The ROYAL COLLEGE of OPHTHALMOLOGISTS

focus

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Update on Facial Nerve Palsy: Diagnosis and management of ocular complications

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Introduction

The facial nerve carries the motor supply to the muscles of facial expression (frontalis, orbicularis oculi, buccinators and orbicularis oris), stapedius, parasympathetic supply to the lacrimal and submandibular glands, and sensory input from the anterior two thirds of the tongue. Paralysis of the nerve therefore causes facial droop, drooling, hyperacusis, altered taste, otalgia and speech articulation problems[1], creating significant functional disability and negatively impacting quality of life. This update examines the ophthalmic consequences of facial nerve palsy (FNP), how to accurately diagnose the condition and create an appropriate management plan.

Ophthalmic features of FNP

Orbicularis oculi is the main protractor of the eyelids. When affected by FNP it results in incomplete lid closure, reduced blink frequency and amplitude and therefore excessive tear evaporation and subsequent corneal desiccation. Weakening of the eyelid protractor results in the unopposed action of the eyelid retractors (levator palpebrae superioris and Muller's muscle in the upper lid and inferior tarsal muscle in the lower lid), leading to upper and lower eyelid retraction and widening of the vertical palpebral fissure. Reduced resting tone in orbicularis and the muscles of the lower face leads to ectropion due to gravitational pull on the lower eyelid. These changes all result in increased exposure of the ocular surface. Dryness of the ocular surface may be confounded by injury to the intracanalicular or intracranial segment of the facial nerve, compromising branches of the sympathetic fibres to the Vidian nerve, impairing tear production from the lacrimal gland[2]. Furthermore, cranial nerve palsies do not always occur independently. The proximity of the facial nerve to the trigeminal nerve means that compressive lesions may result in palsies of both nerves. Corneal epithelial damage, ulceration and perforation are more common if there is also loss of corneal sensation (originating from the trigeminal nerve).

Patients with FNP are at risk of developing corneal epithelial defects, ulcers, perforations and even endophthalmitis. Accurate diagnosis and timely management through conservative, medical and surgical means can reduce the chance of loss of vision in the affected eye.

Diagnosis

When an individual presents with facial weakness it is essential for the treating clinical to establish a diagnosis. At the forefront of the diagnostic process must be determining whether an upper motor neurone (UMN) or lower motor neurone (LMN) lesion is causative. Classical neurological teaching denotes a bilateral innervation of the facial nuclei supplying the forehead. Therefore, forehead movement is preserved in UMN lesions, whereas LMN lesions cause a weakness of the entire side of the face, including the forehead. When an UMN is suspected then a cerebrovascular event (CVE) must be ruled out with appropriate imaging to assess the need for thrombolysis.

Once an UMN cause for the FNP has been excluded, a detailed examination of the ears, mastoid region, oral cavity, eyes, scalp, and parotid glands is required to look for specific signs that may aid diagnosis. **Table 1** highlights the majority (although not an exhaustive list) of common causes of LMN FNP and the key examination findings. Percentages are based on combined epidemiological data on LMN FNP of over 6000 patients[3].

Table 1

Causes of lower motor neurone FNP	Examination findings
Idiopathic (59-70%) Bell's palsy	• Diagnosis of exclusion – none of the below findings should be noted
Traumatic (10-23%) Temporal bone fracture Iatrogenic (post-surgical) Facial trauma Birth canal trauma	• Periorbital or mastoid bruising, blood in the ear canal and haemotympanum is suggestive of skull base fracture • Recent mastoid, parotid or submandibular surgery
Viral (4.5-7%) Herpes Zoster Virus (Ramsey Hunt Syndrome) Mumps HIV	• Small vesicular eruptions affecting the tympanic membrane, ear canal, external pinna or oral cavity is suggestive of herpetic infection
Neoplastic (2-2.5%) Acoustic neuroma Parotid malignancy	• Acoustic neuroma may present with unilateral gradual sensorineural hearing loss and reduced corneal reflex • Parotid lump associated with regional malignancy, pain and gradual onset FNP is highly suggestive of parotid malignancy
Other (3-5%) Acute or chronic otitis media Malignant otitis externa Acute mastoiditis Lyme disease	• Assess in external and internal ear canal for signs of otitis media/ externa • Tender swelling in the mastoid area with middle ear inflammation suggests acute mastoiditis • Erythematous 'bullseye' lesions on limbs and trunk with arthralgia indicates potential Lyme disease

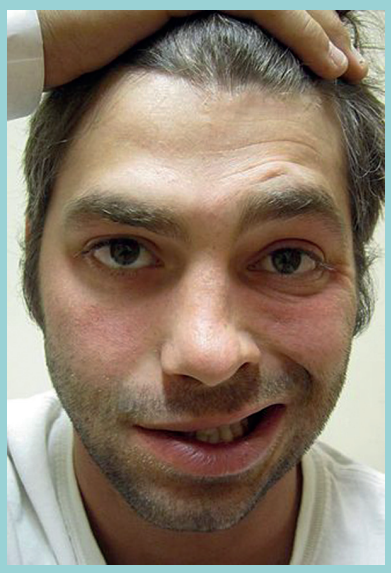
Photo credit: James Heliman MD

As mentioned in **Table 1**, idiopathic FNP remains a diagnosis of exclusion. Studies suggest that the condition develops over 24-48 hours[4]. Over 60% of cases are associated with mild post-auricular pain. Neither ethnicity nor sex is related to the incidence of the disease, but diabetes is associated with up to 10% of cases and the idiopathic FNP occurs more often in the last trimester of pregnancy. Presentation with incomplete FNP is a reassuring prognostic sign, indicating a 94% chance of full recovery, as opposed to 61% in those with complete paralysis[5].

Management

Management of FNP depends on the working diagnosis and primarily aims to treat the underlying cause, be this surgical resection of tumour, thrombolysis of a stroke or anti-viral treatment of herpes zoster infection. Ophthalmic consultation is recommended in the setting of facial nerve palsy when ocular symptoms are present or there is concern regarding the corneal sensation.

In this section, we will focus primarily on the management of idiopathic FNP, which can be divided into *conservative, medical* and *surgical* treatment options, however the management of the ocular sequelae is largely transferable, irrespective of cause.

Conservative management aims to lubricate the ocular surface, retain moisture, improve tear quality and obstruct tear outflow. Facial rehabilitation is also used to try and restore premorbid appearance and function.

- **Lubrication:** Regular lubrication of the ocular surface with the use of artificial tears and thicker gels or ointments remains the mainstay to avoid excessive corneal drying and subsequent epithelial damage
- **Lid Taping:** Taping the eyes shut when sleeping or resting can be an effective measure to retain moisture, alongside humidification goggles or room air humidifiers
- **Tear film enhancement:** The use of regular lid hygiene with hot compresses, omega-3 fatty acids and oral doxycycline tablets can all enhance the quality of the tear film
- **Facial rehabilitation:** Soft tissue mobilisation can improve facial muscle motor control. Realistic functional outcomes include improved ability to smile, eat, drink, speak clearly and blink on the affected side[5]

Medical management of idiopathic FNP remains contentious, however the use of oral steroids +/- oral antivirals has been shown to provide some benefit.

- **Oral steroids:** A Cochrane review found that significantly more patients taking oral steroids recovered complete motor function, compared to those taking placebo, if started within 72 hours after onset. They also had significantly fewer motor synkinesis symptoms[3]
- **Oral antivirals:** The role of anti-viral treatment (acyclovir or valaciclovir) for idiopathic FNP was also investigated. A meta-analysis of combination therapy (anti-viral + corticosteroid) for idiopathic FNP suggested a marginal benefit, only when smaller studies of poor quality were included[3]

Surgical management is withheld for those patients with longstanding functional or cosmetic impairment, or for any patient who is showing signs of significant damage to the ocular surface.

- **Botox ptosis:** Protective ptosis may be induced with botulinum toxin injected into the levator muscle. Onset of ptosis begins commonly 3-4 days following the injection and should last for around 6 weeks
- **Temporary/ Permanent tarsorrhaphy:** Surgical closure of the eyelid margin provides protection of the ocular surface, at the loss of visual acuity. A tarsorrhaphy however is understandably not appropriate in monocular patients
- **Upper eyelid weights:** Gold or platinum weights can be secured to the tarsal plate to induce a mechanical ptosis and improve passive eyelid closure with increased closure during blink
- **Lower lid tightening:** Lower lid malposition/retraction should be treated aggressively. Procedures such as lateral tarsal strip or wedge excision can allow better apposition of the lid to the globe
- **Reanimation:** Over recent years, exciting new developments have occurred within eyelid reanimation. Cross-facial nerve grafting can be utilised if the contralateral facial nerve is intact and functional. Nerve transfer procedures have been described using a variety of donor nerves: hypoglossal, spinal accessory, masseteric branch of the trigeminal nerve and motor branches of the cervical plexus. The most commonly used procedure is the hypoglossal-facial transfer

Summary

Management of FNP requires good clinical acumen and an open mind when arriving at a diagnosis and management plan. The mainstay of an ophthalmologist's role in the condition is to promote the corneal health with conservative management. However surgical intervention and particularly the exciting development of reanimation has the potential to greatly improve the patients functional and cosmetic outcome[6].

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