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Case Report

Facial Diplegia in a Patient with Chronic Hepatitis B Infection: Case Report

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Abbreviations FNP: Facial Nerve Palsy; GBS: Guillain-Barré syndrome; COPD: Chronic Obstructive Pulmonary Disease; CSF: Cerebrospinal Fluid; ESR: Erythrocyte Sedimentation Rate; CBC: Complete Blood Count; EMG: Electromyography; NCS: Nerve Conduction Study; CNS: Central Neural System; MRI: Magnetic Resonance Imaging; PCR: Polymerase Chain Reaction; SLE: Systemic Lupus Erythematosus; PAN: Polyarteritisnodosa; ACE: Angiotensin Converting Enzyme; AIDP: Acute Inflammatory Demyelinating Polyradiculopathy

Abstract

Facial diplegia or bilateral facial nerve palsy is an extremely rare condition, caused in the majority of patients by an underlying condition. The broad differential diagnosis of facial diplegia causes may present a diagnostic challenge. We report the case of a 71-year-old patient with sudden onset of bilateral facial nerve palsy and mild hypoglossal nerve paresis. The diagnosis of Guilain-Barré syndrome was the most likely and the patient recovered after intravenous immunoglobulin administration.

Introduction

Bell's palsy is a common neurological entity, with an incidence of 25 per 100,000 populations. It is considered as the main cause in 70% of the cases of unilateral facial nerve palsy [1].

On the other hand bilateral facial nerve palsy is a very rare disorder with an incidence of 1 case per 5 million populations. An underlying etiology is present in the majority of these cases with Guilain-Barré syndrome variants, Lyme disease and neurosarcoidosis and Ebstein-Barr virus infections being the most common [2-4].

We report the case of a 71-year-old patient who was diagnosed with a GBS variant after presenting acute bilateral facial nerve palsy, and recovered successfully after intravenous immunoglobulin administration. The diagnostic evaluation and the differential diagnosis of the patient is discussed, since the number of cases presented in literature is small and such cases may be a diagnostic challenge to medical practitioners.

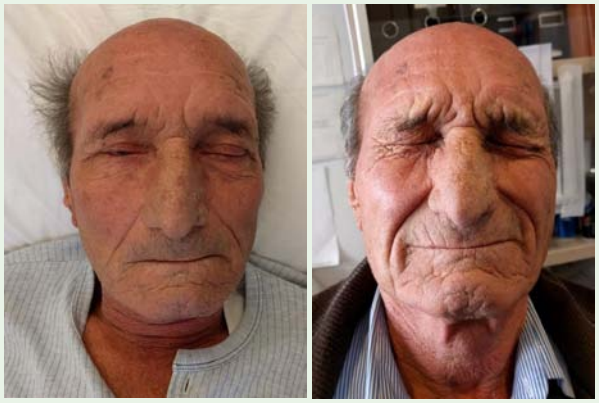
Case Report

A 71-year-old male was admitted to our clinic with acute onset of dysarthria, facial muscle and mimic muscles weakness. The patient suffered from chronic hepatitis B infection (treated with Tenofovir) and his past medical history included hypertension, COPD and poliomyelitis (during childhood). The patient recalled travelling to a Greek island almost a month ago prior to his admission.

Physical examination revealed bilateral peripheral facial nerve palsy of moderate severity (House-Brackmann grade 4) being worse on the left side, with mild weakness of the patient's tongue muscles. His rest physical examination and basic laboratory tests (CBC, biochemistry, ESR) were unremarkable. Further imaging with an MRI brain scan was normal. The patient had undergone a lumbar puncture that showed a raised cerebrospinal fluid (CSF) protein level (proteins: 69 mg/dl), mild leukocytosis (leukocytes: 39 /mm^3), normal CSF erythrocytes and glucose (Glucose: 56 mg/dL, Erythrocytes: 10 mg/dL) and negative CSF culture. Further CSF evaluation for Cryptococcus antigen, Indian ink stain, acid-fast bacilli stain, angiotensin converting enzyme, CSF electrophoresis for oligoclonal bands and cytology were negative. CSF viral screening and polymerase chain reaction (PCR) specifically for West Nile Virus (due to travel history) tested negative.

The patient tested negative for tuberculin skin testing, syphilis and Lyme disease. In addition viral screening was negative, besides the known chronic hepatitis B. Thyroid function, vitamin B12/folic acid levels, immunoglobulin's (IgG, IgM, IgA) were normal. Due to mild proteinuria (protein: 745mg /24 hour urine collection) further autoimmune screening was performed, with anti-dsDNA antibodies being borderline increased (36 IU/mL) and complement (C3, C4) levels were found normal

Furthermore the patient's facial nerve electromyography (EMG) and nerve conduction study (NCS) showed electrophysiological evidence of severe denervation in the facial nerve territory and delayed conduction of facial and trigeminal nerves, consistent with multiple lower cranial nerve neuropathies of recent onset.



(1a) (1b)

Figure 1: The patient's clinical presentation with bilateral facial nerve palsy, during his admission (1a) and 2 weeks after the administration of intravenous immunoglobulin (1b) (the patient was ordered to close eyes tightly).

The diagnosis of Guillain-Barré syndrome was most dominant, thus the patient was administered Intravenous Immunoglobulin 0.4 grams/kilogram of body weight for seven days along with vitamin B-complex supplementation and natural tears. In parallel the patient underwent facial nerve exercises, physiotherapy and electrical nerve stimulation.

The patient showed mild improvement during his hospitalization and facial nerve palsy improved dramatically during his follow-up 2 weeks after his discharge. Follow-up EMG and NCS showed facial and trigemal nerve innervation and improvement in comparison with the first study (Figure 1).

Discussion

Facial diplegia has an incidence of 1 case per 5 million people according to literature [2] and may be secondary of life-threatening diseases such as autoimmune diseases, traumas, central neural system (CNS) infections, neoplasm's or demyelinating diseases [3,4].

Any secondary causes (Table 1) of bilateral facial nerve palsy (FNP) should be excluded in order to attribute such a clinical manifestation to idiopathic Bell's palsy.

The patient suffered from chronic hepatitis B infection and was treated with Tenofovir for the last decade. Tenofovir has not, to-date, been reported in the bibliography to cause cranial nerve palsy. However, an association between HBV infection or HBV-HDV co-infection and unilateral cranial nerve palsy has been recorded in the bibliography, although the mechanism is not known [5-7].

Table 1: List of the most common etiologies of bilateral facial nerve palsy [1,4].

A/A	Etiology
1.	Lyme disease
2.	Acute Inflammatory Demyelinating Polyradiculopathy or Guilain – Barré syndrome
3.	Neurosarcoidosis
4.	Viral infections (HSV, HIV, VZV, EBV, West Nile Virus, etc.)
5.	Autoimmune diseases (vasculitis, systemic lupus erythematosus, polyarteritisnodosa
6.	Syphilis
7.	Tuberculosis
8.	CNS tumors / hematological malignancies
9.	Amyloidosis

Infection by *Borrelia burgdorferi* or Lyme disease is among the commonest causes of bilateral FNP, especially in countries endemic for the specific spirochete [8]. The patient had a history of travelling to a Greek island without any exposure to ticks but with possible exposure to mosquitoes. Thus the possibility of Lyme disease was distant; however CSF PCR for West Nile Virus infection (that is endemic in some areas of Greece) and other viruses was also negative [9,10].

Due to the increased protein level and leukocytes in the CSF (confirmed by lumbar puncture) a thorough investigation for secondary causes of bilateral FNP ensued. Further CNS imaging with MRI Brain scan excluded multiple other causes of facial diplegia such as space occupying lesions, neoplasms, brain lymphoma and demyelinating disease (the CSF electrophoresis for oligoclonal bands was also negative).

Low ESR and negative autoimmune screening made the diagnosis of systemic lupus erythematosus (SLE), sarcoidosis and polyarteritisnodosa (PAN) less probable [11,12]. Moreover, negative angiotensin converting enzyme (ACE) in CSF was more compatible with a diagnosis other than neurosarcoidosis [13].

Acute inflammatory demyelinating polyradiculopathy (AIDP) or Guilain-Barré syndrome (GBS) was a possible diagnosis due to mild impairment of the XII cranial nerve in addition to bilateral FNP. GBS may manifest with bilateral FNP in 27-50% of its cases [14,15].

Nerve conduction study and EMG performed had electrophysiological findings compatible with demyelination and neuropathies of lower cranial nerves. These findings in addition to CSF findings and normal deep tendon reflexes of lower limbs, made the diagnosis of GBS variant most probable. The mild CSF Leukocytosis (leukocytes greater than 11/mm^3) may be found in approximately 4% of GBS cases [16].

The disease usually presents with bilateral symmetric lower limbs weakness with concomitant or reduced deep tendon reflexes. During the disease's progression complete paralysis of extremities, facial, respiratory and extra ocular muscles may present. Other manifestations of the disease include paresthesias of upper and lower extremities in 80% and dysautonomia (tachycardia, bradycardia, hypotension, hypertension, orthostatic hypotension, urinary retention) in 70% of patients [16].

Patients with GBS are treated with either intravenous immunoglobulin in doses 0.4 gram/kg for five to seven days or plasmapheresis for 5 days or more according to clinical response. The disease prognosis is very good and in almost 90% of patients no neurological deficits are found in long-term (in 6 months) follow-up [16].

Conclusion

The rarity of simultaneous and bilateral facial nerve palsy makes the diagnosis of a possible underlying cause a necessity. The broad differential diagnosis of those causes is a challenge for a physician and includes a variety of diseases such as neoplasm's, autoimmune

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diseases, inflammatory and infectious diseases. Bilateral facial diplegia with possible concomitant paresis of other cranial nerves, the CSF abnormalities described and electrophysiological evidence of denervation of cranial nerves confirm the diagnosis of GBS. Appropriate management with plasmapheresis or intravenous immunoglobulin's and ventilator support when needed are of profound importance for a favorable clinical improvement of patients.

Informed Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

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