CASE STUDY

Bell's Palsy as a Possible Complication of Hepatitis B Vaccination in A Child

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ABSTRACT

Bell's Palsy is the sudden onset of unilateral temporary paralysis of facial muscles resulting from seventh cranial nerve dysfunction. Presented here is a two-year old female patient with right peripheral facial palsy following hepatitis B vaccination. Readers' attention is drawn to an uncommon cause of Bell's Palsy, as a rare complication of hepatitis B vaccination.

Key words: Bell's Palsy; Case Study; Hepatitis B; Hepatitis B vaccine; Vaccination; Turkey

INTRODUCTION

Bell's Palsy is the sudden onset of unilateral temporary paralysis of facial muscles, resulting from seventh cranial nerve dysfunction. Bell's Palsy has a frequency of 20/100,000 individuals per year, and a recurrence rate of 9% of cases. Although Bell's Palsy is a well-known and relatively-common disease, its aetiology is still unclear (1-3). Following the introduction of hepatitis B vaccination, rare serious reactions, such as demyelinating myelitis, demyelinations of the peripheral and central-nervous system, vasculitis, auto-immune diseases, Bell's Palsy, and opthalmological abnormalities, have been described (4-12). We would like to draw the reader's attention to a possible cause of Bell's Palsy as a rare complication of hepatitis B vaccination.

Case report

On 10 May 2004, a two-year old female patient was admitted to the paediatric department, Medical Faculty, Ataturk University, because of a right peripheral facial palsy. On admission, the child looked well, and she had no fever. There was no history of an upper respiratory tract infection. Car-

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diovascular, respiratory, abdominal and otoscopic examinations were all normal. She had Bell's sign on the right side of her face. There was no involvement of other cranial nerves, and the remainder of neurologic examination was normal.

Haematological and urine analyses were normal. Magnetic resonance imaging of brain demonstrated no abnormality, and blood serology was negative for Epstein-Barr virus, cytomegalovirus, herpes simplex virus, rubella, toxoplasma, and *Mycoplasma pneumoniae*. Six days before admission, she had been given recombinant hepatitis B vaccine. Bell's Palsy was thought most probably to be a sequel of vaccination against hepatitis B. No treatment was given, and her symptoms improved spontaneously. The patient began to improve on the 22nd day after the onset of symptoms, and 90 days after presentation, Bell's Palsy had completely resolved.

DISCUSSION

Over the past 30 years, representatives of the medical establishment have discussed and warned about neurologic complications of various vaccines (13-16). Vaccination against hepatitis B virus (HBV) is important to reduce the incidence of HBV-associated infection. Although HBV vaccine is among the safest of all vaccines, it has been associated with adverse effects (8-12,17). Shaw et al. reported a postmarketing surveillance study, recording neurologic events after hepatitis B vaccination (11). An estimated 850,000 individuals had received the vaccine over the time of their study. They found three cases of brachial plexus neuropathy, four cases of transverse myelitis, five cases of optic neuritis, five cases of lumbar radiculopathy, nine cases of Gullian-Barre Syndrome, and 10 cases of Bell's Palsy.

Seven hundred reports of adverse reactions to the hepatitis B vaccine were sent into the Vaccine Adverse Events Reporting Systems (VAVERS). Sixteen percent of these reports were of damage presumed to be to the myelin of the nervous system. There were 21 cases of facial paralysis (17).

The aetiology and pathogenesis of Bell's Palsy remains unclear. There is a concern that reactivation of latent herpes simplex virus-associated infections of the geniculate ganglia of facial nerves may be one of the causes of Bell's Palsy. Auto-immune processes have also been considered (18-19). It has been hypothesized that an immunomediated segmental demyelination may be involved. Also, it is known that hepatitis vaccine is associated with Gullian-Barre Syndrome and demyelinating disease, possibly through an immune response mechanism (9-12,17). Therefore, it is at least theoretically possible that hepatitis B vaccines may trigger Bell's Palsy through a smilar mechanism, although there is no current evidence to support this theory. We did not find any of the classical aetiologies of Bell's Palsy in our patient. The sole cause suspected was the vaccination against hepatitis B six days before the diagnosis of this disease.

The prognosis in this disorder is excellent. More than 85% of cases recover spontaneously with no residual facial weakness as in our case; only 5% are left with permanent severe facial weakness (1,2). Further studies are needed to confirm whether hepatitis B vaccines are associated with an increased risk of Bell's Palsy and, if so, to investigate details of the pathogenesis. Also, we are conducting a population-based controlled study to determine whether this association is causal or co-incidental.

We conclude that Bell's Palsy must be considered in all patients with this disease, following hepatitis B vaccination but the present universal vaccination programme to eradicate hepatitis B should not be changed.

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