Recovery of central fever after GH therapy in a patient with GH deficiency secondary to posttraumatic brain injury

Hale Unver Tuhan[1], Ahmet Anik[1], Gonul Catli[1], Ayhan Abaci[1], Tulay Ozturk[2], Handan Guleryuz[2], Ece Bober[1]

[1] Department of Pediatric Endocrinology, [2]Department of Pediatric Radiology, Dokuz Eylül University, Faculty of Medicine, İzmir

Background

Hypopituitarism is a partial or complete insufficiency of pituitary hormone secretion that may be derived from pituitary or hypothalamic disease. Traumatic brain injury has been identified as a rare cause of hypopituitarism despite the increase in recent reports on this subject. It has recently been shown that patients with GH deficiency have a reduced sweating capacity which increases the risk of developing hyperthermia.

Aim

To the best of our knowledge, central fever that developed due to growth hormone deficiency and improved with growth hormone treatment has not been reported until now despite the association between hyperthermia and growth hormone deficiency is known. In this article, we report a dramatic improvement of a central fever with recombinant human growth hormone (rhGH) therapy in a patient with posttraumatic hypopituitarism.

Case Report

A seven-year-old boy, who was involved in a traffic accident, presented with coma. He was born by spontaneous vaginal delivery after an uneventful pregnancy with a birth weight of 2750g. His growth and development were compatible with his chronological age. On the admission to emergency room,

On physical examination;

He was unconscious (coma stage I, Glasgow scale), at times requiring intubation and mechanical ventilation.

Weight was 25 kg (0.21 SDS), Height was 118 cm (-1.20 SDS)

Pubic hair: Tanner stage 1, Bilateral testicular size: 3 ml.

Imaging studies;

Cranial computed tomography : Multiple skull fractures, sella fracture, hydrocephalus, pneumocephalus and subarachnoid hemorrhage (Figure 1).

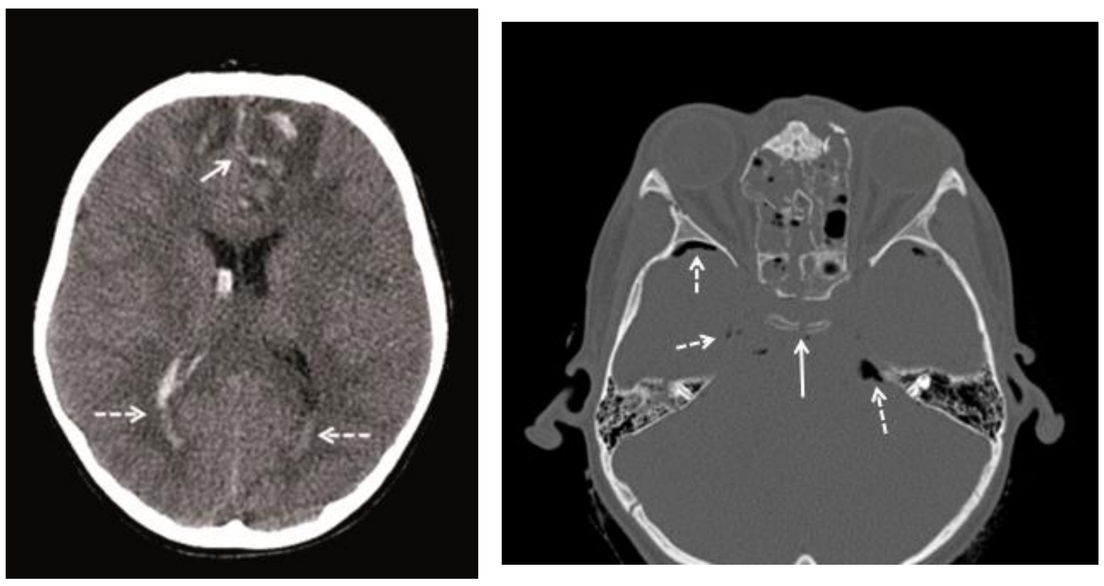

Figure 1. Axial nonenhanced computed tomography images

Clinical Progress

On the second day following head trauma, the patient had polyuria (>2000 cc/m^2/day) and hypernatremia (serum sodium 167 mmol/L with a paired urine density of 1004 gr/cm^3), thus he was diagnosed with central diabetes insipidus and desmopressin treatment was initiated (2x10 ug/m^2). Therewithal, after testing the anterior pituitary hormones, hydrocortisone and L-thyroxine treatments were started due to central hypothyroidism [TSH: 0.31 µIU/mL (normal range: 0.35–5.6), fT4: 0.2 ng/dL (normal range: 0.61-1.12)]; and hypocortisolism [cortisol 2.3 µg/dL (normal range: 3.7-19.4)]. The patient was stayed in the hospital for six months. During the past six months, he also had recurrent episodes of a prolonged febrile illness of unknown origin (Figure 3). All investigations related to infectious, autoimmune and neoplastic diseases were negative and the fever was considered to be of central origin due to the hypothalamic injury.

On follow-up, the patient had a decreased height velocity [2cm/year (-4.4 SDS)] together with reduced serum insulin-like growth factor-1 (IGF-1) (50.3 ng/ml, -2.03 SDS) and serum IGF binding protein-3 (1570 ng/ml, -3.45 SDS) concentrations. Therefore, GH stimulation test were done with L-dopa and clonidine showed a peak GH of 0.7 ng/ml, 0.6 ng/ml, respectively. Because of inadequate response to growth hormone in two provocative tests, he was started on rhGH at a dose of 25 µg/kg/d. On the fifth day of the rhGH treatment, his fever was controlled and he never had fever during three months follow-up.

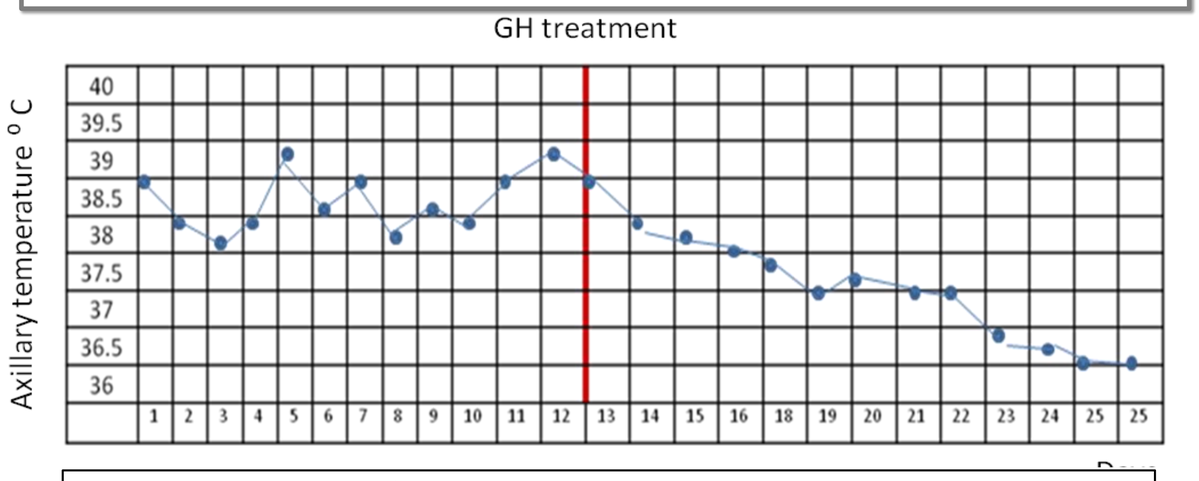

Figure 2. Fever chart of the patient

Discussion

Several studies show that GH, either directly or through IGF-1, influences sweat secretion. GH receptors have been demonstrated in human eccrine sweat gland cells. Thus, GH may effect directly on the eccrine sweat gland cells. Sweating, that increased with GH, is a known phenomenon of hyperpituitarism. Therewithal, it has recently been shown that patients with GH deficiency have a reduced sweating capacity which increases the risk of developing hyperthermia. Thus, the patients with GH deficiency might be at risk for developing hyperthermia secondary to disruption of sweat secretion. Therewithal, it was reported that sweat secretion rate correlates significantly to serum IGF-I levels in patients with GH deficiency.

Patients with hypopituitarism secondary to posttraumatic brain injury, may have highly variable symptoms. This case report describes a patient with prolonged febrile illness of unknown origin, who presents with GH deficiency due to a previous head trauma. With this report, we would like to emphasize that in cases with a fever of unknown origin and traumatic brain injury, GH deficiency should be considered, and complete pituitary evaluation should be performed.