Bilateral facial nerve palsy: A rare association with hepatitis A

Sir,

A 17-year-old female presented with a history of sudden onset of inability to close eyes, epiphora as well as inability to drink fluids from cup associated with drooling of fluids along the corners of the mouth bilaterally of 2 days duration. There was no associated history of taste disturbances or hearing abnormalities in either of the ears. No other associated cranial nerve disturbances or long tract symptoms were present. No associated fever, cough, headache, vomiting or other relevant systemic symptoms. Examination was unremarkable except for bilateral lower motor neuron type of facial nerve palsies with sparing of taste sensation and without hyperacusis. A clinical diagnosis of bilateral lower motor neuron facial palsy was made with the possible anatomical site distal to the origin of nerve to stapedius muscle. On detailed assessment, she revealed to have been convalescing from an episode of jaundice, which she had acquired 1 month prior to the onset of the present focal deficit. Work-up was carried out to find out the etiology of bilateral facial palsy. Hemogram parameters were within normal limits including erythrocyte sedimentation rate. (hemoglobin –11.9 g/dl [10-12], total leucocyte count –4580 cells/cumm [4000-11,000], differential count [P –62%, L –30%, M –2%, E –6%] platelet count - 1,89,000 [1,50,000-4,00,000]). Liver function parameters had normalized (S. bilirubin - 0.4 mg/dl [0-1.3], conjugated bilirubin - 0.2 mg/dl, serum glutamic axaloacetic transaminas –16 U/L [0-36], serum glutamic pyruvic transaminas –31 U/L [0-52], alkaline phosphatase –59 U/L [38-126], total protein –7.8 g/dl [6.0-8.2], S. albumin –4.5 g/dl [3.5-5.0]). Metabolic parameters were normal: S. calcium –9.1 mg/dl (8.4-10.2), S. phosphorous –3.5 mg/dl (2.5-4.5) and S. creatinine –0.8 mg/dl (0.7-1.2). Viral markers were assessed: Human immunodeficiency virus - *non*-reactive by enzyme-linked immunosorbent assay (ELISA), herpes simplex 1, 2 immunoglobulin (Ig)M and IgG antibody (ELISA): Negative, hepatitis B surface antigen (ELISA)-negative, anti-hepatitis C virus-negative, anti-hepatitis E virus IgM (ELISA)-negative, anti-hepatitis A virus (HAV) IgM (ELISA)-positive; 2.66 EU/ml (cut-off value: 0.600 EU/ml). Serum angiotensin converting enzyme levels were 102.0 IU/L (8-65). Nerve conduction studies revealed bilateral facial nerve axonopathy only. Neuroimaging study [gadolinium contrast enhanced magnetic resonance imaging brain with constructive interference in steady state (CISS) sequences] did not reveal any structural pathology. High resolution computerised tomography scan of the chest did not reveal any structural pathology. Mantoux test was borderline positive (8 mm induration at 72 h). Cerebrospinal fluid evaluation was not performed in view of lack of any clue towards meningeal based pathology. As the dedicated work up for etiology of bilateral lower motor neuron type facial palsy was negative, we postulated it to be probably an immune phenomenon as a consequence to the recent HAV infection from, which she was convalescing. With conservative neurorehabilitative measures and facial nerve stimulation procedures our patient had significant improvement in her neurological status over duration of 8 weeks.

Only a handful of reports are present in the world literature stating the association of hepatitis A with Bell's palsy, with majority of them being unilateral.[1-3] A recent review on the prevalence of serological markers of hepatitis, cytomegalovirus and rubella in patients with Bell's palsy revealed a serological positivity for hepatitis B in 15 out of 21 patients (71%) as against 32.1% positivity in the control group. No relation was noted in the above study with hepatitis A serological positivity.[4] Neurological complications during the acute and convalescent phase of acute HAV infection have been reported in the form of aseptic meningitis, cranial neuropathies (trigeminal, facial, vestibulocochlear) and visual disturbances. However, bilateral facial nerve palsies associated with recent acute HAV infection has not been reported so far. The purpose of this letter is to make health care professionals more aware of facial palsy complication of hepatitis A infection. Even though a rare association, the common viral tests for facial palsy could include hepatitis viruses especially, if there is past history suggestive of acute hepatitis.

Sudhir Sharma, Praveen Kesav[1]

Departments of Neurology, Indira Gandhi Medical College, Shimla, Himachal Pradesh, [1]Postgraduate Institute of Medical Education & Research, Chandigarh, India

For correspondence:
Dr. Sudhir Sharma, Department of Neurology, Indira Gandhi Medical College, Shimla - 171 001, Himachal Pradesh, India
E-mail: sharmasudhir21@gmail.com

References

1. Swaroop A, Parihar N, Jain S. Bell's palsy — A rare association with hepatitis A. J Indian Acad Clin Med 2008;9:51-2.
2. Thapa R, Mallick D, Biswas B, Ghosh A. Childhood hepatitis A virus infection complicated by Bell's palsy. Clin Pediatr (Phila) 2009;48:427-8.
3. Yildiz B, Yakut A, Bor O, Yarar C. Bell's palsy and hepatitis infection. Pediatr Int 2006;48:493-4.
4. Unlu Z, Aslan A, Ozbakkaloglu B, Tunger O, Surucuoglu S. Serological examinations of hepatitis, cytomegalovirus and rubella in patients with Bell's palsy. Am J Phys Med Rehabil 2003;82: 28-32.

Access this article online

Quick Response Code:

Website: www.annalsofian.org

DOI: 10.4103/0972-2327.120432