A Case of Sheehan’s Syndrome that Manifested as Bilateral Ptosis

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INTRODUCTION

Ptosis may be due to myogenic, neurogenic, aponeurotic, mechanical or traumatic causes. The most frequent disorder causing myogenic ptosis is myasthenia gravis (1). Hypothyroidism can cause a variety of signs and symptoms of the neuromuscular system (2). However, in the absence of other obvious etiologies, ptosis in a patient with hypothyroidism is very rare (3-5). We report here on a case of central hypothyroidism that was due to Sheehan’s syndrome and the patient manifested with bilateral ptosis, and the patient was initially suspected as having myasthenia gravis.

CASE DESCRIPTION

The patient was a 51-yr-old woman with swelling of the extremities and bilateral ptosis on March 8, 2010. She complained of exertional dyspnea and weakness. About 25-yr ago, she had a history of severe postpartum vaginal bleeding. The laboratory studies demonstrated hypopituitarism with secondary hypothyroidism. The ptosis was improved by replacement of thyroid hormone. Hypothyroidism should be considered in the differential diagnosis of patients who manifest with ptosis and that prompt replacement of hormone can lead to a complete recovery.

Key Words: Hypothyroidism; Hypopituitarism; Ptosis

Hypothyroidism can cause a variety of signs and symptoms of the neuromuscular system. However, ptosis in a patient with hypothyroidism is very rare. We report here on a case of central hypothyroidism that was due to Sheehan’s syndrome and it manifested as bilateral ptosis in a 51-yr-old woman. She complained of exertional dyspnea and weakness. About 25-yr ago, she had a history of severe postpartum vaginal bleeding. The laboratory studies demonstrated hypopituitarism with secondary hypothyroidism. The ptosis was improved by replacement of thyroid hormone. Hypothyroidism should be considered in the differential diagnosis of patients who manifest with ptosis and that prompt replacement of hormone can lead to a complete recovery.

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a history of massive postpartum vaginal bleeding. Thereafter she did not resume menses. The basal levels of other hormones were characterized by a serum cortisol of 4.03 μg/dL, the adrenocorticotropic hormone (ACTH) was 29.64 pg/mL, the growth hormone (GH) was 0.06 ng/mL, the IGF-1 was 25 ng/mL (normal reference: 71-263 ng/mL), the prolactin was 1.59 ng/mL, the luteinizing hormone (LH) was 1.49 IU/L, the follicle-stimulating hormone (FSH) was 4.91 IU/L, the E2 was 17.29 pg/mL and the testosterone was 0.02 ng/mL. The combined pituitary stimulation test, including the insulin tolerance test, the thyrotropin-releasing hormone (TRH) stimulation test and the gonadotropin-releasing hormone (GnRH) stimulation test showed panhypopituitarism (Table 1). Brain magnetic resonance imaging (MRI) showed a finding of an empty sella turcica and there was no evidence of an intracranial mass, hemorrhage and aneurysm (Fig. 2).

Nerve conduction study (NCS) and electromyography (EMG) of the limbs revealed normal results with the exception of incidentally detected carpal tunnel syndrome. Needle EMG of the orbicularis oculi showed no evidence of dysfunction of the neuromuscular junction.

We diagnosed her as having Sheehan’s syndrome, bilateral ptosis and subclinical myopathy caused by secondary hypothyroidism. Replacement with prednisolone of 10 mg/day and thyroxine (T4) 100 μg/day was started. The dose of prednisolone was decreased to 5 mg/day after 4 weeks. At 3 months follow-up, she had become euthyroid with normalized muscle enzymes. She recovered from her presenting symptoms and the bilateral ptosis was much improved (Fig. 1B).

**DISCUSSION**

Sheehan’s syndrome occurs as a result of ischemic pituitary necrosis due to severe postpartum hemorrhage. Although a small percentage of patients with Sheehan’s syndrome may have an abrupt onset of severe hypopituitarism immediately after delivery, most patients have mild disease and they go undiagnosed for a long time and they are treated inappropriately (6). In this current patient, chronic fatigue and weakness had persisted, but it had gone undiagnosed for about 20 yr.

Initially, her symptoms such as ptosis, progressive dyspnea and weakness were suspicious for myasthenia gravis. Myasthenia gravis is an acquired neuromuscular disorder that is characterized by fatigability and fluctuating weakness of the skeletal muscles, and especially eye muscle weakness. The weakness is

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**Table 1.** Results of the combined pituitary stimulation test

| Time (min) | Glucose (mg/dL) | GH (ng/mL) | Cortisol (μg/dL) | Prolactin (ng/mL) | TSH (μU/mL) | LH (IU/L) | FSH (IU/L) |
|------------|-----------------|------------|------------------|------------------|-------------|-----------|------------|
| 0          | 82              | 0.06       | 4.03             | 1.59             | 2.08        | 1.49      | 4.91       |
| 30         | 39              | 0.07       | 4.32             | 2.02             | 2.66        | 2.56      | 5.27       |
| 60         | 202             | 0.16       | 4.44             | 1.59             | 2.65        | 3.04      | 4.96       |
| 90         | 108             | 0.14       | 4.42             | 1.55             | 2.66        | 3.51      | 5.67       |
| 120        | 62              | 0.06       | 3.87             | 1.40             | 2.6         | 3.22      | 5.60       |

GH, growth hormone; TSH, thyroid-stimulating hormone; LH, luteinizing hormone; FSH, follicle-stimulating hormone.
caused by circulating antibodies that block the acetylcholine receptors at the postsynaptic neuromuscular junction, and this inhibits the stimulative effect of the neurotransmitter acetylcholine (7). In most cases, the first noticeable symptom is weakness of the eye muscles. Therefore, the presence of ptosis necessitates the exclusion of underlying ocular myasthenia gravis (4). In this patient, the acetylcholine receptor binding antibody was negative and needle EMG of the orbicularis oculi showed no evidence of dysfunction of the neuromuscular junctions. Also, the ptosis was improved by replacement of thyroid hormone. Therefore, myasthenia was adequately excluded.

Ptosis has been previously reported in patients with primary hypothyroidism, but there have been only a few case reports (3-5). In patients with hypothyroidism, myopathy, mononeuropathy and diffuse peripheral polyneuopathy may encountered with various incidences (8). Carpal tunnel syndrome is the most frequently observed in hypothyroidism. Polyneuropathy and myopathy are also well known (8). Patients can complain of muscle and joint pain, cramps, fatigue and weakness. However, involvement of a cranial nerve in a patient with hypothyroidism is very rare. Although neuromuscular complications are well recognized in thyroid disorders, their pathophysiology remains unclear. Generally, these abnormalities are secondary to mucopolysaccharide infiltration of various peripheral organs (5). Cho et al. (9) described a case of pituitary apoplexy with Sheehan’s syndrome and isolated third cranial nerve palsy. In their case, direct mechanical compression of the third cranial nerve or the vascular supply to the nerve resulted in a sudden onset of isolated third cranial nerve palsy. The patient’s ptosis was improved by surgical decompression. However, our patient had long standing secondary hypothyroidism and the ptosis had an insidious onset. The patient’s ptosis was improved by replacement of hormone.

Moderate myopathy frequently develops in patients with hypothyroidism. This myopathy is most often manifested as myalgia, muscle stiffness, cramps and sometimes elevated plasma levels of creatinine phosphokinase. Infrequently, rhabdomyolysis can also develop in patients with hypothyroidism (10). Our patient had no specific symptoms of myopathy, but she had elevated levels of muscle enzyme. After replacement of hormone, the muscle enzymes were normalized.

Adrenal insufficiency is can produce generalized muscle weakness, muscle cramping and fatigue. However, except for generalized weakness, musculoskeletal manifestations have rarely been described in adrenal insufficiency (11) and which is thought to be due to the electrolyte abnormalities in primary adrenal insufficiency (12). Our patients had secondary adrenal insufficiency and mild hyponatremia. Therefore we thought that our patient’s musculoskeletal manifestations with ptosis is due to mainly central hypothyroidism.

We report here on an unusual case of ptosis associated with central hypothyroidism due to Sheehan’s syndrome. It is concluded that hypothyroidism should be considered in the differential diagnosis of patients who manifest with ptosis and that prompt replacement of hormone can lead to a complete recovery.

REFERENCES

1. Finsterer J. Ptosis: causes, presentation, and management. Aesthetic Plast Surg 2003; 27: 193-204.
2. Penza P, Lombardi R, Camozzi F, Ciano C, Lauria G. Painful neuropathy in subclinical hypothyroidism: clinical and neuropathological recovery after hormone replacement therapy. Neurol Sci 2009; 30: 149-51.
3. Green PH. Bilateral ptosis and proximal myopathy—muscular manifestations of hypothyroidism. Aust N Z J Med 1977; 7: 418-19.
4. Lo YL, Ho SC, Koh LK, Khoo DH. EMG myokymia as a cause of ptosis in hypothyroidism. Eur J Neurosurg 2003; 10: 87-90.
5. Narberhaus Donner B, Aguilar Cortés E, Playán Usón J, Berdún Cheliz MA, Bernat Badía A. Third cranial nerve paralysis associated with hypothyroidism. J Neurol 1992; 239: 176-7.
6. Keleştimur E. Sheehan’s syndrome. Pituitary 2003; 6: 181-8.
7. Conti-Fine BM, Milani M, Kaminski HJ. Myasthenia gravis: past, present, and future. J Clin Invest 2006; 116: 2843-54.
8. Somay G, Oflazoğlu B, Us O, Surardamar A. Neuromuscular status of thyroid diseases: a prospective clinical and electrodiagnostic study. Electromyogr Clin Neurophysiol 2007; 47: 67-78.
9. Cho WJ, Joo SP, Kim TS, Seo BR. Pituitary apoplexy presenting as isolated third cranial nerve palsy with ptosis: two case reports. J Korean Neurosurg Soc 2009; 45: 118-21.
10. Kisakol G, Tunc R, Kaya A. Rhabdomyolysis in a patient with hypothyroidism. Endocr J 2003; 50: 221-3.
11. Mor F, Green P, Wysenbeek AJ. Myopathy in Addison’s disease. Ann Rheum Dis. 1987; 46: 81-3.
12. Kendall-Taylor P, Turnbull DM. Endocrine myopathies. Br Med J (Clin Res Ed) 1983; 287: 705-8.