Int J Endocrinol Metab. 2010;8(2):97-99
International Journal of
Endocrinology & Metabolism
Journal home page: www.IJEM.org

Panhypopituitarism and empty sella after ischemic stroke: A case report

Mahmood Soveid [1*], Gholam Hossein Ranjbar Omrani [1]

[1] Endocrinology and Metabolism Research Center, Shiraz University of Medical Sciences, Shiraz, IR Iran

ARTICLE INFO

Article Type:
Case Report

Article history:
Received: 20 Jan 2010
Revised: 1 Feb 2010
Accepted: 16 Feb 2010

Keywords:
Stroke
Pituitary disease
Empty sella syndromes

ABSTRACT

Stroke is a leading cause of death and disability. Recent studies indicate that some degree of pituitary dysfunction may be observed after an acute stroke and this condition is commonly misdiagnosed. Reported here is the case of a 65 year- old man who developed symptoms of pituitary deficiency a few months after a frontal lobe stroke and finally presented with hypoglycemia and coma. Investigation revealed panhypopituitarism and presence of empty sella and a very small pituitary. He responded to treatment well.

Conclusion: Patients with history of ischemic stroke and suggestive symptoms need evaluation of pituitary function.

Please cite this paper as:
Soveid M, Ranjbar Omrani GH. Panhypopituitarism and empty sella after ischemic stroke: A Case report. *Int J Endocrinol Metab*.2010;**8**(2):97-99.

Introduction

Strokes, a common cause of mortality and morbidity in Iran and other countries, are mostly due to ischemic cerebral infraction (1). Pituitary insufficiency is a known complication of subarachnoid hemorrhage and traumatic brain injury (2) but there is limited data available pituitary dysfunction after brain ischemia. These is growing evidence that pituitary function can be reduced after ischemic stroke (3- 5). Since a substantial number of survivors of ischemic stroke suffer from fatigue and depression, and these symptoms overlap with those of hypopituitarism, the diagnosis may be missed or delayed with serious consequences (6). We report a patient who developed extreme form of pituitary dysfunction i.e. panhypopituitarism and empty sella after an ischemic stroke.

* *Corresponding author at:* Mahmood Soveid, Department of Internal Medicine, Namazi Hospital, P.O BOX: 71937-11351, Shiraz, IR Iran. Fax: +98-7116474316.

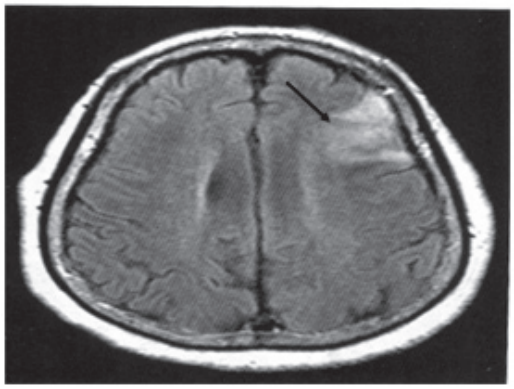
E-mail: msoveid@sums.ac.ir

Case Report

A 65 year old man and the father of four children was brought to emergency room due to loss of consciousness for 2 hours duration. About one year prior to admission he developed right sided weakness. Brain MRI at that time showed ischemic stroke in the territory of left middle cerebral artery and pituitary gland was grossly normal *(Figure 1)*. He was treated with antiplatelet agents and atorvastatin and had some improvement, but a few months later he gradually developed anorexia, occasional vomiting, weakness, and a 30 kg weight loss. He referred to some physicians and his symptoms were attributed to depression and sequella of stroke and anti depressant agents were started without any improvement. He gradually became bedridden. He had episodes of drowsiness and finally became comatose. On admission he was cachectic and unconscious, blood pressure was 90/70 mmHg.

On physical examination he had right sided weakness, muscle wasting, and superficial bed sores; his body hair and genitalia were normal. Initial lab data revealed Hb=10 g/dL, BUN=12 mg/dL, creat=0.9 mg/dL, Na=118 meq/L, K=3.1 meq/L, blood sugar=38mg/dL. He was treated with hypertonic saline and dextrose. Hydrocortisone was also

a)



b)

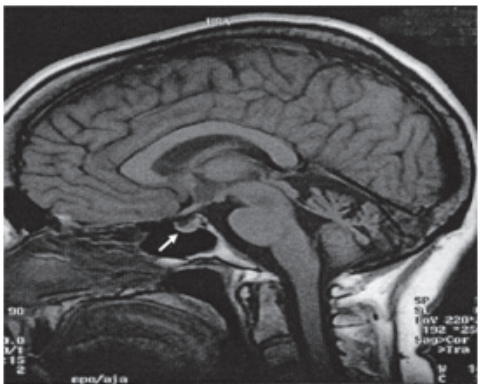


Figure 1. a) MRI of brain taken one year before admission showing ischemic infarction in left frontal lobe ; b) normal appearance of pituitary gland

started with possible impression of adrenal insufficiency and he had a dramatic clinical response. Result of hormonal work up during hypoglycemia was as follows: LH = 5.2 Iu/L (NL: 2.8-6.8), FSH = 3.8 Iu/L (NL = 1.3-11.8), testosterone = 1.3 ng/mL (NL = 2.8-8), cortisol = 3.2 μg/dL (NL = 5-25), ACTH = 2.6 pg/mL. (NL = 7.2-63.3), growth hormone = 1.5 μg/L (NL = 0.5-17), prolactin = 4.5 μg/L (NL = 0-20), TSH = 1.1 mIu/L (NL = 0.5-4.5), T4 = 2.1 μg/dL (NL = 5.5-12), T3 = 50 ng/dL (NL = 80-180), T3Ru = 22% (NL = 25-35%). Serum ferritin was 42 μg/L (NL = 29-250). 24 hours urine volume and specific gravity were 1400 cc and 1.018 respectively. Pituitary MRI showed empty sella with a very narrow rim of pituitary *(Figure 2)*.

After three days of treatment, the patient was fully conscious, able to sit and walk and had a good appetite. He was discharged on prednisolone 7.5 mg/day and levothyroxine 75 μg/day. His follow up showed him to be active despite a mild right sided weakness and he gained 25 kg weight. Growth hormone and testosterone were not replaced because he had high PSA and urologic investigation re-evealed stage 1 prostatic cancer. The patient was re-evaluated for hypopitutarism nine months later, which showed persistence of deficiency of all anterior pituitary hormones.

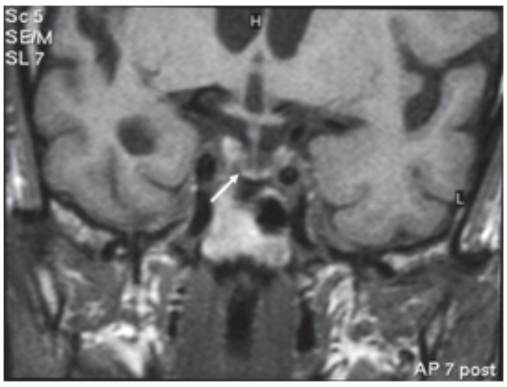


Figure 2. MRI of pituitary taken during admission which shows empty sella and small remnant of pituitary

Discussion

Recent studies have indicated that isolated or multiple pituitary deficits are common after ischemic stroke (3, 7) and, if untreated, these can aggravate the morbidity observed after cerebral injury (8). Most of the post stroke deficiencies are isolated, growth hormone being the most frequent followed by central hypogonadism and rarely ACTH deficiency, but no case of TSH deficit is reported (7). Our patient is unique in that he had deficiency of all pituitary hormones and development of pituitary necrosis leading to empty sella. TSH deficiency has not been reported previously in post-ischemic stroke hypopituitarism. Our patient had persistent low TSH and thyroid hormones, nine months after presentation, excluding the possibility of non-thyroid illness syndrome. The cause and effect relationship between pituitary deficiency and stroke in our patient is supported by the fact that he had no symptoms referrable to pituitary dysfunction before his stroke and initial pituitary view was normal. Infarction of pituitary is a known cause of empty sella (9). The mechanisms responsible for pituitary damage after ischemic stroke are not fully understood. Several mechanisms have been hypothesised, including vasospasm, development of brain edema, and abnormalities in local neuroendocrine mediators (10). It is more frequently associated with involvement of cortical structures compared with nuclear localization and related to severity of stroke (7). According to one hypothesis, disturbance of long hypophyseal portal system which is highly vulnerable to ischemia may be responsible for pituitary deficiency (6, 11). The fact that growth hormone and LH/FSH deficiencies are the most common pituitary alterations after ischemic stroke, support the vascular damage concept (6). The post stroke pituitary deficiency can improve or worsen over time but in the majority of the patients, as in our case, it persists permanently (7). Our case illustrates that patients with history of stroke and symptoms such as anorexia, weight loss, and hypotension should be evaluated for pituitary function disturbances.

Financial support

None declared.

Conflict of interest

None declared.

References

1. Azarpazhooh MR, Etemadi MM, Donnan GA, Mokhber N, Majdi MR, Ghayour-Mobarhan M, et al. Excessive incidence of stroke in Iran: evidence from the Mashhad Stroke Incidence Study (MSIS), a population-based study of stroke in the Middle East. *Stroke.* 2010;**41**(1):e3-e10.
2. Schneider HJ, Kreitschmann-Andermahr I, Ghigo E, Stalla GK, Agha A. Hypothalamopituitary dysfunction following traumatic brain injury and aneurysmal subarachnoid hemorrhage: a systematic review. *JAMA.* 2007;**298**(12):1429-38.
3. Boehncke S, Ackermann H, Badenhoop K, Sitzer M. Pituitary function and IGF-I levels following ischemic stroke. *Cerebrovasc Dis.* 2011;**31**(2):163-9.
4. Bondanelli M, Ambrosio MR, Onofri A, Bergonzoni A, Lavezzi S, Zatelli MC, et al. Predictive value of circulating insulin-like growth factor I levels in ischemic stroke outcome. *J Clin Endocrinol Metab.* 2006;**91**(10):3928-34.
5. Bondanelli M, Ambrosio MR, Zatelli MC, Basaglia N, Degli Uberti EC. Prevalence of hypopituitarism in patients with cerebrovascular diseases. *J Endocrinol Invest.* 2008;**31**(9 Suppl):16-20.
6. Bondanelli M, Ambrosio MR, Zatelli MC, De Marinis L, degli Uberti EC. Hypopituitarism after traumatic brain injury. *Eur J Endocrinol.* 2005;**152**(5):679-91.
7. Bondanelli M, Ambrosio MR, Carli A, Bergonzoni A, Bertocchi A, Zatelli MC, et al. Predictors of pituitary dysfunction in patients surviving ischemic stroke. *J Clin Endocrinol Metab.* 2010;**95**(10):4660-8.
8. Bondanelli M, Ambrosio MR, Cavazzini L, Bertocchi A, Zatelli MC, Carli A, et al. Anterior pituitary function may predict functional and cognitive outcome in patients with traumatic brain injury undergoing rehabilitation. *J Neurotrauma.* 2007;**24**(11):1687-97.
9. Degli Uberti EC, Teodori V, Trasforini G, Tamarozzi R, Margutti A, Bianconi M, et al. [The empty sella syndrome. Clinical, radiological and endocrinologic analysis in 20 cases]. *Minerva Endocrinol.* 1989;**14**(1):1-18.
10. Franceschini R, Tenconi GL, Zoppoli F, Barreca T. Endocrine abnormalities and outcome of ischaemic stroke. *Biomedecine & Pharmacotherapy.* 2001;**55**(8):458-65.
11. Qiao M, Meng S, Foniok T, Tuor UI. Mild cerebral hypoxia-ischemia produces a sub-acute transient inflammatory response that is less selective and prolonged after a substantial insult. *Int J Dev Neurosci.* 2009;**27**(7):691-700.