Bell Palsy Mimics

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A 61-year-old woman presented for evaluation of right facial drooping and vertigo.

Facial paralysis is a common medical complaint—one that has fascinated ancient and contemporary physicians alike.[1] An idiopathic facial nerve paresis involving the lower motor neuron was described in 1821 by Sir Charles Bell. This entity became known as a Bell's palsy, the hallmark of which was weakness or complete paralysis of the muscles of one side of the face, with no sparing of the muscles of the forehead. However, not all facial paralysis is due to Bell's palsy.

We present a case of a patient with a Bell's palsy mimic to facilitate and guide the differential diagnosis and distinguish conditions from the classical presentation that Bell first described to the more concerning symptoms that may not be immediately obvious. Our case further underscores the importance of performing a thorough assessment to determine the presence of other neurological findings.

Case

A 61-year-old woman presented to the ED for evaluation of right facial droop and sensation of "room spinning." The patient stated both symptoms began approximately 36 hours prior to presentation, upon awakening. She further noted that the right side of her face felt "funny" and numb.

The patient denied any headache, neck or chest pain, extremity numbness, or weakness, but stated that she felt like she was going to fall toward her right side whenever she attempted to walk. The patient's medical history was significant for hypertension, for which she was taking losartan. Her surgical history was notable for a left oophorectomy secondary to an ovarian cyst. Regarding the social history, the patient admitted to smoking 90 packs of cigarettes per year, but denied alcohol or illicit drug use.

Upon arrival at the ED, the patient's vital signs were: blood pressure, 164/86 mm Hg: pulse, 89 beats/min; respiratory rate, 18 breaths/min; and temperature, 98.6°F. Oxygen saturation was 98% on room air.

Physical examination revealed the patient had a right facial droop consistent with right facial palsy. She was unable to wrinkle her right forehead or fully close her right eye. There were no field cuts on confrontation. The patient's speech was noticeable for a mild dysarthria. The motor examination revealed mild weakness of the left upper extremity and impaired right facial sensation. There were no rashes noted on the face, head, or ears. The patient had slightly impaired hearing in the right ear, which was new in onset. The remainder of the physical examination was unremarkable.

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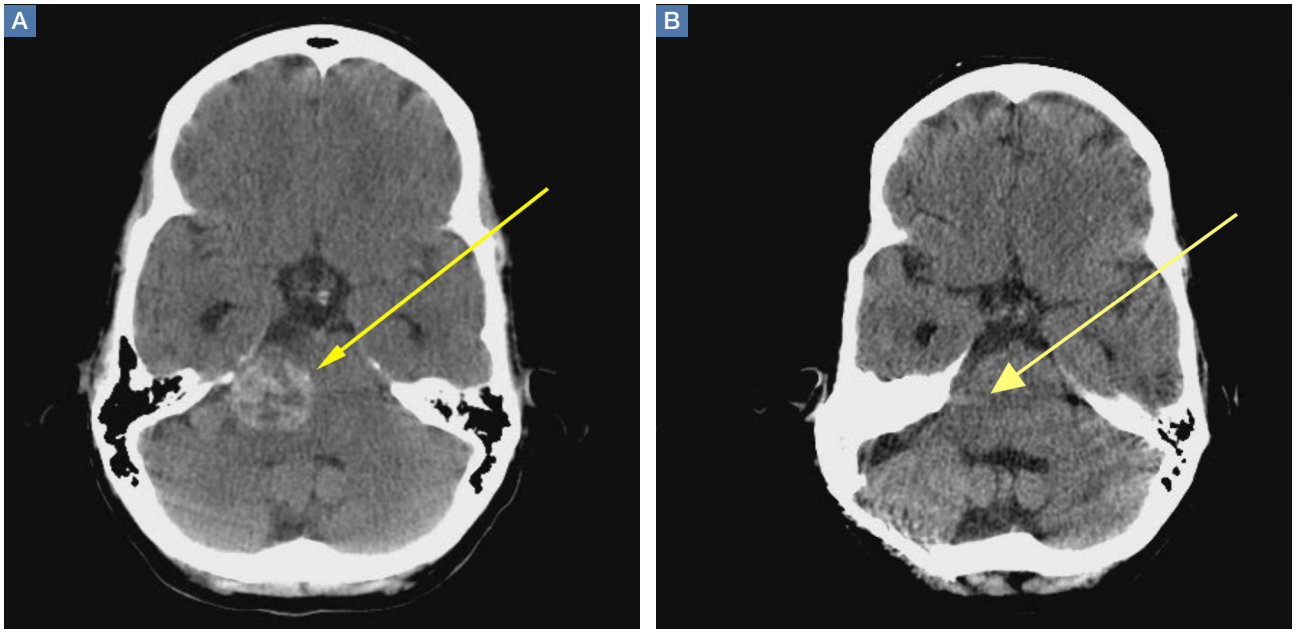

Figure 1. Computed tomography (CT) image of the patient's head (**A**) demonstrates a large mass lesion centered in the right petrous apex with associated large component extending medially into the right cerebellopontine angle, causing mass effect on the adjacent brainstem (**yellow arrow**). (**B**) A follow-up CT image taken 2 months after a right suboccipital craniotomy with subtotal removal of the tumor demonstrates absence of the mass (**yellow arrow**).

Although the patient exhibited the classic signs of Bell's palsy, including complete paralysis of the muscles of one side of the face, inability to wrinkle the muscle of the right forehead, and inability to fully close the right eye, she also had concerning symptoms of vertigo, dysarthria, and contralateral upper extremity weakness.

A computed tomography (CT) scan of the head was ordered, which revealed a large mass lesion centered in the right petrous apex, with an associated large component extending medially into the right cerebellopontine angle (CPA) that caused a mass effect on the adjacent brainstem (**Figures 1a** and **1b**).

Upon these findings, the patient was transferred to another facility for neurosurgical evaluation. Magnetic resonance imaging (MRI) studies performed at the receiving hospital demonstrated a large expansile heterogeneous mass lesion centered in the right petrous apex with an associated large, probable hemorrhagic soft-tissue component extending medially into the right CPA, causing a mass effect on the adjacent brainstem and mild obstructive hydrocephalus (**Figures 2a** and **2b**).

The patient was given dexamethasone 10 mg intravenously and taken to the operating room for a right suboccipital craniotomy with subtotal tumor removal. Intraoperative high-voltage stimulation of the fifth to eighth cranial nerves showed no response, indicating significant impairment.

While there were no intraoperative complications, the patient had significant postoperative dysphagia and resultant aspiration. A tracheostomy and percutaneous endoscopic gastrostomy tube were subsequently placed. Results of a biopsy taken during surgery identified an atypical meningioma. The patient remained in the hospital for 4 weeks, after which she was discharged to a long-term care (LTC) and rehabilitation facility.

A repeat CT scan taken 2 months after surgery demonstrated absence of the previously identified large mass (**Figure 1b**). Three months after discharge from the

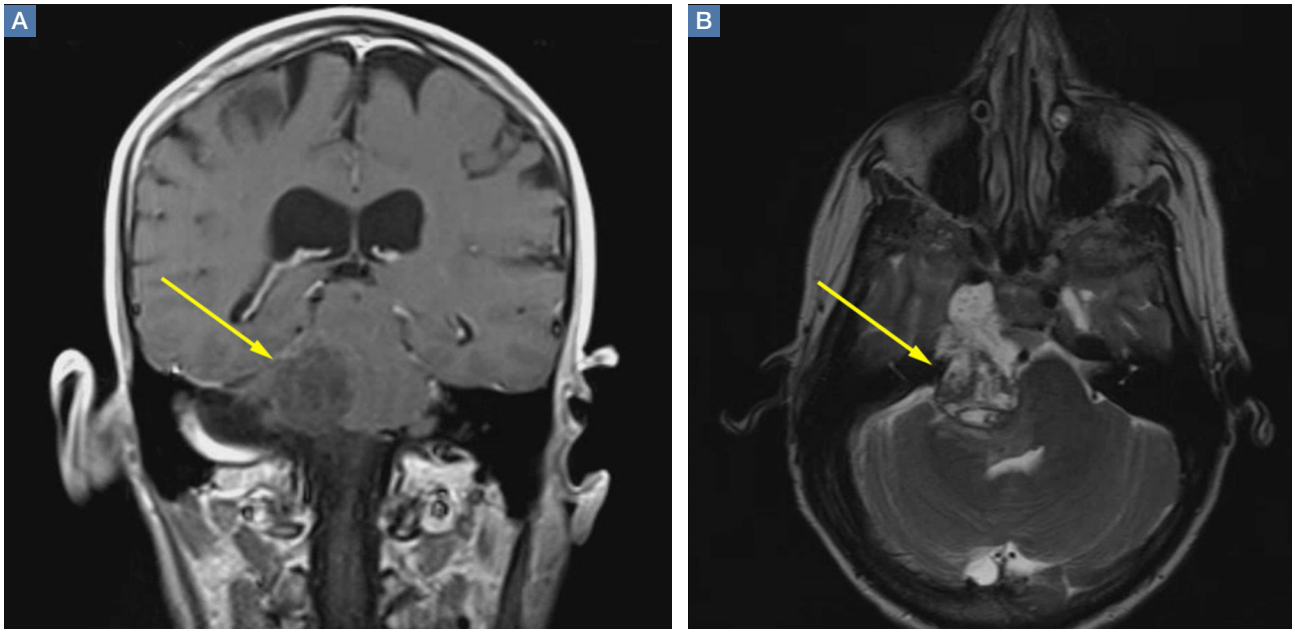

Figure 2. Magnetic resonance imaging of the patient's brain (**A** and **B**) demonstrates a large expansile heterogeneous mass lesion (**yellow arrow**) centered in the right petrous apex with associated large probable hemorrhagic soft-tissue component extending medially into the right cerebellopontine angle, causing mass effect on the adjacent brainstem and probable mild obstructive hydrocephalus.

LTC-rehabilitation facility, MRI of the brain showed continued interval improvement of the previously noted mass centered in the right petrous apex (**Figures 3a** and **3b**).

Discussion

Accounts of facial paralysis and facial nerve disorders have been noted throughout history and include accounts of the condition by Hippocrates.[1] Bell's palsy was named after surgeon Sir Charles Bell, who described a peripheral-nerve paralysis of the facial nerve in 1821. Bell's work helped to elucidate the anatomy and functional role of the facial nerve.[1,2]

Signs and Symptoms

The classic presentation of Bell's palsy is weakness or complete paralysis of the muscles of one side of the face, with no sparing of the muscles of the forehead. The eyelid on the affected side generally does not close, which can result in ocular irritation due to ineffective lubrication.

A scoring system has been developed by House and Brackmann which grades the degree impairment based on such characteristics as facial muscle function and eye closure.[3,4] Approximately 96% of patients with a Bell's palsy will improve to a House-Brackmann score of 2 or better within 1 year from diagnosis,[5] and 85% of patients with Bell's palsy will show at least some improvement within 3 weeks of onset (**Table**).[2]

Although the classic description of Bell's palsy notes the condition as idiopathic, there is an increasing body of evidence in the literature showing a link to herpes simplex virus 1.[5-7]

Ramsey-Hunt Syndrome

The relationship between Bell's palsy and Ramsey-Hunt syndrome is complex and controversial. Ramsey-Hunt syndrome is a constellation of possible complications from varicella-virus infection. Symptoms of Ramsey-Hunt syndrome include facial paralysis, tinnitus, hearing loss, vertigo, hyperacusis (increased sensitivity

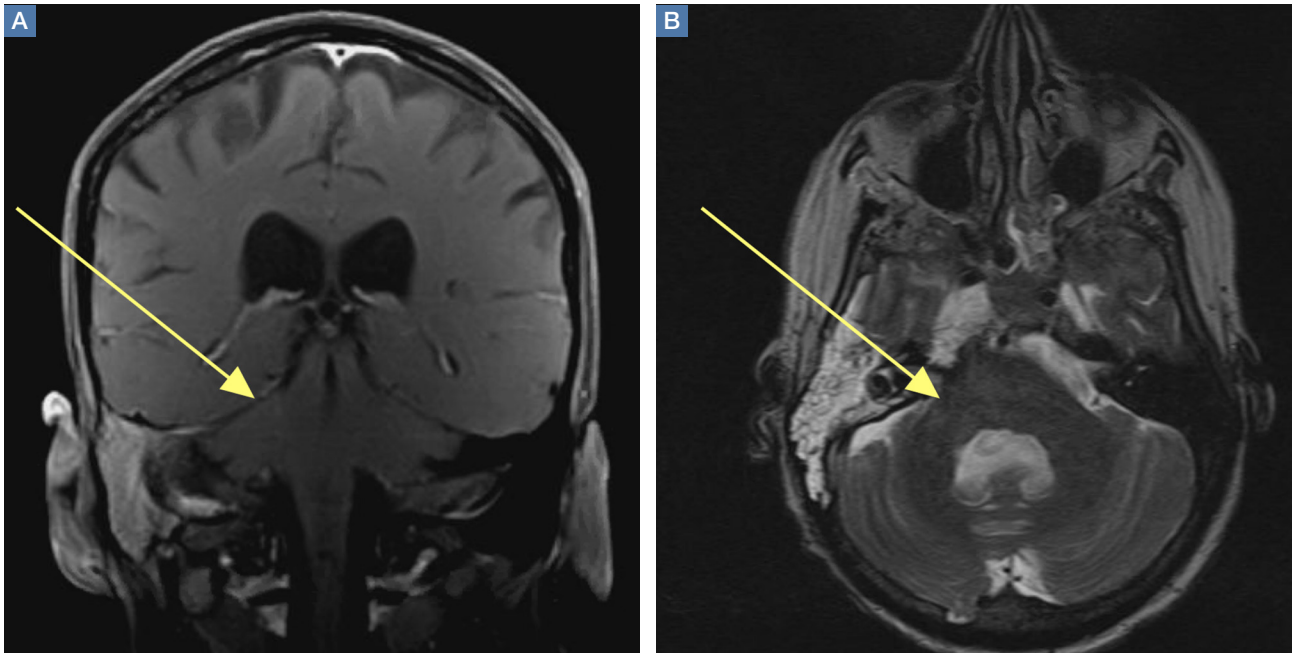

Figure 3. Magnetic resonance imaging of the patient's brain 3 months after debulking of the previously noted mass lesion centered in the right petrous apex (**A** and **B**) demonstrates a continued interval improved mass effect (**yellow arrow**).

to certain frequencies and volume ranges of sound), and decreased ocular tearing.[8] Due to the nature of symptoms associated with Ramsey-Hunt syndrome, it is apparent that the condition involves more than the seventh cranial nerve. In fact, studies have shown that Ramsey-Hunt syndrome can affect the fifth, sixth, eighth, and ninth cranial nerves.[8]

Ramsey-Hunt syndrome, which can present in the absence of cutaneous rash (referred to as zoster sine herpete), is estimated to occur in 8% to 20% of unilateral facial nerve palsies in adult patients.[8,9]

Regardless of the etiology of Bell's palsy, a review of the literature makes it clear that facial nerve paralysis is not synonymous with Bell's palsy.[10] In one example, Yetter et al[10] describe the case of a patient who, though initially diagnosed with Bell's palsy, ultimately was found to have a facial palsy due to a parotid gland malignancy.

Likewise, Stomeo[11] describes a case of a patient with facial paralysis and profound ipsilateral hearing loss who ultimately was found to have a mucoepithelial carcinoma of the parotid gland. In their report, the authors note that approximately 80% of facial nerve paralysis is due to Bell's palsy, while 5% is due to malignancy.

In another report, Clemis[12] describes a case in which a patient who initially was diagnosed with Bell's palsy eventually was found to have an adenoid cystic carcinoma of the parotid. Thus, the authors appropriately emphasize in their report that "all that palsies is not Bell's."

Differential Diagnosis

Historical factors, including timing and duration of symptom onset, help to distinguish a Bell's palsy from other disorders that can mimic this condition. In their study, Brach VanSwewaringen[13] highlight the fact that "not all facial paralysis is Bell's palsy." In their review, the authors describe clues to help distinguish conditions that mimic Bell's palsy. For example, maximal weakness from Bell's Palsy typically occurs within 3 to 7 days from symp-

Table. House-Brackmann Grading System

Grade	Characteristics
1	Normal facial muscle function, normal eye closure.
2	On close inspection, slight facial muscle weakness noticeable. Complete eye closure with minimal effort.
3	Obvious difference between two sides of face, but not disfiguring. Complete eye closure with maximal effort.
4	Obvious facial muscle weakness and/or disfiguring asymmetry. Incomplete eye closure with maximal effort.
5	Facial muscle motion barely perceptible. Incomplete eye closure with maximal effort.
6	No facial muscle movement. Inability to close eye with maximal effort.

Adapted from House JW, Brackmann DE. Facial nerve grading system. *Otolaryngol Head Neck Surg*. 1985;93(2):146-147.[3]

tom onset, and that a more gradual onset of symptoms, with slow or negligible improvement over 6 to 12 months, is more indicative of a space-occupying lesion than Bell's palsy.[13] It is, however, important to note that although the patient in our case had a central lesion, she experienced an acute onset of symptoms.

The presence of additional symptoms may also suggest an alternative diagnosis. Brach and VanSwearingen[13] further noted that symptoms associated with the eighth nerve, such as vertigo, tinnitus, and hearing loss may be found in patients with a CPA tumor. In patients with larger tumors, ninth and 10th nerve symptoms, including the impaired hearing noted in our patient, may be present. Some patients with ninth and 10th nerve symptoms may perceive a sense of facial numbness, but actual sensory changes in the facial nerve distribution are unlikely in Bell's palsy. Gustatory changes, however, are consistent with Bell's palsy.

Ear pain is consistent with Bell's palsy and is a signal to be vigilant for the possible emergence of an ear rash, which would suggest the diagnosis of herpes zoster oticus along the trajectory of Ramsey-Hunt syndrome. Facial pain in the area of the facial nerve is inconsistent with Bell's palsy, while hyperacusis is consistent with Bell's palsy. Hearing loss is an eighth nerve symptom that is inconsistent with Bell's palsy.

Similarly, there are physical examination findings that can help distinguish a true Bell's palsy from a mimic. Changes in tear production are consistent with Bell's palsy, but imbalance and disequilibrium are not.[14]

As previously noted, the patient in this case had difficulty walking and felt as if she was falling toward her right side.

One way to organize the causes of facial paralysis has been proposed by Adour et al.[15] In this system, etiologies are listed as either acute paralysis or chronic, progressive paralysis. Acute paralysis (ie, the sudden onset of symptoms with maximal severity within 2 weeks), of which Bell's palsy is the most common, can be seen in cases of polyneuritis.

A new case of Bell's palsy has been estimated to occur in the United States every 10 minutes.[8] Guillain-Barré syndrome and Lyme disease are also in this category, as is Ramsey-Hunt syndrome. Patients with Lyme disease may have a history of a tick bite or rash.[14]

Trauma can also cause acute facial nerve paralysis (eg, blunt trauma-associated facial fracture, penetrating trauma, birth trauma). Unilateral central facial weakness

can have a neurological cause, such as a lesion to the contralateral cortex, subcortical white matter, or internal capsule.[2,15] Otitis media can sometimes cause facial paralysis.[16] A cholesteatoma can cause acute facial paralysis.[2]

Malignancies cause 5% of all cases of facial paralysis. Primary parotid tumors of various types are in this category. Metastatic disease from breast, lung, skin, colon, and kidney may cause facial paralysis. As our case illustrates, CPA tumors can cause facial paralysis.[15]

It is important to also note that a patient can have both a Bell's palsy and a concurrent disease. There are a number of case reports in the literature that describe acute onset of facial paralysis as a presenting symptom of malignancy.[17]

In addition, there are cases wherein a neurological finding on imaging, such as an acoustic neuroma, was presumed to be the cause of facial paralysis, yet the patient's symptoms resolved in a manner consistent with Bell's palsy.[18]

For example, Lagman et al[19] described a patient in which a CPA lipoma was presumed to be the cause of the facial paralysis, but the eventual outcome showed the lipoma to have been an incidentaloma.

Conclusion

This case demonstrates a presenting symptom of facial palsy and the presence of a CPA tumor. The presence of vertigo along with other historical and physical examination findings inconsistent with Bell's palsy prompted the CT scan of the head. A review of the literature suggests a number of important findings in patients with facial palsy to assist the clinician in distinguishing true Bell's palsy from other diseases that can mimic this condition. This case serves as a reminder of the need to perform a thorough and diligent workup to determine the presence or absence of other neurologic findings prior to closing on the diagnosis of Bell's palsy.

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