Case Report

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Primary Hypothyroidism with Facial Paralysis: A Case Report

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Abstract

Objective: Neurological features like parasthesias and carpel tunnel syndrome is not uncommon in patients with hypothyroidism but facial palsy with hypothyroidism has scarcely been reported. Case report: 40 year old male patient presented as isolated right sided infranuclear facial nerve palsy. Prednisolone (60 mg/day) was prescribed as we expected it to be Bell's palsy, however, the patient's facial palsy did not improve completely. Further inquiry revealed that he had overt primary hypothyroidism. He was put on Levothyroxine and there was complete recovery of facial palsy in two months. Conclusions: This case report highlights that facial palsy is not always idiopathic (Bell's) palsy which could be part of some underlying multisystemic disorder and prompt recognition and treatment of the associated disorder should offer the best chance for complete recovery.

Keywords: Hypothyroidism; Bell's Palsy; Levothyroxine.

Introduction

The anatomy of the facial nerve is such that it has a long intracranial course and has to pass through the narrow facial canal. Hence, it is highly prone to injury due to middle ear or temporal bone infections, trauma, surgery or compression by a tumor. Peripheral, unilateral facial palsy may be Bell's palsy but it could also be the first presentation of an underlying multisystem disorder.[1] Very few case reports have found facial palsy to be associated with hypothyroidism in adults. We report a case of severe primary hypothyroidism with palsy of the right facial nerve.

Case Report

A 40 year old male patient presented as isolated right sided infranuclear facial nerve palsy. Prednisolone (60 mg/day) was prescribed as we expected it to be Bell's palsy however; the patient's facial palsy did not improve completely. On further inquiry he gave a history of lethargy, easy fatigability, cold intolerance, constipation and weight gain over the past one year. The patient had no history of ear ache or discharge, any viral infection, medication or exposure to high levels of iodide.

On general physical examination, he had a pulse rate of 60 beats per minute. His blood pressure was 110/70 mmHg and he was pale and puffy with coarse facial features, hoarse voice, dry and thickened skin and non-pitting pedal edema. However, no goiter was found. Delayed relaxation of ankle jerk was present bilaterally. His fundus was normal.

Laboratory data revealed normocytic normochromic anemia (hemoglobin, 10.0 g/dL), haematocrit of 32 percent. Kidney function tests and serum electrolytes were normal. Serum cholesterol was elevated (350 mg/dl) and serum triglycerides were also elevated (395mg/dL). Further testing showed elevated thyroid stimulating hormone (TSH) >100 µIU/mL (normal range, 0.5 to 5.5 µIU/mL), decreased total thyroxine level (2.3 µg/dL) (normal range 4.5 to 12.0 µg/dL), decreased total triiodothyronine level (0.5 ng/mL) (1.19 to 2.18 ng/mL).Serum antithyroid peroxidase antibodies were elevated (1,200 IU/mL) (>35 IU/mL).

The patient was started on a dose of 50 mcg daily of

Levothyroxine and dose was gradually increased to 100 mcg daily. After levothyroxine treatment was initiated, the symptoms of facial palsy recovered completely and the patient's clinical condition also improved within eight weeks (Figure 1 & 2). When last seen the patient was euthyroid while on 100 mcg of thyroxin daily. His thyroid function tests were normal (Table1).

Discussion

The manifestations of hypothyroidism are varied and may affect any of the body systems. In recent years, increased emphasis has been placed on the neuromuscular manifestations of hypothyroidism. The neurologic manifestations of hypothyroidism are highly variable and can affect both the central nervous system as well as the peripheral nervous system [2]. The most common form of mononeuropathy seen in such patients is carpal tunnel syndrome, which results from compression of the median nerve by myxedematous tissue around it [3]. The anatomical course of the facial nerve is such that it has a long intracranial course and its confinement in the narrow, bony facial canal makes it subject to the usual Bell's palsy. This could also make it highly vulnerable to myxedematous infiltration and soft tissue swelling seen in hypothyroidism. Thus, peripheral facial palsy with hypothyroidism may be a nerve entrapment syndrome like carpal tunnel syndrome. Bell palsy is an acute unilateral idiopathic paralysis of the facial nerve. Its pathophysiology is unknown, but the autoimmune system was proposed to be involved by causing local damage to myelin after activation by a viral infection [4].

In our patient, classic unilateral facial palsy developed in association with acquired hypothyroidism. Our patient had no history of a viral infection. Prompt recognition and correction of the hypothyroid state was associated with rapid and complete recovery from the facial palsy. Earll JM, Kolb FO described two case of hypothyroidism with facial palsy [5]. The hypothyroid state was not recognized at first and the facial paralysis did not completely abate in spite of giving steroids in our patient. It is unlikely that the therapeutic dose of prednisolone briefly used in the patient played a significant role in the patient's improvement. Hence, the facial palsy of our patient may not have been due to Bell's palsy, but rather due to an entrapment neuropathy. Cox NH and coworkers illustrate a case of Bell's palsy associated with hypothyroidism [6]. Similarly a case of facial palsy with hypothyroidism in a child of 13 years age was reported by Lee HJ et al [7]. The most common cause of acquired hypothyroidism in children and adults is chronic autoimmune thyroiditis. Mostly such

Table 1: Thyroid functions and lipid profile before and after thyroxine treatment

Parameter	Before treatment	After Treatment
T3(ng/ml)	0.5	1.90
T4(ug/dl)	2.3	9.0
TSH (uIU/ml)	>100	2.8
Total cholesterol(mg/dl)	350	170
Triglycerides(mg/dl)	395	150
HDL(mg/dl)$	50	55
LDL(mg/dl)#	221	85
VLDL(mg/dl)*	79	30

HDL=High density lipoprotein$, LDL=Low density lipoprotein#, VLDL=Very low density lipoprotein*.

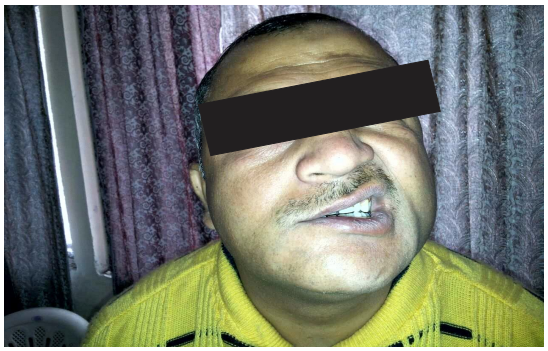

Fig. 1: Patient with Right Sided Facial Palsy before treatment with L- Thyroxine.

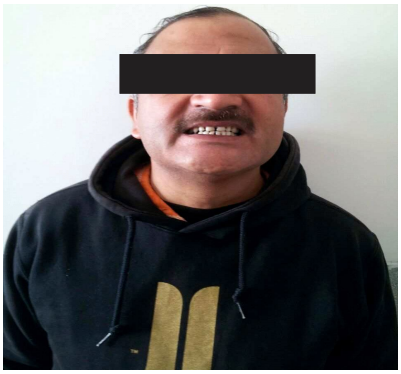

Fig. 2: The same patient with complete recovery of the facial paralysis following treatment with L-Thyroxine at 8 weeks.

patients has goiter at presentation. However, some patients have no goiter or even have an atrophic thyroid gland [8].

Conclusions

This case report highlights that all facial palsy is not Bell's palsy and could be part of some underlying disorder and prompt recognition and treatment of the associated disorder should offer the best chance for complete recovery.

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