The Complexities of Management of Facial Nerve Palsy in the Eye Care Setting

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Abstract

Facial nerve palsy is a relatively common neurological condition that often presents along with severe ocular manifestations that require treatment. While facial nerve palsy is most commonly idiopathic (when it is also known as Bell's palsy), other more ominous causes need to be ruled out. Identification of the correct etiology relies on proper anatomical localization. Management is typically multidisciplinary, and encompasses the correct diagnosis, treatment of the ocular complications and often the use of oral medications to treat the palsy itself, as demonstrated in this case report.

KEY WORDS
Bell's palsy, facial nerve palsy, seventh nerve palsy, facial neuropathy

INTRODUCTION

Facial nerve palsy (FNP) is a neurological condition characterized by the onset of facial weakness along the distribution of the branches of the facial nerve (cranial nerve VII), with signs and symptoms including impaired facial expression, ocular dryness (due to both exposure keratitis and lacrimal gland dysfunction), epiphora, inability to close or wink the eye, inability to close the mouth, and drooping of the eyebrow and mouth. It may be accompanied by pain and numbness in the area surrounding the face and chin, altered taste, increased sensitivity to sound and decreased tearing.[1] The primary challenge faced by clinicians who encounter a seventh cranial nerve palsy is identifying the underlying etiology, if present. Unilateral peripheral FNP 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, including herpes zoster virus, Lyme disease and even stroke.[1-3] All cases of FNP carry a significant risk of ocular morbidity. Thus, it is crucial for eye care providers to be familiar with this clinical entity.

CASE REPORT

A 69-year-old white male presented as a new patient to the eye clinic at the request of his primary care physician (PCP) for a same-day consultation. The patient complained of a sudden-onset left-sided eyelid and facial droop that had begun four days previously (the patient did not seek medical care for the first three days of his symptoms). The patient's PCP diagnosed a left-sided seventh cranial nerve palsy and initiated treatment that morning with oral prednisone (60 mg/day for 5 days, with a taper of 10 mg/day for 5 more days) and oral valacyclovir (3000 mg/day for 7 days). The patient denied any severe pain or tingling sensation on the left side of his face, a history of rashes, bug bites, fever, discomfort, or cold sores, changes in vision, dizziness, loss of vision, ataxia, or limb weakness. His medical history was significant for hyperlipidemia, anxiety disorder and a history of kidney cancer that had been previously treated with nephrectomy (he did not have active kidney disease). His ocular history was unremarkable, and his last eye exam was 3 years prior at another clinic.

Entering visual acuities with habitual correction were 20/25 in the right eye and 20/60 in the left eye with improvement to 20/40 with pinhole in the left eye. An external exam was significant for a left-sided facial paresis

with obvious ectropion and droop of the left corner of the mouth (Figures 1-3). Slit lamp exam revealed left lower lid ectropion and lid erythema, inferior punctate keratitis and moderate injection of the palpebral and bulbar conjunctiva, all in the left eye. Intraocular pressures (IOPs) were 22 mm Hg and 28 mm Hg for the right eye and left eye, respectively, at 10:11 AM by applanation tonometry. Dilated fundus exam revealed a cup-to-disc ratio of 0.6 for the right eye and 0.8 for the left eye with characteristic neuroretinal rim thinning in the left eye. The retinae and blood vessels were otherwise unremarkable in both eyes.

The patient was diagnosed with exposure keratitis in the left eye secondary to left-sided FNP. He was started on preservative-free artificial tears every hour and a lubricating ointment to use at night in the left eye. The patient was also instructed to tape his left eyelid closed at night. He was also diagnosed as a glaucoma suspect and asked to return in two weeks for a follow-up exam with further glaucoma testing.

At the two-week exam, the patient reported adherence to the oral medication regimen prescribed by his PCP and the topical treatment prescribed by the eye clinic. The patient remained symptomatic for persistent, but improved, epiphora in the left eye and reported improving eyelid closure on the left side. Visual acuity was stable and slit lamp exam remained significant for lagophthalmos and ectropion with increased punctate keratitis of the left eye with an area of fluorescein pooling in the peripheral inferior cornea, which was suspected to be possible early dellen formation. The patient was instructed to continue with hourly preservative-free artificial tears during the day, replace the lubricating ointment with erythromycin ointment (due to worsening keratitis), and continue lid-taping at night. Suspected glaucoma was to be addressed at subsequent exams.

Figure 1: *Acute presentation of left-sided peripheral facial nerve palsy. Note the prominent ectropion and drooping of the corner of the mouth.*

Figure 2: *Left-sided peripheral facial nerve palsy. Patient attempts eyelid closure. Note the left sided lagophthalmos, smoothing out of forehead wrinkles, and drooping of the corner of the mouth.*

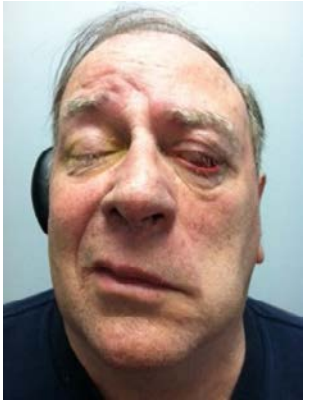
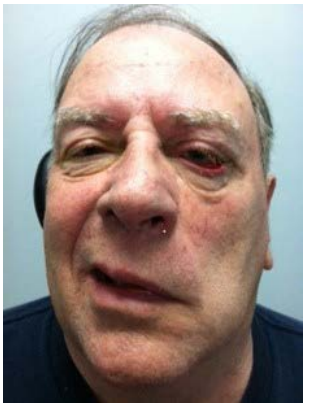


Figure 3: *Same patient now attempts smile. Note the inability to raise the corner of the mouth on the affected side.*



The patient was followed at the eye clinic at 2- to 4-week intervals thereafter. While he continued to adhere to the treatment regimen, the keratopathy and the visual acuity in the left eye did not improve rapidly, and the patient was offered a consultation with an oculoplastics specialist to consider surgical measures. The patient declined the consult and was instructed to continue hourly preservative-free artificial tears, increase erythromycin ointment to four times a day, and to incorporate daytime lid-taping while continuing overnight lid-taping.

After four months, visual acuity in the left eye remained slightly reduced at 20/50 with pinhole, and the ocular surface status was commensurate with the acuity. There was significant improvement of the overall facial droop and additional improvement in lid closure and the ability to blink the left eye (Figures 4, 5). Nonetheless, significant lower lid ectropion and exposure keratitis remained, and the patient was maintained on the same topical treatment regimen. After six months, the patient demonstrated significant recovery of the eyelid position and function, including improved blink, decreased lagophthalmos, and markedly improved lower lid ectropion (Figures 6, 7). Nevertheless, moderate corneal staining persisted, and the patient was asked to continue aggressive lubricating treatment with both preservative-free artificial tears and lubricating ointment at bedtime. Over the next 11 years, his condition remained stable with minimal residual dryness of the left eye, which was managed with topical lubricants, and his vision in the left eye improved to 20/25. He has had no recurrence of facial nerve palsy to date.

Figure 4: *Same patient, at the four month follow up. Note the significant improvement in facial symmetry and mouth droop, however, obvious left lower eyelid ectropion remains.*

Figure 5: *Same patient, at the four month follow up, attempting eyelid closure.*

Figure 6: *Same patient, at the six month follow up. Note the dramatic improvement to the lower lid ectropion.*

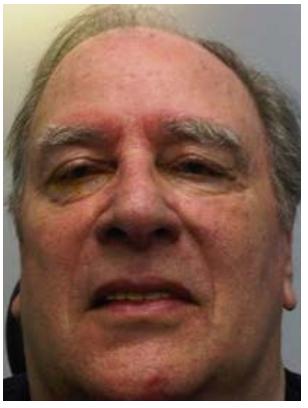
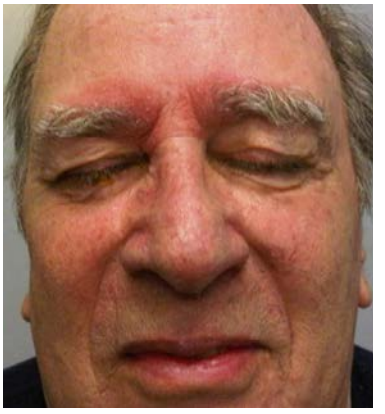


Figure 7: *Same patient, at the six month follow up, successfully attempting eyelid closure.*



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SUN PHARMA

DISCUSSION

An understanding of the anatomy of the facial nerve distribution is essential to appreciating the consequences and the potential etiology of a seventh cranial nerve palsy. The facial nerve is composed of four types of functional fibers.[4] Branchial motor fibers innervate the muscles of facial expression and the stapedius muscle in the ear. Visceral motor fibers supply the secretory glands including the lacrimal, submandibular and sublingual glands. General sensory fibers provide sensory innervation around the ear and special sensory fibers provide taste sensation to the anterior two-thirds of the tongue.

Cortical input to the facial nerve originates in the primary motor cortex of the frontal lobe.[4] Cortical input to the upper facial muscles is bilateral, meaning that a unilateral upper motor neuron or cortical lesion will only affect the contralateral lower muscles of facial expression, sparing the upper facial muscles.[4] A cortical lesion is usually caused by a vascular event or brain tumor and is a rare cause of FNP.[4] In contrast, peripheral facial nerve palsy is caused by a lower motor neuron lesion, i.e., a lesion that is at or distal to the seventh nerve nucleus in the pons, which results in paresis of both the upper and lower muscles of facial expression on the side ipsilateral to the insult.[4] This is an important differentiation as a seventh nerve palsy involving only the bottom portion of the face can indicate a potential acute stroke and is a medical emergency.

Brainstem lesions involving the facial nerve are commonly accompanied by a wide array of other neurological deficits due to the close proximity of seventh nerve fibers to other structures in the area. These neurological deficits include contralateral hemiparesis, ipsilateral gaze palsy, ipsilateral corneal and facial anesthesia, sixth nerve palsy, and cerebellar ataxia.[4,5] These are commonly caused by infarction, pontine glioma and multiple sclerosis.[4]

The facial nerve may also be compromised at the cerebellopontine angle. Concomitant involvement of cranial nerves V, VI and VIII and cerebellar signs localize the lesion to this anatomic area.[4,5] Tumors, including schwannoma and meningioma, are the most likely culprits here.

After traversing through the cerebellopontine angle, the seventh cranial nerve enters the internal acoustic meatus, where it travels alongside the eighth cranial nerve.[3] Here, FNP may be caused by an intracanalicular neuroma and will present with decreased hearing, tinnitus and vestibular impairment.[4] The seventh cranial nerve then travels through the facial canal (also known as the fallopian canal),[4] exits the skull base through the stylomastoid foramen,[2] and then divides into its six major branches within the parotid gland.[4] Prior to its exit from the skull base, both the chorda tympani branch (which carries taste sensation from the tongue) and the nerve to the stapedius muscle (which functions to dampen sound) branch off.[4]

Traumatic causes of seventh nerve palsy stem from injury to the nerve within the facial canal, stylomastoid foramen or the parotid gland.[4] Tumors or infiltration of the parotid gland are other po-

tential etiologies of FNP. Bell's palsy is thought to result from damage or inflammation to the seventh cranial nerve specifically as it travels in the facial canal.[1,4]

The diagnosis of FNP is generally straightforward due to its prominent distinguishing unilateral characteristics including facial weakness, drooping of the eyebrow, lagophthalmos, smoothing of forehead wrinkles, inability to close or blink the eye, drooping at the corner of the eye, drooping of the corner of the mouth, absence of the nasolabial fold, and inability to smile (Figure 1).[1-3]

FNP can be idiopathic or caused by a wide array of conditions including infections, inflammation, tumors, trauma and vascular events.[1-3, 5-7] Bell's palsy and facial nerve trauma represent the first- and second-most common etiologies, respectively.[2] Facial nerve palsy caused by herpes zoster infection is referred to as Ramsay Hunt syndrome (RHS) and is the third-most common cause of peripheral facial nerve palsy. RHS is characterized by peripheral FNP in conjunction with a vesicular rash on the ear or in the mouth. Patients often complain of severe pain and may suffer from postherpetic neuralgia upon resolution of the palsy.[5] Another infectious etiology that should be routinely considered in seventh nerve palsy is Lyme disease. The initial infection typically manifests as a "bull's eye" rash at the site of the tick bite. Facial paralysis may occur during the next stage of disseminated infection, which occurs days to weeks after rash onset and often consists of nonspecific flu-like symptoms and joint pain.[8] Other less common infectious causes of facial nerve palsy include HIV, tuberculosis, leprosy, mumps, polio and cat scratch disease.[1,3,6,7] Coronavirus infectious disease-19 (COVID-19) has also been purported as a possible viral etiology for FNP, although the link remains tenuous.[9] Another study found a higher incidence of FNP (82 per 100,000) within 8 weeks of an initial COVID-19 diagnosis compared to that in individuals who were vaccinated against COVID-19.[10] Similarly, FNP has also been reported as a rare complication following COVID-19 vaccination. [11]

Neoplastic and infiltrative processes can involve the seventh cranial nerve in the cerebellopontine angle, within the fallopian canal and in the parotid gland. Of these, parotid malignancies are the most common cause of peripheral FNP.[12] The hallmark of FNP caused by parotid gland malignancies is an insidious onset, progressive nature and lack of spontaneous recovery.[12] Rarely, FNP secondary to parotid malignancy can present acutely, making the differential diagnosis even more challenging.[12]

A traumatic and iatrogenic etiology should be relatively simple to rule out with a thorough case history. Temporal bone fractures and lacerating injuries can damage the facial nerve.[2] FNP is also a frequent complication of surgical intervention to remove acoustic neuromas[13] and after parotidectomies.[14]

Bell's palsy, also referred to as idiopathic peripheral facial nerve palsy, is the most common presentation of FNP, and accounts for up to 75% of the cases.[1] Morales and colleagues conducted a large population-based study on the incidence of Bell's palsy in adults.[15] During the 2001-2012 study period, 14,460 cases of Bell's palsy were identified and the overall incidence was calculated to be 37.7 per 100,000, which is considerably higher than previous estimates.[15] A more recent longitudinal study in Korea, conducted from 2008 to 2018, identified 156,211 cases of Bell's palsy, which were specifically treated with steroids, and reported an increase in incidence from 23.0 per 100,000 in 2008 to 30.8 per 100,000 in 2018.[7] As Bell's palsy remains a diagnosis of exclusion, the astute clinician must be able to differentiate Bell's palsy from some of the aforementioned less common but potentially more serious causes.

Bell's palsy typically presents with sudden-onset unilateral facial nerve paresis with the possible progression of symptoms over the first week.[2] Bell's palsy has an excellent prognosis for recovery of facial nerve function even without treatment: approximately 70% of affected individuals achieve complete, spontaneous recovery by 6 months, with recovery often beginning 3-4 weeks after the onset of symptoms.[2,6] A younger age is associated with a better prognosis for complete recovery.[2] Failure to show signs of improvement or persistence of facial nerve paresis beyond the six-month point should raise suspicion regarding an etiology other than Bell's palsy, and clinicians should initiate appropriate testing, whether independently or via a referral to a specialist, such as an otolaryngologist or neurologist. Generally, recurrence is infrequent and occurs years later.[1] In a 2019 meta-analysis, Dong and colleagues estimated the mean incidence of recurrent Bell's palsy to be 6.5% with lower recovery rates in recurrent cases compared to primary Bell's palsy; laterality of recurrent Bell's palsy location compared to the initial occurrence did not affect the prognosis for recovery.[16] In contrast, Lee and Kim found a much lower recurrence rate of 1.5% in a large Korean population over the course of a decade, with a mean interval of 145 weeks between the first and second occurrences.[7] Since recurrence of idiopathic palsies is so uncommon, radiologic and neurological imag-

ing is typically indicated in recurrent cases to rule out any underlying neoplastic or systemic etiology for the palsy. Although most cases of Bell's palsy resolve without permanent sequelae, there is still a significant risk of morbidity from ophthalmic complications including persistent corneal dryness, scarring, ulceration and vision loss.[1,2]

Long-term sequelae of seventh nerve palsy include permanent facial dysfunction as well as synkinesis, which causes involuntary contracture of the facial muscles.[6] Synkinesis results from misdirection of facial nerve fibers during the course of FNP recovery.[7] Signs include tearing while eating, narrowing of the palpebral fissure while smiling, and contracture of the mouth during eyelid closure.[4]

The primary goals of treatment/management of seventh nerve palsies are twofold: identification and management of the secondary cause of the palsy, if present, and protection of the cornea from vision-threatening sequelae due to lagophthalmos, paralytic ectropion and dryness.

Supportive treatment for all cases of FNP is essentially identical. Acute care focuses on protecting the cornea from exposure keratitis secondary to incomplete lid closure.[1] The presence of Bell's phenomenon, an upward rolling of the eyeball with attempted lid closure, can serve as a protective mechanism to decrease corneal exposure.[4] Dysfunction of the seventh nerve may also cause decreased tear production and loss of Bell's phenomenon, which exacerbates the impact of the corneal exposure.[17] Common treatment modalities include intensive lubrication with preservative-free artificial tear drops during daytime and a lubricating ointment for nighttime use.[2] Antibiotic ointment can be used for lubrication and to prevent infection. Other beneficial measures include eyelid-taping at night, moisture goggles and punctal occlusion.[2] Short-term soft bandage contact lens with adjunct antibiotic prophylaxis may be helpful in more severe cases of keratitis. Scleral contact lenses are another tool for protecting the ocular surface that shows promise in cases of severe exposure keratitis.[18]

Careful monitoring of the ocular surface status is recommended in all cases. When the more conservative interventions already discussed 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.[17] In some cases, a reversible tarsorrhaphy may provide additional relief with the disadvantages of poor cosmesis and reduced peripheral vision.[2,6] The use of botulinum toxin injections has also been advocated, both as a means to induce protective ptosis during the acute phase of FNP and as a way to relieve the synkinesis and facial spasms that may persist upon incomplete recovery from the palsy.[1,2] Thus, a referral for an oculoplastics consultation may be warranted in recalcitrant cases. Currently, there is no conclusive evidence that physical therapies, including electrostimulation, acupuncture and facial exercises are beneficial for the management of FNP.[19]

Medical management in cases of Bell's palsy remains controversial. Due to the purported infectious and inflammatory pathophysiology of Bell's palsy, both antiviral and corticosteroid oral treatment regimens have been proposed. The most recent Cochrane meta-analysis to address the use of systemic corticosteroids in Bell's palsy was updated in 2016.[20] The authors report a significant benefit with the use of oral corticosteroids in reducing the rate of incomplete recovery of Bell's palsy at six months. Steroid use had minimal effects on the rates of cosmetically disabling sequelae, but there was a significant reduction in motor synkinesis in the steroid-treated group. Steroids were found to be safe and well tolerated.[20] Similarly, a Cochrane meta-analysis updated in 2019 on the use of antiviral drugs in Bell's palsy found that "the combination of antivirals and corticosteroids may have little to no effect on rates of incomplete recovery in comparison to corticosteroids alone in Bell's palsy."[21] At present, the only evidence-based treatment protocol for Bell's palsy involves oral corticosteroids, initiated within 3 days of the onset of Bell's palsy.[20-22]

CONCLUSION

Idiopathic facial nerve palsy, or Bell's palsy, remains a diagnosis of exclusion. As demonstrated in this case report, careful monitoring is needed to ensure that both the facial paresis and ophthalmic sequalae improve within the expected time frame. Lack of palsy resolution by the six-month mark, the presence of other neurological symptoms at onset, or recurrence even years later should prompt a thorough neurological workup and imaging studies to rule out other potential causes of FNP. Early treatment with oral steroids remains the most effective systemic treatment modality for Bell's palsy while the management of ocular complications should be at the forefront of therapy in all cases of FNP, regardless of etiology. While initial aggressive topical lubrication and lid-taping are the mainstays of ophthalmic treatment, if the corneal status fails to improve or deteriorates, surgical treatment should be considered. ●

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REFERENCES

1. Finsterer J. Management of peripheral facial nerve palsy. Eur Arch Otorhinolaryngol. 2008 Jul;265(7):743-52. doi: 10.1007/s00405-008-0646-4. Epub 2008 Mar 27. PMID: 18368417; PMCID: PMC2440925.
2. Rahman I, Sadiq SA. Ophthalmic management of facial nerve palsy: a review. Surv Ophthalmol. 2007;52(2):121-144. doi:10.1016/j.survophthal.2006.12.009
3. Gilchrist JM. Seventh cranial neuropathy. Semin Neurol. 2009;29(1):5-13. doi:10.1055/s-0028-1124018
4. Bhatti MT, Schiffman JS, Pass AF, Tang RA. Neuro-ophthalmologic complications and manifestations of upper and lower motor neuron facial paresis. Curr Neurol Neurosci Rep. 2010;10(6):448-458. doi:10.1007/s11910-010-0143-1
5. Kline LB. Neuro-ophthalmology Chapter 11 In: Kline LB. 2009 2010 Basic and Clinical Science Course, Section 5 Neuro-Ophthalmology. San Francisco: American Academy of Ophthalmology; 2009: 283-288.
6. Hazin R, Azizzadeh B, Bhatti MT. Medical and surgical management of facial nerve palsy. Curr Opin Ophthalmol. 2009;20(6):440-450. doi:10.1097/ICU.0b013e3283313cbf
7. Lee JS, Kim YH. Epidemiological trends of Bell's palsy treated with steroids in Korea between 2008 and 2018. Muscle Nerve. 2021 Jun;63(6):845-851. doi: 10.1002/mus.27213. Epub 2021 Mar 13. PMID: 33651414.
8. Smith IS, Rechlin DP. Delayed diagnosis of neuroborreliosis presenting as bell palsy and meningitis. J Am Osteopath Assoc. 2010;110(8):441-444.
9. Gupta S, Jawanda MK, Taneja N, Taneja T. A systematic review of Bell's Palsy as the only major neurological manifestation in COVID-19 patients. J Clin Neurosci. 2021;90:284-292. doi:10.1016/j.jocn.2021.06.016
10. Tamaki A, Cabrera CI, Li S, et al. Incidence of Bell Palsy in Patients With COVID-19. JAMA Otolaryngol Head Neck Surg. 2021 Aug 1;147(8):767-768. doi: 10.1001/jamaoto.2021.1266. Erratum in: JAMA Otolaryngol Head Neck Surg. 2021 Aug 1;147(8):775. PMID: 34165518; PMCID: PMC8227441.
11. Garg RK, Paliwal VK. Spectrum of neurological complications following COVID-19 vaccination. Neurol Sci. 2022;43(1):3-40. doi:10.1007/s10072-021-05662-9
12. Quesnel AM, Lindsay RW, Hadlock TA. When the bell tolls on Bell's palsy: finding occult malignancy in acute-onset facial paralysis. Am J Otolaryngol. 2010;31(5):339-342. doi:10.1016/j.amjoto.2009.04.003.
13. Veronezi RJ, Fernandes YB, Borges G, Ramina R. Long-term facial nerve clinical evaluation following vestibular schwannoma surgery. Arq Neuropsiquiatr. 2008;66(2A):194-198. doi:10.1590/s0004-282x2008000200010.
14. Huang CC, Tseng FY, Chen ZC, et al. Malignant parotid tumor and facial palsy. Otolaryngol Head Neck Surg. 2007;136(5):778-782. doi:10.1016/j.otohns.2006.10.043.
15. Morales DR, Donnan PT, Daly F, Staa TV, Sullivan FM. Impact of clinical trial findings on Bell's palsy management in general practice in the UK 2001-2012: interrupted time series regression analysis. BMJ Open. 2013;3(7):e003121. Published 2013 Jul 16. doi:10.1136/bmjopen-2013-003121
16. Dong SH, Jung AR, Jung J, et al. Recurrent Bell's palsy. Clin Otolaryngol. 2019 May;44(3):305-312. doi: 10.1111/coa.13293. Epub 2019 Feb 22. PMID: 30672667.
17. Amer TA, El-Minawi HM, El-Shazly MI. Low-level versus high-level placement of gold plates in the upper eyelid in patients with facial palsy. Clin Ophthalmol. 2011;5:891-895. doi:10.2147/OPTH.S21491.
18. Gire A, Kwok A, Marx DP. PROSE treatment for lagophthalmos and exposure keratopathy. Ophthalmic Plast Reconstr Surg. 2013;29(2):e38-e40. doi:10.1097/IOP.0b013e3182674069
19. Teixeira LJ, Valbuza JS, Prado GF. Physical therapy for Bell's palsy (idiopathic facial paralysis). Cochrane Database Syst Rev. 2011 Dec 7;(12):CD006283. doi: 10.1002/14651858.CD006283.pub3. PMID: 22161401.
20. Madhok VB, Gagyor I, Daly F, et al. Corticosteroids for Bell's palsy (idiopathic facial paralysis). Cochrane Database Syst Rev. 2016;7(7):CD001942. Published 2016 Jul 18. doi:10.1002/14651858.CD001942.pub5
21. Gagyor I, Madhok VB, Daly F, Sullivan F. Antiviral treatment for Bell's palsy (idiopathic facial paralysis). Cochrane Database Syst Rev. 2019;9(9):CD001869. Published 2019 Sep 5. doi:10.1002/14651858.CD001869.pub9
22. Engström M, Berg T, Stjernquist-Desatnik A, et al. Prednisolone and valaciclovir in Bell's palsy: a randomised, double-blind, placebo-controlled, multicentre trial. Lancet Neurol. 2008;7(11):993-1000. doi:10.1016/S1474-4422(08)70221-7.