Transient facial nerve paralysis (Bell's palsy) following administration of hepatitis B recombinant vaccine: a case report

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IN BRIEF

- Highlights an unusual complication of hepatitis B vaccine and discusses its causes.
- This is imperative to the differential diagnosis of Bell's palsy, a common presentation in oral surgery departments.

PRACTICE

Bell's palsy is the sudden onset of unilateral transient paralysis of facial muscles resulting from dysfunction of the seventh cranial nerve. Presented here is a 26-year-old female patient with right lower motor neurone facial palsy following hepatitis B vaccination. Readers' attention is drawn to an uncommon cause of Bell's palsy, as a possible rare complication of hepatitis B vaccination, and steps taken to manage such a presentation.

INTRODUCTION

A 26-year-old dental nurse presented to our outpatient department approximately six hours following administration of a hepatitis B vaccine with symptoms of right-sided facial weakness. There were no symptoms of being unwell in her presenting complaint, no described localised erythema, swelling or pain at the vaccination inoculating site (left deltoid region). There was no history of recent travel or symptoms of upper or lower respiratory tract infection. Her medical history was unremarkable, she was generally fit and healthy, she was on no regular medications and did not report any allergies, and she was in no distress.

On examination, her blood pressure was 128/76 mmHg, her respiratory rate was 16, pulse 72 per minute and her temperature was 36.8°C. There was no facial or head asymmetry, the parotid glands were not enlarged and there was no facial or cervical lymphadenopathy palpable. There was no rash or ear canal vesicles and no tinnitus (ringing sensation in the ear); there were no symptoms of dizziness so Ramsay Hunt Syndrome (this is seen in shingles, caused by the Varicella zoster virus, with symptoms of pain, vesicular rash in the ear canal, dizziness, tinnitus) was ruled out. There was loss of wrinkling on the right side of the forehead, weakness of the right side of the face, and inability to close the eye completely. The patient also reported loss of taste on the tongue.

The inoculation site (left deltoid) did not exhibit any tenderness, erythema or signs of infection. Her chest X-ray and routine haematinic investigations were normal (a full blood count, urea and electrolytes, liver function tests). The serum ACE (angiotensin converting enzyme) was 12 (normal level is between 9-67 units, though this may vary for different laboratories. The relevance of this test is discussed later). There was no involvement of the other cranial nerves and an otoscopy (examination of the middle ear canal with an otoscope) was unremarkable. Magnetic resonance imaging of the brain did not reveal any intra cranial pathology, and blood serology was negative for diabetes, herpes simplex virus, and sarcoid.

A diagnosis of lower motor neurone facial palsy was made.

Local measures such as lubricating the eye/artificial tears were provided and an eye-patch was provided. An ophthalmological appointment was made to attend the same day, where the patient was reassured following the examination and no review appointment was made. Following two review outpatient appointments at our department, the patient was seen to improve spontaneously with conservative management at the end of day 21 and the Bell's palsy completely resolved by day 30.

DISCUSSION

Bell's palsy is a well known entity and relatively common; however, its aetiology is still unclear. The frequency of Bell's palsy is roughly 20/100,000 individuals per year with an estimated recurrence rate of 9%.[1] Patients presenting with Bell's palsy typically may have decreased forehead movement on the ipsilateral side and inability to close the eye, a disappearance of the nasolabial fold, and possibly a feeling of an altered sensation on the affected side of the face, with drawing of the mouth to the contralateral side.

Some additional symptoms such as hyperacusis, decreased production of tears, and altered taste may also be present. Maillefert *et al.* in 1997 reported a case of mental nerve neuropathy following hepatitis B vaccination.[2] Bell's palsy is typically self-limiting with a favourable prognosis; however, its sudden onset, rapid progression, and dramatic presentation can be alarming, both for the patient and the clinician.

Bell's palsy is a diagnosis of exclusion (Table 1). Known congenital and acquired causes of facial nerve paralysis need to be excluded before the diagnosis of Bell's palsy is made.[3]

Following a post-marketing surveillance study by Shaw *et al.* in 1988 which recorded adverse events following administration of the hepatitis B vaccine in 850,000 people, the researchers 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 Guillain-Barré Syndrome, and ten cases of Bell's palsy.[4]

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Table 1 Differential diagnosis of facial nerve palsy[7–8]

Disease	Cause	Distinguishing factors
Nuclear (peripheral)		
Lyme disease	Spirochete *Borrelia burgdorferi*	History of tick exposure, rash, or arthralgias; exposure to areas where Lyme disease is endemic
Otitis media	Bacterial pathogens	Gradual onset; ear pain, fever, and conductive hearing loss
Ramsey Hunt syndrome	Herpes zoster virus	Pronounced prodrome of pain; vesicular eruption in ear canal or pharynx
Sarcoidosis or Guillain-Barré syndrome	Autoimmune response	More often bilateral
Tumour	Cholesteatoma, parotid gland	Gradual onset
Supranuclear (central)		**Forehead spared**
Multiple sclerosis	Demyelination	Additional neurologic symptoms
Stroke	Ischaemia, haemorrhage	Extremities on affected side often involved
Tumour	Metastases, primary brain	Gradual onset; mental status changes; history of cancer

neuropathy following hepatitis B vaccination.[2] Vaccination against the hepatitis B virus (HBV) is important to reduce the incidence of HBV-associated infection.

Although the HBV vaccine is among the safest of all vaccines, it has been associated with adverse effects. Seven hundred reports of adverse reactions to the hepatitis B vaccine were sent into the Vaccine Adverse Events Reporting Systems (VAERS); 16% of these reports were of damage presumed to be to the myelin of the nervous system. There were 21 reported cases of facial paralysis.[5]

The aetiology and pathogenesis of Bell's palsy remains unclear. It is thought 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. An immunomediated segmental demyelination as a hypothesis has also been proposed. It is known that the hepatitis vaccine is associated with Guillain-Barré Syndrome and demyelination, possibly through an immune response mechanism.[5,6] It may be possible that the HBV vaccine triggers Bell's palsy through a similar mechanism, although there is no current evidence to confirm this.

The first priority in the workup is to rule out Guillain-Barré syndrome, which can be life threatening. If this is suspected, the patient should be admitted to the hospital and monitored closely for any possible airway complications and the anaesthetic team contacted.

Workup should include full blood count, fluorescent treponemal antibody test, HIV serology, fasting blood glucose and erythrocyte sedimentation rate; Lyme polymerase chain reaction (PCR) titres is essential and antinuclear antibody level measurement. Magnetic resonance imaging can identify any seventh cranial nerve lesions, intracranial lesions and widening or narrowing of the internal acoustic canal. Special emphasis should be given to visualising the central nervous system, skull base, meninges, and cerebellopontine angle. A discussion of iatrogenic Bell's palsy, Lyme disease, Guillain-Barré syndrome, sarcoidosis, and reactivation of herpes virus follows.

LYME DISEASE

The most common infectious cause of bilateral facial paralysis is Lyme disease, caused by *Borrelia burgdorferi*, a spirochaete. It commonly begins in the summer with a skin lesion, erythema migrans. The diagnosis is made by an immunologic assay using antibody titres against the spirochaete. Treatment with an antibiotic should be started immediately and not delayed until there is serological confirmation.

The type of therapy is determined in part by the clinical features and stage of the disease.[7]

GUILLAIN-BARRÉ SYNDROME

Guillain-Barré syndrome on the other hand, or ascending inflammatory demyelinating polyneuropathy (AIDP), clinically presents with involvement of voluntary muscles of the upper and lower limbs, trunk, and the face. The most commonly affected cranial nerves are VII, IX, and X. In 27-50% of cases, the facial nerve is involved.[7] Fifty percent of patients with a facial palsy have bilateral involvement. The prognosis is good if caught early. Therapy consists of plasma exchanges and administration of intravenous immunoglobulin within ten days of the onset of symptoms.

SARCOIDOSIS

In sarcoidosis, diagnosis is made by blood analysis (serum angiotensin converting enzyme), biopsy of the affected organ, and enlargement of hilar lymph nodes on chest radiograph or on chest computed tomography (CT). With neurosarcoidosis, the cerebro-spinal fluid (CSF) protein level is usually elevated, whereas the CSF glucose level is usually within the reference range or slightly low. A predominantly lymphocytic cerebrospinal fluid is common.[8,9]

IATROGENIC BELL'S PALSY

A review of the literature describes incidence of iatrogenic/post-operative facial paralysis by many authors. Different aetiologies have been described, such as: local anaesthesia tooth extraction, infections, steotomies, preprosthetic procedures, excision of tumours or cysts, surgery of the temporomandibular joint and surgical treatment of facial fractures and cleft lip/palate.[10] Tympanomastoid surgery has also been implicated in facial palsy, although with the advent of modern microscopes and facial nerve stimulators, the incidence is decreasing.[11] The literature reports three mechanisms in which a dental procedure could damage a nervous structure: direct trauma to nerve from a needle; intraneural haematoma formation or compression; and local anaesthetic toxicity.[12,13]

Several attempts have been made to grade facial palsy but none have been universally accepted. The House-Brackmann Grading System has been recommended as a universal standard for assessing the degree of facial palsy.[14]

Following dental injections, vasospasm of the vessels supplying the facial nerve has been suggested as a mechanism for facial palsy, which may be thought to be medicated via the sympathetic nerve plexus arising from the external carotid arteries. Local anaesthetic with adrenaline as a vasoconstrictor, and possible direct trauma from the needle, could be a potential stimulus for vasospasm.[15]

MANAGEMENT

Facial nerve weakness in a patient may bring about undue anxiety to an alarming degree. It is of paramount importance that the dentist assessing the patient should take all the necessary steps to reduce this fear in patients presenting with facial nerve palsy and introduce measures to treat the same.

It has been suggested that patients who are in severe discomfort, with discharge or redness or with visual problems or are unable to close their eye(s), should be assessed urgently, the same day of presentation, by an ophthalmologist, and may benefit from lubricating eyedrops in the day and ointment during night time. They should be instructed to cover/patch/tape the affected eye at night using plain gauze/micropore dressing. Some patients may need botulinum toxin/tarsorrhaphy.

A large, randomised study has proposed that predisolone (25 mg twice a day) alone for ten days is an effective treatment, along with local measures to assess and protect the eye. In patients with palsy, early treatment with prednisolone significantly improves the chances of complete recovery. There is no evidence of the benefit of acyclovir given alone or an additional benefit of acyclovir in combination with prednisolone.[16]

CONCLUSION

The history should include time sequence of onset, any recent dental treatment under local anaesthesia, prior history of facial paralysis, recent viral or upper respiratory tract infection, recent history of travel, otological symptoms, change in taste, facial numbness, ear canal vesicles, and recent immunisation.

In this patient, the most likely cause for her transient Bell's palsy was thought to be secondary to administration of the HBV recombinant vaccine. This patient was a healthcare assistant, commencing her training following occupational health clearance. It will be interesting to know the variant type of the recombinant HBV used in various sectors of healthcare for vaccinations and the onset of facial palsy correlating to that particular type. This would also show if any one particular variant is implicated in cases of facial palsy, or other complications, compared to the other types in use. Following a literature review, it is not obvious as to the type of vaccines used that have caused reported cases of facial palsy. We suggest conducting a population-based controlled study to determine whether this association is causal or coincidental.

In all healthcare workers (doctors, dentists, nurses etc) commencing their training or course, who present with a Bell's palsy, and have recently been vaccinated, the HBV vaccine should be considered as one of the probable causes of symptoms after all other possible causes have been ruled out and on encountering a distressed patient with facial palsy, attempts should be made to alleviate the patient's anxiety, protect the eye and commence medical management, if appropriate.

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