Immediate Postoperative Bell's Palsy: Viral Etiology or Post-Traumatic Phenomena?

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Original Hypothesis

Immediate Postoperative Bell's Palsy: Viral Etiology or Post-Traumatic Phenomena?

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Abstract

Introduction: Bell's palsy is a sudden unilateral paralysis of the facial nerve. Postoperative Bell's palsy following surgery is rare. It occurs in less than 1% of operations.

The hypothesis: We premise that the main cause of immediate postoperative Bell's palsy is latent herpes viruses (herpes simplex virus type 1 and herpes zoster virus), which are reactivated from cranial nerve ganglia. Inflammation of the nerve initially results in a reversible neurapraxia, but ultimately Wallerian degeneration ensues. The palsy is often sudden in onset and evolves rapidly, with maximal facial weakness developing within two days. Associated symptoms often seen in idiopathic Bell's palsy are tearing problems, hyperacusis and altered taste.

Evaluation of the hypothesis: Facial paralysis presenting postoperatively is distressing and poses a diagnostic challenge. A complete interruption of the facial nerve at the stylomastoid foramen paralyzes all the muscles of facial expression. Taste sensation may be lost unilaterally and hyperacusis may be present. Idiopathic Bell's palsy is due to inflammation of the facial nerve in the facial canal. Bell's palsy may also occur from lesions that invade the temporal bone (carotid body, cholesteatoma, dermoid cyst, acoustic neuromas). Although traumatic Bell's palsy cannot be ruled out, it seems logic to postulate that the main cause of immediate postoperative Bell's palsy is latent herpes viruses.

Key words: Bell's palsy; Herpes virus; Trauma; Facial nerve.

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Introduction

Sudden unilateral paralysis of the facial nerve is known as Bell's palsy (BP) [1]. It is the most common form of facial paralysis. The pathogenesis of the paralysis is unknown and the few autopsied cases of this disease have shown only nondescript changes in the facial nerve and not inflammatory changes as is commonly presumed [1]. Idiopathic BP occurring immediately postoperatively is rare. Although idiopathic BP is largely a diagnosis of exclusion, certain features i.e. abrupt onset with complete, unilateral facial weakness on the affected side within 24 to 48 hours, numbness or pain around the ear, a reduction in taste, and hypersensitivity to sounds help distinguish it from facial paralysis (FP) or BP due to other conditions. The latter three symptoms are not present in traumatic BP following maxillofacial procedures (which can damage only the 5 terminal motor branches of the facial nerve exiting the stylomastoid foramen. Corticosteroids and antivirals given within 10 days of onset have been shown to help treat idiopathic BP. BP may also resolve spontaneously without treatment within 6 months in most patients [2-4]. Surgical damage to the nerve may be permanent.

The hypothesis

We premise that the main cause of immediate postoperative BP is latent herpes viruses (herpes simplex virus type 1 and herpes zoster virus), which are reactivated from cranial nerve ganglia. Inflammation of the nerve initially results in a reversible neurapraxia, but ultimately Wallerian degeneration ensues. The palsy is often sudden in onset and evolves rapidly, with maximal facial weakness developing within two days. Associated symptoms often seen in idiopathic BP are tearing problems, hyperacusis and altered taste [3,4]. The following is an example of such a case.

A 30 year-old man referred for treatment of asymmetry of the facial skeleton that had been present since adolescence. The patient had no systemic diseases. Routine laboratory tests were reported to be within normal limits. Radiographic studies of the face and the chest did not reveal any other abnormalities. The patient was admitted for orthognathic surgery. Mandibular setback was done via a short sagittal split osteotomy procedure (Dal Pont technique) without complications. No transgression of the posterior border of the ramus was done and retractors were not placed behind the ramus either. Rigid fixation was done using miniplates placed on the oblique ridge via an intraoral approach without a trocar. The surgery took 2 hrs. Immediately after surgery in the recovery room (RR), paralysis of the right mandibular nerve became apparent. Paralysis progressed within several hours, accompanied by pain over right mastoid process. All five facial branches of the ipsilateral facial nerve were involved within 24 hrs (Figures 1 and 2). He complained of hyperacusis increased tearing and pain in and around the ear and taste problems. Consultations were done with a neurologist and a neurosurgeon. The patient was treated with

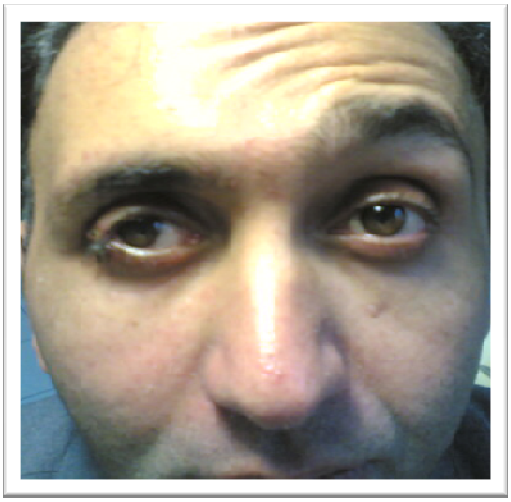

Figure 1. Postperative view of the patient exhibiting inability to raise the right eyebrow.

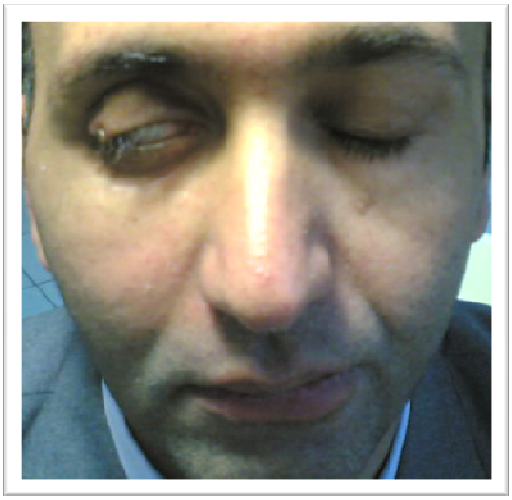

Figure 2. Postoperative view of the patient exhibiting inability to close the right eye and drooping of the ipsilateral oral commissure.

50 mg of prednisone daily for 5 days, and then the dosage of prednisone was reduced by 20 mg per two days for the next 10 days. Electromyography was done 20 days after insult and showed right facial nerve temporary conduction delay with prominent demyelinating pattern. The patient was also given non-invasive electrode-pulse electric stimulation daily for one month. He recovered fully within 4 mos. (Figures 3 and 4).

Evaluation of the hypothesis

FP presenting postoperatively is distressing and poses a diagnostic challenge. A complete interruption of the facial nerve at the stylomastoid foramen paralyzes all the muscles of facial expression. Motor function of the facial nerve can be tested by asking the patient to wrinkle the forehead, close the eyelid tightly, show the teeth, pucker the lips and grimace. The corner of the mouth droops. The creases and skin folds disappears, the forehead is unfurrowed, the eyelids will not close upon attempted closure of the lids, and the eye on the paralyzed side is seen to roll upward (Bell's phenomena). The lower lid sags also and the punctum falls away from the conjunctiva, permitting tears to spill over the cheek. Food collects between the teeth and lips and saliva may dribble form the corner of the mouth. 1 The sensory component of the nerve is small and conveys taste sensation from the anterior two-thirds of the tongue. Thus, taste sensation may be lost unilaterally and hyeracusis may be present in idiopathic (nontraumatic) BP as well as loss of motor function. Clinically, the onset of BPis fairly abrupt, maximum weakness being attained by 48 hrs [2]. About 80 percent of patients recover within a few weeks or months. EMG may be of value in distinguishing a temporary conduction defect with demyelination pattern instead of axonal damage which is more in favor of traumatic nerve damage [3,4].

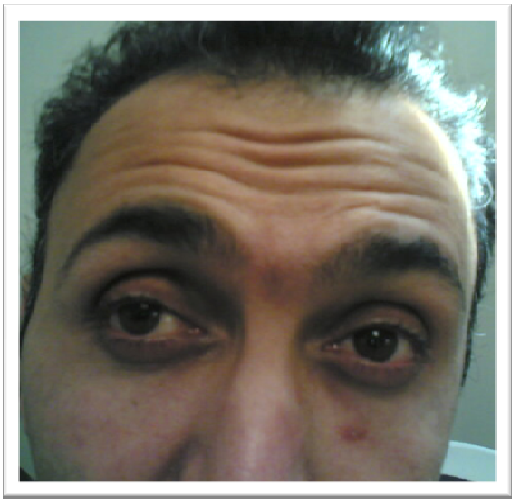

Figure 3. Post-treatment view of the patient exhibiting return of corrugator muscle function.

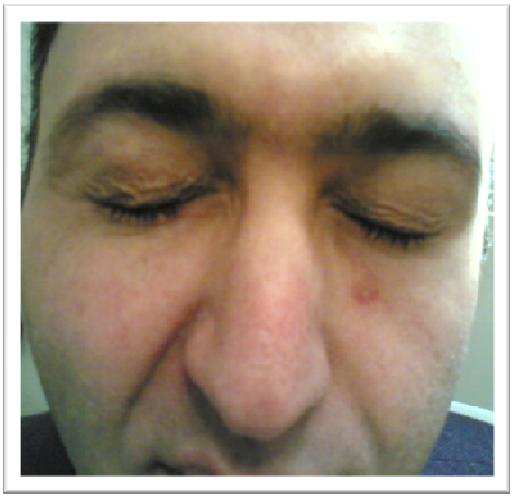

Figure 4. Post-treatment view of the patient exhibiting return of eyelid function.

Idiopathic BPis due to inflammation of the facial nerve in the facial canal. BP may also occur from lesions that invade the temporal bone (carotid body, cholesteatoma, dermoid cyst, acoustic neuromas) and produce FP, but the onset is insidious and the course progressive and the signs and symptoms depend on the part of the nerve damaged (nerve trunk or terminal branches).

The Ramsay-Hunt syndrome presumed to be due to herpes zoster of the geniculate ganglion is a severe hemi-facial palsy associated with vesicular eruptions in the pharynx, external auditory canal and other parts of the cranial integument. Infarcts are common pontine lesions which also may interrupt the facial nerve fibers. These forms of peripheral FP must be distinguished from the supranuclear type; in the latter the frontalis and orbicularis occuli muscles are involved less than those of the lower part of the face since the upper facial muscles are innervated by corticobulbar pathways from both motor cortices, whereas the lower facial muscles are innervated only by the opposite hemisphere. In supranuclear lesions there may be a dissociation of emotional and voluntary facial movement.

For above reported case electroneurography performed between day 7 and 10 for BP does not provide accurate information on the prognosis or recovery rate of the facial paralysis [5]. However, evidence suggests a possible association with herpes simplex virus in idiopathic Bell's palsy. In this type of BP, symptoms nonrelated to palsy of the 5 terminal branches of the facial nerve are also often seen [3,4]. Although there are limited clinical and epidemiological data on patients diagnosed with Bell's palsy, in a survey of 258 patients diagnosed with idiopathic facial (Bell's) palsy, the most common symptoms accompanying BP were increased

tearing (63%), pain in or around the ear (63%), and taste abnormalities (52%); and a significant number of patients reported neurological symptoms not attributable to the facial nerve (similar to our case). They concluded that patients diagnosed with BP have a variety of neurological symptoms, many of which cannot be attributed solely to a facial nerve disorder (in contrast to traumatic BP) [3].

Facial nerve palsy (FNP) following a traumatic sagittal split osteotomy is a rare. Vries reported the incidence of facial nerve injury in a group of 1747 patients who had undergone a bilateral sagittal split osteotomy (3494 sagittal splits) to be 0.26% [7]. Additionally, reported cases of FNP related to surgery also are more delayed in onset [8-10] and we have reported such a case (but only the frontal branch was involved 48 hrs postoperatively) [10]. In contrast, the present case presented with paralysis of the mandibular nerve branch immediately postoperatively in the RR which progressed to involve all branches within 24 hrs. The surgical protocol used was routine, atraumatic and bilateral; we could not find a relationship with the surgical operation (although this has been reported). A past medical history of BP (reported postoperatively), pain and tenderness in mastoid and tragus area, exposure of the right side of the face to a continual draft of cold air and moisture of the face (from irrigation during surgery) during the course of the procedure, EMG pattern (demyelinating vs axonal damage), tearing, taste problems, and hyperacusis in addition to palsy of all the branches of the facial nerve, are strongly in favor of idiopathic (nontraumatic) BP (although post-traumatic BP cannot be ruled out). Cold has been found to play a role the presentation of BP and reactivation of herpes simplex has been implicated [11,12]. It was interesting to find that our patient was not distressed or concerned as much as we would expect in a patient with postoperative BP. Further inquiry into this issue led him to recall a past history of Bell's palsy (5 years back) which resolved without sequelae. This issue was also strongly in favor of recurrent idiopathic Bell's palsy.

Treatment

The main aims of treatment are to speed-up recovery and to prevent corneal complications. Treatment should begin immediately to inhibit viral replication and the effect on subsequent pathophysiological processes that affect the facial nerve. Psychological support is also essential, as is follow up [4]. In viral facial palsies as yet there is no reliable investigation or test to determine who will make a full recovery. Patients with BP should focus on protecting the cornea from drying and abrasion due to problems with lid closure and lacrimation. Lubricating drops should be applied hourly during the day and an eye ointment should be used at night [2-4].

The use of steroids has been suggested as a means of limiting facial nerve damage in the acute phase. In this phase, clinicians play a pivotal role in preventing blindness from corneal exposure [6]. A course of prednisolone beginning with 50 to 80 mg daily during the first 5 days and then tapered ever the next 5 days has been deemed beneficial. Some have found treatment of patients with BP with a com-

bination of acyclovir and prednisone to be more effective [13-16]. Non-invasive electrode pulse electric stimulation at facial points has also shown therapeutic effects [16,17].

Immunocompetent patients without specific contraindications can be prescribed prednisone at 1 mg/kg/d (maximum 80 mg) for the first week, which is tapered over the second week.

Treatment with antivirals seems logical in BP because of the probable involvement of herpes viruses. Aciclovir, a nucleotide analogue, interferes with herpes virus DNA polymerase and inhibits DNA replication [3,4]. Evolving treatments for BP with some evidence of effect include: Methylcobalamin—an active form of vitamin B-12, hyperbaric oxygen—may be useful in patients who show degeneration despite maximal therapy, Facial retraining—"mime therapy", botulinum toxin for synkinesis and hemifacial spasm [4].

Prognosis

Overall, BP has a fair prognosis without treatment, with almost three quarters of patients recovering normal mimetical function and just over a tenth having minor sequelae. By 6 months it is clear who will have moderate to severe sequelae [4]. A sixth of the patients are left with either moderate to severe weakness, contracture, hemifacial spasm, or synkinesis. Patients with a partial palsy fair better, with 94% making a full recovery. Treatment is probably more effective before 72 hours and less effective after seven days. Indicators of poor prognosis in BP include: Complete facial palsy, no recovery by three weeks, age over 60 years, severe pain, Ramsay Hunt syndrome (herpes zoster virus), associated conditions—hypertension, diabetes, pregnancy and severe degeneration of the facial nerve shown by electrophysiological testing [3,4]. Our patient recovered fully within 4 mos.

As climate and cold have been shown to predispose to viral BP, attention to control of ambient air temperature and drafts in the operating room is important. Additionally, noting a history of BP in such cases preoperatively may be paramount in preventing liability and litigation postoperatively. Our patient did not give a history of previous BP until after surgery.

List of abbreviations

BP: Bell's palsy.
FP: Facial paralysis.
FNP: Facial nerve palsy.
RR: Recovery room.

Conflicts of interests

The authors declare that they have no competing interest. MM has editorial involvement with *Dental Hypotheses.*

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Authors' contributions

- ***Main idea:*** by MS.
- ***Literature search:*** by MM.
- ***Data collection***: by MS.
- ***Data interpretation:*** by AS.
- ***Manuscript preparation:*** by MM.
- ***Funds Collection:*** N/A.

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