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CASE REPORT

Bilateral Facial Palsy as a Presenting Feature of HIV – A Case Report

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Abstract

Bilateral facial palsy (BFP) is a rare entity with some documented evidence as an uncommon manifestation of meningitis due to Kawasaki disease, Lyme's disease, viral, and bacterial etiology. We present a case of a young male with no comorbidities who presented with fever, headache, and gradually progressive weakness of all four limbs for 1 week, with BFP.

Key Words

Bilateral facial palsy, HIV infection, Meningitis

Introduction

Bilateral facial palsy (BFP) is a rare entity that makes up only 0.3 to 2% of all cases of facial palsy.[1] In comparison to unilateral facial palsy, most cases of which are attributed to Bell's Palsy, BFP has been observed to be associated with underlying systemic disease and hence requires urgent intervention.[2] Although other cranial nerve palsies such as oculomotor, abducens, and hypoglossal nerve palsies, have been described in association with meningitis, the literature on facial nerve palsy associated with HIV infection is scarce.[3-5] We present the case of a 29-year-old male with newly detected retroviral disease and acute lymphocytic meningitis complicated by BFP.

Case Report

A 29-year-old male, with no comorbidities, presented to the outpatient department of our tertiary care hospital with a 1 week history of low-grade fever, relieved on taking oral over-the-counter medication, with no associated chills or rigors. The patient also complained of an intermittent headache usually following a fever episode, holocranial in location, dull aching type. Furthermore, the patient described a history of gradually progressive weakness of all four limbs, greater in the left upper and lower limbs, with no aggravating or relieving factors.

The patient also complained of facial weakness for the past five days with an inability to consume solids and liquids, associated with regurgitation at the angles of the mouth. He had also developed difficulty in speech, where he took more time than usual to complete sentences. The patient also described a 1 day history of giddiness, sudden onset, and non-progressive, which aggravated on sitting up from a supine position. Each episode lasted a few seconds with no associated tinnitus. He also had an episode of vomiting, associated with nausea, consisting of food particles, not bloodor bile-tinged, with no aggravating or relieving factors.

The patient denied a history of loose stools, abdominal pain, difficulty in eating, involuntary limb movements, tongue bite, gait changes, loss of sensation of taste or

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Figure 1: Bilateral Bell's phenomenon on examination.

smell, double vision, facial asymmetry, loss of sensation over the face, or hearing disturbances. bowel and bladder dysfunction, fall, or trauma. The patient had no significant past medical or family history.

On an initial examination, the patient appeared drowsy but was arousable. His vital signs were within normal limits. On nervous system examination, neck rigidity was present. The patient moved all four limbs with full power, and the superficial and deep reflexes were intact. On facial nerve examination, findings were significant for bilateral lower motor neuron lesions. The patient had Bell's phenomenon bilaterally (Figure 1). Cardiovascular, respiratory, and abdominal examination were within normal limits.

On admission, routine laboratory investigations were within normal limits. Retroviral test by chemiluminescence immunoassay (CLIA) was positive, which was confirmed by a repeat sample. A lumbar puncture was performed, and an opening pressure of 4 cm of water was observed. Cerebrospinal fluid (CSF) analysis showed a protein level of 225 mg/dl, glucose level of 42 mg/dl, with a corresponding random blood sugar level of 95 mg/dl, 270 cells with 100 % lymphocytes without any atypical cells. A CSF Genexpert, India ink, and Gram stain were negative, and culture and sensitivity showed no growth after 48 hrs. An MRI brain with contrast showed no significant abnormalities.

The patient was started on empirical anti-tubercular therapy and corticosteroids. Prednisone was administered at a daily dose of 40 milligrams for a week, with tapering over the next week. During the course of hospital stay, the patient improved, with a reduction in headache but persistent facial weakness. The patient was discharged with the advice to continue follow-up with us every two weeks and stay adherent to anti-tubercular therapy.

Discussion

Bilateral facial palsy (BFP) is a rare condition with etiologies reported to be Lyme's disease, Guillain-Barré syndrome, Bell's palsy, sarcoidosis, viral infections, and trauma, among others.[6] Its association as a presenting feature of HIV has been described in limited literature.[3-5] However, the association of palsies of other cranial nerves, such as abducens, hypoglossal, and oculomotor nerves, has been described.[7-9] HIV infection has been described to be associated with both unilateral and BFP.[10] In most cases, and in our case, BFP presented at the same time as disclosure of positive HIV serology.[10] Hence, excluding HIV early in BFP patients is essential to prevent HIV related complications.[10]

In our patient, the CSF analysis effectively ruled out a bacterial cause due to its lymphocytic nature and normal fluid glucose, and the possible etiology included viral or tubercular causes. Retroviral test by CLIA was positive, which was further confirmed by a repeat sample. The patient was treated empirically with anti-tubercular therapy. Corticosteroids were given for bilateral facial palsy, which led to significant symptomatic improvement.

A previous case reported from Japan described full resolution of bilateral oculomotor palsy with a three-week course of corticosteroids and immunoglobulins in addition to antibiotics for bacterial meningitis.[8] A European case of bilateral abducens palsy reported a complete resolution with prednisone administration for two weeks.[7]

Based on the above evidence, corticosteroids seem to have a beneficial effect on cranial nerve palsies. A study has shown that corticosteroids have a beneficial effect on the outcome of meningitis, though no significant difference was noted in the neurological sequelae.[11] The reason for the beneficial effect is controversial, and more research needs to be done in this regard.

Conclusions

Our case report describes a young male presenting with acute lymphocytic meningitis complicated by BFP. He was managed with corticosteroids, empiric anti-tubercular therapy, and antibiotics. Regular follow-up and adherence to anti-tubercular therapy were recommended. Owing to the rarity of this association, there is a need for clinicians to be alert about the presentation and begin immediate management with corticosteroids.

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