalk and disappearance of the posterior pituitary lobe can be observed. The newly formed ectopic posterior lobe is identified as a bright spot at the proximal stump of the transected stalk in each patient (*arrow*)

was not determined. Provocative endocrinological tests were first performed when he was 14 years of age. The maximal GH responses to insulin, L-dopa, and propranol-glucagon were 0.9, 1.0 and 2.3 μg/l, respectively. The results of the thyrotrophin releasing hormone and the metyrapone tests were normal. The peak follicle stimulating hormone level after gonadotrophin releasing hormone injection was 10.0 IU/l; however, at the age of 20 years, he was diagnosed with gonadotrophin deficiency (peak leuteinising follicle stimulating hormone levels after gonadotrophin releasing hormone injection at that time were 2.1 and 3.5 IU/l, respectively). He has never exhibited anosmia. T1-weighted 1.5 tesla MR images produced when he was 18 years old demonstrated transection of

the pituitary stalk, loss of high intensity posterior pituitary lobe signal, and formation of an ectopic posterior lobe at the proximal stump of the transected pituitary stalk (Fig. 2).

Discussion

Here we have reported two boys with complete GH deficieny that occurred after head trauma. It is noteworthy that their head injuries were outwardly minor traumas and that there was no loss of consciousness and no subsequent neurological deficits; the head trauma was an ordinary one for their age. Although such results are relatively rare after minor head trauma, these findings indicate that the pituitary stalk and surrounding vascular system are fragile with regard to the effects of external forces.

We have recently reported some patients with GH deficiency who had a history of perinatal insult and who showed transection of the pituitary stalk on T1-weighted MR images [5, 9]. On the other hand, there have been reports of several GH deficiency patients who did not experience perinatal insult but who showed the same findings on MR as our patients [1, 2, 10, 11, 13]. However, the authors of these reports do not seem to have paid much attention to any history of head trauma after birth. Besides events such as impaired blood supply or disturbance of the fusion of pituitary primordia during intra-uterine life, "minor" head trauma during childhood could also give rise to transection of the pituitary stalk. Indeed, the normal pretraumatic growth patterns of our two patients does not suggest that they might have already had GH deficiency when the head trauma occurred.

The rate of growth in these two patients was retarded within 2 years after the head injury, however, polyuria, which is often seen after surgical transection of the pituitary stalk, was not noted in either patient. Patient 2 showed gonadotrophin deficiency, not in the initial provocative tests, but in the later tests. We speculate that impaired blood supply to the hypophysis and pituitary stalk, as well as partial transection of pituitary stalk, had occurred at the time of the head injury. This damage gradually progressed until the transection of the pituitary stalk was revealed on the MR images several years later; the formation of the ectopic posterior lobe preserved water homeostasis [12, 13].

There have been several reports of patients with transected pituitary stalks who exhibited isolated GH deficiency [9, 10]. We speculate that collateral circulation and/or recanalization of the hypothalamo-hypophyseal portal system might play a role in the residual pituitary function. The normal basal prolactin level of our two patients suggests a partial connection between the hypothalamus and the pituitary gland.

Children with deprivation dwarfism usually show transient GH deficiency, although some of them do have permanent GH deficiency [8]. In these patients, it is possible that there might be abnormal MR findings such as those shown in our two patients, since there is a close relationship between psychosocial deprivation and child abuse.

In conclusion, minor head trauma as well as perinatal insult and congenital malformation, could be a cause of GH deficiency, although the former might be a less common cause than the latter two. Careful attention should be paid when following up children who have a history of even slight head trauma and who show a retarded rate of growth.

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