International Journal of Scientific Reports
Srinivas V et al. Int J Sci Rep. 2015 Jun;1(2):136-138
http://www.sci-rep.com
pISSN 2454-2156 | eISSN 2454-2164

DOI: http://dx.doi.org/10.18203/issn.2454-2156.IntJSciRep20150222

Case Report

Bilateral facial palsy with hypertension: a case report

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Received: 18 May 2015
Accepted: 13 June 2015

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ABSTRACT

Bilateral facial paralysis is a rare condition, we report a case of a 70-year-old healthy, illiterate man with sequential bilateral facial paralysis as a manifestation of unknown cause after exclusion of various known causes. He initially presented with a left sided Bell's palsy 12 months back, within a month there was facial palsy on the contra lateral side, but patient has not taken any measures to seek any medical attention initially. Later on 4 months back, he developed episodic headache and reeling sensation. His physical examination revealed bilateral lower motor neuron facial palsy with high blood pressure. As the investigations revealed no cause, we have arrived at the diagnosis of Bilateral Bell's palsy and treated his hypertension.

Keywords: Bell's palsy, Guillain Barre syndrome, Sarcoidosis, Amyloidosis

INTRODUCTION

Bilateral simultaneous facial paralysis is a rare clinical entity, which unlike the unilateral presentation, is seldom secondary to Bell's palsy. Adour found only 3 bilateral cases in a consecutive series of 1000 patients with Bell's palsy.[1] Simultaneous onset is defined as the involvement of the opposite side within 30 days of the onset of the first side.

CASE REPORT

A 70 year old patient came with symptoms of unable to close the eyes, drooling of saliva from the angles of the mouth since 12 months involving the right half of face initially, subsequently left half over one month. Subsequently he developed episodic headache and reeling sensation 4 months back. There was no history of head injury, ear discharge, joint pains, febrile illness or hypo pigmented patches. On examination, the patient was conscious, coherent. His temperature was 98.6°F, pulse 70 bpm, respiratory rate 20/min, and blood pressure 200/110 mmHg. The head, eyes, ears, nose, and throat were all normal. The Trachea was in midline, no masses in the neck.

There was no thyromegaly, parotid gland enlargement, adenopathy. Auscultation of the heart and lungs was normal. The abdomen was soft and nontender, no organomegaly, bowel sounds were normal.

Neurological examination revealed a bilateral peripheral facial paralysis with a Grade V (House-Brackmann grading system) weakness on both the sides of face. His taste sensation was distorted and there was evidence of Bell's phenomenon, loss of nasolabial folds, loss of forehead wrinkles, transverse smile, sagging of corners of mouth (Figure 1 & 2) suggesting bilateral LMN Facial palsy.

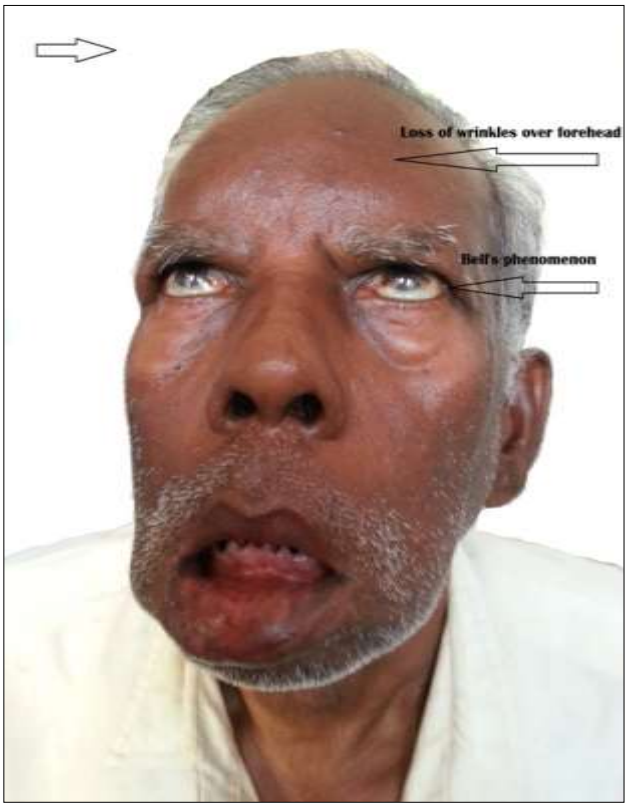

Figure 1: Neurological examination - loss of forehead wrinkles, evidence of Bell's phenomenon.

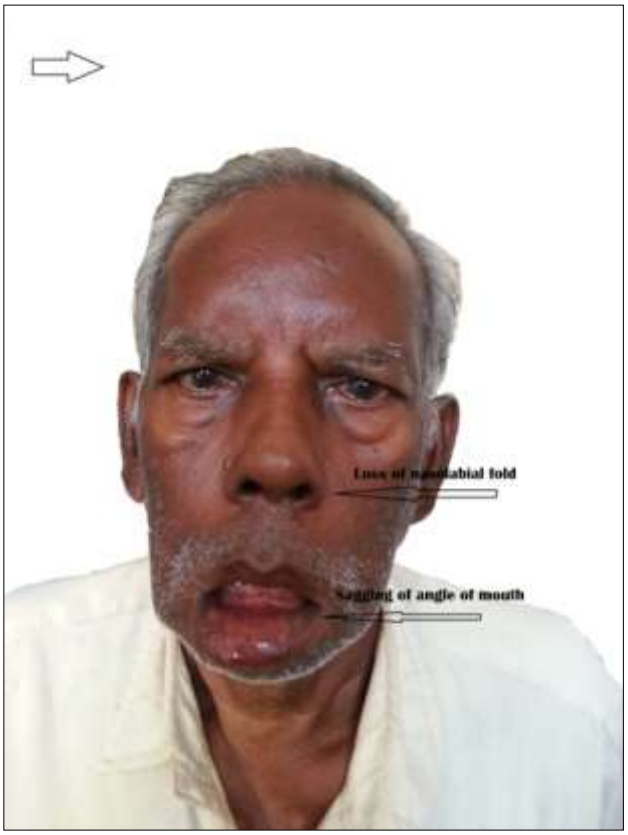

Figure 1: Neurological examination - loss of nasolabial folds, sagging of corners of mouth.

His blood counts Hb% 12 gm, WBC - 7400 /cu.mm, ESR 12 mm/1 hour, Sugars 99 mg%, urea 27 mg%, creatinine 1.2 mg%, sodium 141 meq/l, potassium 3.9 meq/l, CSF analysis cells 4/cu.mm, all lymphocytes, proteins 16 mg%, glucose 28 mg%, ADA 4U/L. Serological testing for HIV, CMV, EBV, HSV, Syphilis and Borrelia were negative. X-ray chest PA view and CT brain were normal.

The causes of bilateral facial palsy include Lyme disease, Guillan-Barre syndrome, sarcoidosis, amyloidosis, diabetes, leprosy, infectious mononucleosis, HIV, parotid surgery, mastoid surgery, acute porphyria, acute leukemia, Parkinson's disease, idiopathic Bell's palsy and represent <2% of all facial palsy cases.[2,3] Bilateral Bell's palsy was exclusion diagnosis in this patient. Patient was given 1 month of prednisolone but he has not shown any facial recovery as he has delayed in seeking medical intervention. His hypertension was controlled with olmesartan. Patient was stable and his Blood pressure was controlled with no symptoms of reeling.

DISCUSSION

Patient presenting with bilateral facial paralysis with analytical history help us in arriving at diagnosis. The history should include time sequence of onset, prior history of facial paralysis, recent viral or upper respiratory tract infection, otological symptoms, change in taste, facial numbness, vesicles, or recent immunization.

The causes of facial paralysis include many conditions such as congenital, traumatic, infectious, neurological, metabolic, neoplastic, toxic, vascular, and idiopathic. Unlike unilateral facial paralysis, where the cause is mostly idiopathic (over 50%), bilateral facial palsy is less often idiopathic (under 20%). In a review of reported cases over a period of 10 years, Teller and Murphy[4] show that Lyme disease is responsible for 36% of the cases for facial diplegia. Guillain-Barre syndrome (5%), trauma (4%), sarcoidosis (0.9%), and AIDS (0.9%) are other causes.

The most common infectious cause of facial diplegia is Lyme disease, caused by Borrelia burgdorferi.[5] It commonly begins in the summer with a skin lesion, erythema migrans. The diagnosis is made by an immunologic assay using antibody titers against the spirochete. Treatment with an antibiotic should be started immediately and not delayed until there is serological confirmation.

The first priority in the evaluation is to rule out a life-threatening disease such as leukemia or Guillain-Barre syndrome. If these are suspected, the patient should be admitted to the hospital for close observation. The

physical examination should be complete with emphasis on the neurological and head and neck portions of the Patient. Workup should include complete blood count, fluorescent treponemal antibody test, HIV test, fasting glucose, erythrocyte sedimentation rate, Lyme titer, and antinuclear antibody level measurement. Lumbar puncture after a CT scan and also special facial nerve function tests could be performed. Magnetic resonance imaging is useful in the demonstration of seventh cranial nerve lesions, tumor cell infiltration and widening of the internal acoustic canal. Also, the areas that are most important to visualize are the central nervous system, skull base, meninges, and cerebellopontine angle, which are best imaged by enhanced MRI.

Guillain-Barre syndrome or Ascending Inflammatory Demyelinating Polyneuropathy (AIDP) presents as a progressive development of palsy of the voluntary muscles of the legs, arms, trunk, and face. The most commonly affected cranial nerves are IX, X, and VII. In 27% to 50% of the cases, the facial nerve is involved. Fifty percent of the patients with a facial paralysis have bilateral involvement. Prognosis is rather good and therapy consists of plasma exchanges and administration of IV Immunoglobulins within 10 days of the onset of symptoms.

Involvement of the facial nerve is a relatively common neurological finding in sarcoidosis. However, bilateral involvement is very uncommon and is even more unusual as the presenting complaint. Meningitis, myelopathy, optic neuropathy, cerebral mass lesions, and polyneuropathy can also be manifestations of neurosarcoidosis[6]. Diagnosis is made by blood analysis, biopsy of the affected organ, and enlargement of lymph nodes on chest CT. Therapy for facial paralysis secondary to sarcoidosis consists of administration of corticosteroids[7].

The prognosis for bilateral facial palsy is dependent on the underlying etiology. If the etiology can be identified and successfully managed, the prognosis is excellent. The prognosis may be worse with age over 60 years, diabetes mellitus, hypertension, pain, decreased tearing, and the degree of denervation demonstrated on electrical testing.

In spite of performing all the tests, in our case we could not establish the cause for bilateral facial palsy, hence it is marked as bilateral Bell's palsy. His Blood pressure got controlled with antihypertensives and physiotherapy advised.

CONCLUSION

Unilateral peripheral facial palsies are usually idiopathic (presumably virally related), whereas bilateral palsies usually reflect an underlying systemic pathology. Patients with bilateral facial palsies need thorough assessment and follow-up.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Srinivas V, Radha Srinivas V, Vikram Vardhan R. Bilateral facial palsy with hypertension: a case report. Int J Sci Rep 2015;1(2):136-8.