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Original Research Paper

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UNILATERAL FACIAL NERVE PALSY: A RARE ASSOCIATION WITH HEPATITIS A

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ABSTRACT Bell's Palsy is the sudden onset of unilateral paralysis of facial muscles resulting from seventh cranial nerve dysfunction. Neurological complications associated with HAV have been reported in the form of aseptic meningitis, Guillain- Barre syndrome, cranial neuropathies (trigeminal, facial, vestibulocochlear), Myasthenia Gravis, myopathy and visual disturbances. The aim of the present study is to report an 11 year old boy with HAV infection, who in due course of his infection, developed right-sided lower-motor neuron (LMN) type facial nerve palsy.

KEYWORDS :

INTRODUCTION

Bell's palsy is a neuropathy of the peripheral seventh cranial nerve, usually resulting from traumatic, compressive, infective, inflammatory or metabolic abnormalities. The condition is named after Dr Charles Bell, in 1821.1 Bell's Palsy has a frequency of 20/100,000 individuals per year, and a recurrence rate of 9% of cases.2 However, in many cases no aetiology is identified and the eventual diagnosis is idiopathic. We would like to draw the reader's attention to a possible cause of Bell's Palsy as a rare complication of hepatitis A infection.

Case report

An 11-year-old male presented with a history of weakness over right side of face which was sudden in onset and gradually progressive. There was history of inability to close eyes, epiphora and deviation of face to right side of 20 days duration. There was no associated history of taste disturbances or hearing abnormalities in either of the ears, earache, ear discharge or noise intolerance. He had no history of fever, rash, cough, headache, vomiting and weight loss. Examination was unremarkable except for unilateral lower motor neuron type of facial nerve palsies with sparing of taste sensation and without hyperacusis. A clinical diagnosis of unilateral lower motor neuron facial palsy was made. On detailed assessment, he revealed to have been convalescing from an episode of jaundice, which he had acquired 10 days prior to the onset of the present focal deficit. Investigations were done to find out the etiology of unilateral facial palsy. Liver function parameters had normalized (S. bilirubin – 1.6 mg/dl [0-1.3], conjugated bilirubin - 0.6 mg/dl, serum glutamic oxaloacetic transaminase –40 U/L [0-36], serum glutamic pyruvic transaminase –39 U/L [0-52], alkaline phosphatase –230 U/L [38-126], total protein –7.6 g/dl [6.0-8.2], S. albumin –4.4 g/dl [3.5-5.0]). Haematological and other Metabolic parameters were normal. Viral markers were assessed: 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; 4.11 (cut-off value: 0.800 EU/ml). Hence, we postulated it to be probably an immune phenomenon as a consequence to the recent HAV infection from, which he was convalescing. With conservative neuro-rehabilitative measures and facial nerve stimulation procedures our patient had significant improvement in his neurological status.

DISCUSSION

Neurologic manifestations resulting from infection with the hepatitis viruses are relatively rare. The peripheral and central nervous systems (CNS) can be involved either in isolation or in combination. The various neurologic syndromes which have been reported in patients with serologically defined viral hepatitis include Guillain–Barré syndrome, mononeuritis multiplex, sensorimotor polyneuropathy, cranial nerve palsy, encephalitis, meningitis, encephalomyelitis, transverse myelopathy, strokes, auditory neuritis, and cognitive impairment. The first case of infectious hepatitis with neurological complications was reported by Lischer as early as 19443 followed by Byrner and Taylor in 1945, which described 5 cases of jaundice with neurological signs.4 Rao et al reported myelitis and neuritis associated with infectious hepatitis in Delhi in 1968.5 Cranial nerve abnormalities that have been associated with acute viral hepatitis include sensory (trigeminal), motor (seventh and ninth).

Bell's palsy is an idiopathic, unilateral, peripheral facial paralysis of acute onset. It is characterised by the patient being unable to close an eye, dribbling of saliva from angle of mouth, facial asymmetry, inability in frowning and raising the eyebrows, epiphora, hyperacusis, and loss of taste. Aetiologically, it is idiopathic; however, various causes such as congenital (traumatic, syndromic, genetic) or acquired (infectious, inflammatory, neoplastic, traumatic) have been associated. The most common aetiological agent is Herpes simplex virus. Hepatitis A virus can cause many neurological complications; facial nerve involvement has been reported but not well associated. Our case also presented with unilateral LMN type facial paralysis of sudden onset with all the clinical features except hyperacusis and loss of taste sensation, with clinical features of hepatitis and serological positivity for HAV total. The patient was managed conservatively, and complete recovery of hepatitis (normal S. bilirubin, ALT, AST) was recorded. Treatment of Bell's palsy includes the use of steroids and antivirals, ideally within 72 hours but up to 7 days from the onset of symptoms.6 In view of the possible exacerbation of viral hepatitis, steroid was not given to our patient.

CONCLUSION

Only a handful of reports are present in the literature stating the association of hepatitis A with Bell's palsy, out of which majority are unilateral. The purpose of this case report is to make clinicians more aware of facial palsy complication of hepatitis A infection. It is to increase awareness of health care professionals that Hepatitis A should be included in differential diagnosis of unexplained cases of Unilateral facial palsy.

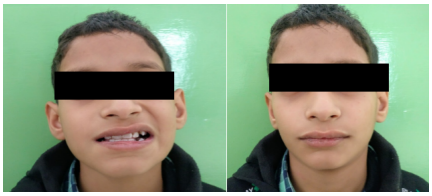

Figure 1: Photograph showing right-sided LMN-type facial paralysis (Bell's Palsy)

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