Seventh Nerve Palsy Secondary to Parotid Malignancy
Authors: Yelena Smart, OD, Justyna Lewczuk, OD

Abstract:

A 65 year old black male presents with chronic right facial paralysis. After poor response to standard treatment, additional testing reveals seventh nerve palsy caused by parotid malignancy.

I. Case History
   - Patient Demographics
     65 year old black male presents for ophthalmic consultation requested by his otolaryngologist.

   - Chief Complaint
     Patient reports onset of right sided facial paralysis between February and March of 2011. Patient complains of significant epiphora and burning of the right eye present for several months. He reports discomfort and lack of symptomatic relief with use of preserved artificial tears.

   - Ocular and Medical History
     Medical History:
     (+) alcohol dependence
     (+) sensorineural hearing loss, bilateral  
     (+) tinnitus
     (+) dysthnia
     (+) panic disorder without agoraphobia
     (+) unspecified personality disorder
     (+) impulse control disorder
     (+) malignant neoplasm of tonsil
     (+) hyperlipidemia
     (+) paresthesias
     (+) chronic low back pain

     Ocular History:
     (+) history of trauma to left eye, 15-20 years ago, when patient was hit with a belt buckle to the eye
     (+) s/p gold weight implantation to upper right eyelid

   - Medications
     Simvastatin
     Hydrochlorothiazide/Lisinopril
     Lubricating ophthalmic ointment
     Artificial Tears

   - Other Salient Information
     Patient originally presented to the emergency room with a one month history of right sided facial swelling and droop and worsening ability to close the right eye. Patient was then referred for an ENT consult where he was diagnosed with right facial nerve palsy. Multitude of subsequent
tests revealed a parotid carcinoma with seventh cranial nerve invasion. Patient has since undergone a gold weight implantation procedure to his right upper eyelid.

II. Pertinent Findings

-Clinical

Best Corrected Visual Acuity right eye (OD) 20/20, left eye (OS) 20/200

Slit lamp:
- Lids/lashes: mild lagophthalmos OD with palpable gold weight implant upper lid OD normal OS
- Conjunctiva: normal OU
- Cornea: inferior punctate keratitis OD, old stromal foreign body scar OS
- Iris: flat and intact OU
- Lens: trace + anterior and posterior cortical and nuclear sclerotic cataracts OU

Goldmann Tonometry: OD 18 mm Hg OS 18 mm Hg at 11:44 AM

Dilated Fundus Exam:
- Optic Nerves: cup to disc ratio: 0.45 OU with pink, distinct rim tissue OU
- Macula: OD: clear OS: ½ disc diameter macular hole with surrounding pigment and large chorioretinal scarring temporally
- Periphery: Unremarkable OU

-Physical

External examination was consistent with right sided facial nerve palsy and revealed right sided facial droop and swelling, incomplete right eyelid closure, inability to elevate the right brow, right lid droop and facial asymmetry most prominent during attempts to smile and talk. Previous examination by ENT physicians also revealed a palpable right parotid mass and lack of cervical adenopathy.

- Laboratory Studies were not required

- Pathology Studies

Early testing initiated by patient’s emergency room and otolaryngology physicians consisted of fine needle aspiration (FNA) biopsy of the right parotid gland which did not reveal any malignant cells. Patient was subsequently diagnosed with right sided Bell’s palsy and treated with oral corticosteroids. Failure to respond to steroid treatment and progression of patient’s symptoms prompted a repeat FNA which was again non-diagnostic. Patient then underwent an open biopsy of the right parotid gland and concurrent right eyelid gold weight placement. Pathology reports from the biopsy were suggestive of salivary gland cyst. Finally, a right superficial parotidectomy with facial nerve dissection was performed for definitive diagnosis and revealed a mucoepidermoid carcinoma of the right parotid gland with perineural invasion.

-Radiology Studies

Upon initial presentation to the emergency room, computed tomography (CT) scan revealed a low attenuating lesion centered in the superficial lobe of the right parotid gland. A subsequent
MRI of the brain and neck revealed a small acute left occipitoparietal infarct and a large right parotid gland tumor suspicious for adenoid cystic and/or mucoepidermoid tumor. A later PET/CT scan revealed hypermetabolic activity in the right parotid gland without evidence of enlarged lymphadenopathy on either side of the neck.

III. Differential Diagnosis

- Primary leading
  Facial nerve palsy secondary to parotid gland malignancy

- Others
  Common causes of unilateral facial nerve palsy include Bell’s Palsy (idiopathic), temporal bone fractures, head and neck tumors, infections (primarily herpes simplex and zoster, and lyme disease), central nervous system lesions (including pontine strokes) as well as drug induced, iatrogenic (post surgical nerve damage), granulomatous disorders and other rare causes.

IV. Diagnosis and Discussion

Facial nerve palsy is a neurological condition characterized by the onset of facial weakness along the distribution of the facial nerve branches with symptoms of impaired facial expression, ocular dryness, epiphora, inability to close or wink the eye, inability to close the mouth, droop of the eyebrow and mouth and may be accompanied by pain and numbness in the surrounding area of the face and chin, altered taste, increased sensitivity to sound and decreased tearing (3). The primary challenge faced by clinicians who encounter a seventh cranial nerve palsy lies in identifying its correct etiology. Unilateral peripheral facial nerve palsy is most commonly idiopathic and referred to as Bell’s palsy. However, Bell’s palsy is a diagnosis of exclusion and more ominous secondary causes must first be ruled out (3). Idiopathic facial nerve palsy is characterized by unilateral, sudden onset of facial paralysis with progression over the first 7-10 days, followed by improvement in symptoms which typically starts at 3-4 weeks and spontaneous recovery within the first 6 months (7). Although the exact mechanism of Bell’s palsy remains to be elucidated, it has been suggested that viral etiology (especially herpes simplex, herpes zoster and lyme disease), autoimmune disease and vascular ischemia may play a role in its pathogenesis (3). Some secondary causes of seventh nerve palsies include trauma to the temporal bone, head and neck tumors, infections, granulomatous disease, pontine infarcts and several others (4).

In the case discussed above, several important entities differentiated the clinical presentation from that of a typical Bell’s palsy. First, this patient presented with an insidious onset of facial paralysis that occurred over approximately a one month period. This is in contrast to the typical sudden onset of symptoms seen with idiopathic facial nerve palsies (7). Nonetheless, Quesnel and associates detailed four case reports of acute onset facial paralysis caused by parotid malignancy and this atypical presentation further delayed diagnosis of the occult parotid tumors (6). Secondly, this patient had demonstrated a failure to improve symptomatically despite administration of systemic corticosteroids. This is the key finding that alerted clinicians to explore secondary causes. The hallmark of Bell’s palsy is marked improvement in signs and symptoms which begins at approximately 3 weeks and complete spontaneous recovery in most patients at the six month mark which occurs even without treatment (4, 7). Eighty-five percent of patients with Bell’s palsy exhibit signs of recovery at 3
weeks and almost all demonstrate some recovery by 12-24 weeks (6). Furthermore, mounting evidence suggests that administration of systemic corticosteroids improves recovery outcomes in patients (3, 4, 7). Thus, when this patient exhibited no notable improvement of symptoms within the expected time frame, the necessity of additional testing became paramount. Finally, it is important to note that this patient had a positive history of left tonsillar carcinoma. The aforementioned factors all contributed to clinicians’ high level of suspicion of a secondary cause of facial nerve paralysis and prompted the systematic efforts at identifying that cause. Imaging for a suspected neoplasm in a patient with a seventh nerve palsy should include fine resolution, contrast enhanced CT scan and gadolinium enhanced MRI of the temporal bone, brain, neck and parotid beds (6). If imaging is nondiagnostic, biopsy of the suspected area(s) may be performed (6).

V. Treatment, Management

Primary goals of treatment and management of seventh nerve palsies are twofold: identification and management of the secondary cause of the palsy if such exists and protection of the cornea from vision threatening sequelae due to lagophthalmos, paralytic ectropion and dryness (6).

Upon extensive testing, this patient was diagnosed with a seventh nerve palsy caused by an adenocarcinoma of the parotid gland with perineural invasion. The primary treatment modality for such a lesion is surgical excision with an emphasis on preservation of the facial nerve when possible (2, 5).

When clinical examination and auxiliary testing suggest an idiopathic etiology, Bell’s palsy is diagnosed and medical treatment often consists of systemic administration of corticosteroids, antiviral agents or both (3, 4, 7).

Supportive treatment for all cases of seventh nerve palsy is essentially identical. Acute care focuses on protecting the cornea from dehydration, drying and abrasions due to exposure from incomplete lid closure (3). Furthermore, dysfunction of the seventh nerve may cause decreased tear production and loss of Bell’s phenomenon which further exacerbates the corneal exposure (1). Common treatment modalities include intensive lubrication with preservative free artificial tear drops during daytime and a lubricating ointment for nighttime use (7). Other beneficial measures include eyelid taping at night, moisture goggles and punctal occlusion (7). Careful monitoring of ocular surface status is recommended (7).

Consistent with the aforementioned guidelines, optometric measures consisted of frequent and copious lubrication with preservative free artificial tears through the day time hours and application of lubricating ophthalmic ointment to the eye at night.

When conservative interventions are insufficient to reduce exposure keratitis, surgical measures can be explored. A common surgical approach is implantation of gold weights into the upper eyelid of the affected eye in hope of reducing lagophthalmos and promoting more complete eyelid closure by maximizing the effects of gravity (1). Our patient has undergone such a procedure prior to the optometric consultation, although he was still quite symptomatic for epiphora and dryness at the time of my examination. In this case, a reversible tarsorrhaphy may provide additional relief (4, 7). In some patients, the use of botulinum toxin has been advocated both as a means to induce protective ptosis during the acute phase of facial palsy (7) and as a way to relieve the synkinesis and facial spasms that may persist upon incomplete
recovery from the palsy (3). Thus, a referral for an oculoplastics consultation was also warranted in the present case.

-Bibliography, literature review encouraged

VI. Conclusion
-Clinical pearls, take away points if indicated

This case highlights several important aspects of management of facial nerve palsies. Although most cases are indeed idiopathic, with a favorable prognosis for recovery within a short time span, the need for a thorough examination cannot be overemphasized. Bell’s palsy is a diagnosis of exclusion and requires the clinician to rule out infiltrative, infectious, autoimmune and vasculopathic causes for the facial paralysis. High clinical suspicion for secondary etiologies should exist if the palsy is not sudden onset, fails to follow the predicted clinical course of improvement or if pertinent patient history and clinical examination suggest a non benign cause.

Regardless of etiology, palliative treatment of ocular manifestations of facial palsy should be promptly initiated. Aggressive ocular surface lubrication and protection from exposure are of utmost importance in order to prevent unwanted sequelae of corneal exposure, including chronic dryness, infection and even blindness. If conservative management is inadequate, referral to an oculoplastic specialist for consideration of other surgical or medical intervention is encouraged.

This case also underscores the importance of co-management of facial nerve palsies, especially in the setting of an atypical presentation and lack of expected resolution. Ideally, a seventh nerve palsy should be evaluated by a neurologist, an otolaryngologist and an eye care provider (3) as a multidisciplinary approach is likely to yield the best patient outcomes in terms of both appropriate diagnosis and suitable management.