Case report

Two different forms of mumps-associated facial palsy

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Abstract

Peripheral facial nerve paralysis is relatively common in the pediatric age group. Infectious agents convincingly associated with acute facial palsy include varicella-zoster virus, herpes simplex virus, cytomegalovirus, Epstein–Barr virus, rubella virus, and more recently, human immunodeficiency virus. However, facial palsy caused by mumps infection is believed to exist in two distinct forms, one representing an initial infection with mumps parotitis and another representing a reinfection without parotitis. We recently saw the two different forms of mumps-associated facial palsy with and without parotitis.

Keywords:
Epidemic parotitis
Facial palsy
Mumps

1. Introduction

Mumps virus infections primarily involve the parotid glands and most frequently affect young children during the winter and spring months. The diagnosis is readily apparent from clinical inspection and palpation. Laboratory investigations are not routinely performed in the outpatient setting, and treatment is entirely symptomatic. Orchitis, oophoritis, mastitis, meningoencephalitis, deafness, nephritis, and arthritis may occur in addition to parotitis. However, facial palsy has rarely been documented in patients with mumps virus infection. Facial palsy caused by mumps infection is believed to exist in two distinct forms, one representing an initial infection with mumps parotitis and another representing a reinfection without parotitis. We recently saw the two different forms of mumps-associated facial palsy with and without parotitis.

2. Case presentation

2.1. Case 1

A 5-year-old Japanese girl was admitted to our hospital with a suspected diagnosis of right facial palsy. It had been noted that her right eye was filmed over with tears and the corner of the mouth on the right side was drooping from 2 days before admission. The patient, who had not been vaccinated against mumps, had had swelling in both parotid glands at the age of 4 years. Mumps parotitis was suspected, but her serum mumps titer was not checked. On admission, the patient was unable to move the forehead upward, close the eye forcefully, or elevate the corner of the mouth on the right side. She was diagnosed with complete right facial palsy of the lower motor neuron type. No other neurological abnormalities were present. No clinical signs of meningitis or encephalitis were present, and a lumbar puncture was not performed.

2.1.1. Laboratory data

Examination of general and biochemical laboratory data showed no abnormalities. We measured the virus antibody levels (IgG, IgM) for mumps, varicella-zoster, and herpes simplex using the enzyme-linked immunosorbent assay (EIA) method. The diagnosis of mumps was proven by detection of serum IgM anti-mumps antibody.

The patient was given mecobalamin from the first day of admission. Two days later, oral prednisolone was prescribed. The symptoms began to improve from the early stage (about the 9th day of illness), and the facial palsy resolved completely by 3 weeks after onset. MRI revealed no abnormal findings. The patient had no parotid gland swelling during her clinical course.

2.2. Case 2

An 8-year-old Japanese boy visited the local pediatric clinic because of pain and swelling in the right parotid gland. The patient
had not been vaccinated against mumps and had no previous mumps infection. A diagnosis of mumps was made, and the initial clinical course was unremarkable. Two days after the swelling in the right parotid gland developed, paralysis of the right lower lip and the angle of the mouth developed, and the patient could not tightly close the right eye. He was diagnosed with right peripheral facial nerve paralysis. No other neurological abnormalities were present, and lumbar puncture was not performed. The diagnosis of mumps was proven by the detection of serum IgM anti-mumps antibody by EIA. Oral prednisolone and intravenous mecobalamin and ATP were started at that time. The facial palsy resolved completely by 12 weeks after onset.

3. Discussion

Peripheral facial nerve paralysis is relatively common in the pediatric age group. Eshel et al. [4] described the pediatric patient with acquired peripheral facial nerve paralysis was an incidence of 22/10,000 pediatric hospitalization. Peripheral seventh cranial nerve palsy, the most frequent cranial neuropathy, may be caused by traumatic, compressive, infective, inflammatory, and metabolic abnormalities. However, the majority of causes has no identified etiology and is eventually diagnosed as idiopathic peripheral facial palsy (IPFP) or Bell’s palsy. Infectious agents convincingly associated with IPFP include varicella-zoster virus, herpes simplex virus, cytomegalovirus, Epstein–Barr virus, rubella virus, and, more recently, human immunodeficiency virus [5].

Since Saunders and Lippy [6] first described four patients with facial palsy associated with mumps virus infection in 1959, several reports have suggested an association between mumps virus infection and facial palsy [1–8]. However, in most of these cases, either mumps was diagnosed serologically in the absence of clinical signs of parotitis or no mention was made of parotid findings, as in our case [4–8]. Njoo et al. [8] described 63 patients with facial palsy clinically attributed to varicella zoster virus infection; 11 of these patients had no serologic evidence of varicella zoster virus involvement. Of the 11 patients, 7 had an IgG or IgM antibody associated with the mumps virus that was detectable by EIA [8]. Since all seven patients with positive mumps serology were older women with IgG antibody to mumps virus in the first serum samples tested, the author questioned whether this finding constituted evidence for mumps reinfection – a suggestion based on previous epidemiological evidence [9] – and concluded that infection with mumps virus can cause facial palsy. Although the pathogenesis of IPFP has not been entirely clarified, it is considered that it is caused by the inflammation triggered by a virus. It is known that there are situations in which problems with the detection of IgM based on serological methods could be experienced because IgM can disappear earlier than expected or remain for months, even years, at low titers. Therefore, serologically determined IgM could be insufficient for the differentiation of primary infections, reinfections, and reactivation of infections [10].

The incidence of facial palsy in conjunction with mumps parotitis is unknown, but the association appears to be rare. Additionally, the clinical picture has rarely been documented. We found only three detailed case reports of facial palsy associated with mumps parotitis in the literature that were similar to our case [1–3]: five patients (three pediatric patients and two adults) were described in these accounts. Facial palsy developed 3–9 days after onset of mumps parotitis in these patients [1–3]. One of these individuals also had an abducens palsy, vertigo, and partial hearing loss [1,2]. Two patients recovered completely from the facial palsy [1–3].

Facial palsy caused by mumps infection is believed to exist in two distinct forms, one representing an initial infection with mumps parotitis and another representing a reinfection or reactivation of infection without parotitis. A study that would be sufficiently large to permit statistical analysis will be necessary to elucidate the epidemiology of these two forms of mumps-associated facial palsy.

References