ON THE MANAGEMENT OF PARALYTIC LAGOPHTHALMOS

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Abstract

Lagophthalmos secondary to facial nerve damage is a serious complication that can lead to exposure to keratopathy, ulceration and even blindness. For definitive treatment and eye rehabilitation, several surgical strategies are available (tarsorrhaphy, palpebral springs, eyelid lengthening, upper lid weights, or dynamic procedures), an appropriate management depending on the severity and duration of recovery.

Keywords: facial palsy, lagophthalmos, surgical treatment.

1. INTRODUCTION

Temporary or permanent facial palsy (FP) is a common result of nerve damage after head and neck surgical interventions, especially after parotid gland tumour removal or skin malignancies. The postoperative facial nerve disfunction is not only a cosmetic, but a functional problem, as well. Depending on the location of the injuries to the nerve trunk or branches, important functions, such as facial expression, eye protection, eating, drinking, and speech can be affected. Paralytic lagophthalmos subsequent to facial nerve damage is a clinical condition characterized by loss of blinking and prolonged corneal exposure which can lead to keratopathy, abrasion, ulceration and even blindness [1]. The maxillofacial surgeon plays an important role in the diagnosis and treatment of facial nerve palsy sequelae. The management of paralytic lagophthalmos depends on many factors, such as the underlying cause, severity of the nerve injury and of the ocular symptoms and expected duration of recovery.

2. ETIOLOGY

The most common cause for FNP reported in literature is idiopathic Bell’s palsy, which is a benign inflammatory condition thought to be caused by Herpes virus infection (Table I) [2]. Another viral cause of FNP is Ramsay Hunt Syndrome, determined by the reactivation of the latent Varicella Zoster Virus in the facial nerve. Other etiologies include inflammation of the nerve due to infection, the most frequently reported determining factor in this case being Lyme disease, which has a high rate of bilateral involvement, unlike Bell’s Palsy [3]. Other conditions that may present bilateral FP include autoimmune disorders (Guillain-Barre Syndrome, Melkersson-Rosenthal Syndrome) or sarcoidosis. FNP may also result from tumours through direct compression or infiltration of the nerve. The most common tumour causing facial paralysis is the acoustic schwannoma and parotid gland malignancies [4]. Also, craniofacial trauma which usually involves a temporal bone fracture can cause severe nerve injury. Iatrogenic nerve damage has been mostly reported after large facial surgeries, including cervicofacial rhytidectomy and parotid gland surgery.

Disfunction of the facial nerve is a common and typical complication of parotid gland surgery, even though its anatomic continuity is preserved. The deficit of the nerve function may be total or partial. According to data from published literature, postoperative transient facial nerve disfunction occurs up to 46 % of cases, whereas permanent damage is less common, occurring only in 1.9–3.9 % of cases [5]. Recovery after transient FP following is expected in most cases in the first 6 months after surgery [6]. The incidence of nerve palsy is higher in total parotidectomy than in superficial parotidectomy, which may be
related to stretch injury or as a result of surgical trauma. Postoperative facial nerve disfunction is not only a cosmetic, but a functional problem, as well. Depending on the location of the injuries to the nerve trunk or branches, important functions, such as facial expression, eye protection, eating, drinking, and even speech can be affected.

**Table I. Aetiology of facial palsy**

<table>
<thead>
<tr>
<th>Category</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic</td>
<td>Bell’s Palsy</td>
</tr>
<tr>
<td>Infectious</td>
<td>Ramsay Hunt Syndrome, Lyme disease, Epstein-Barr virus, tuberculcus chronic middle ear infection</td>
</tr>
<tr>
<td>Autoimmune disorders</td>
<td>Guillain-Barre Syndrome, Melkersson-Rosenthal Syndrome, sarcoidosis</td>
</tr>
<tr>
<td>Traumatic</td>
<td>Temporal bone fracture, soft tissue injuries</td>
</tr>
<tr>
<td>Tumours</td>
<td>Acoustic neurinoma, parotid gland malignancies, cerebellopontine angle lesions, facial nerve schwannomas, lymphomas, nasopharyngeal carcinomas, meningiomas, temporal bone neoplasm, leukaemia</td>
</tr>
<tr>
<td>Iatrogenic</td>
<td>Maxillofacial surgery procedures, otologic procedures, forceps delivery</td>
</tr>
<tr>
<td>Other</td>
<td>Multiple sclerosis, myasthenia gravis, Moebius syndrome, Diabetes, hyperthyroidism, acute porphyria, carbon monoxide toxicity, vitamin A deficiency, ethylene glycol ingestion</td>
</tr>
</tbody>
</table>

3. MANAGEMENT

In cases where spontaneous recovery is expected, temporary measures are indicated, like artificial tears, lid taping overnight, viscous ointments or soft contact lens [7,8]. Also, botulinum toxin injection protects the cornea from drying by temporarily paralyzing the levator palpebrae superioris. The effect lasts for a medium period of 46 days and it is considered to be a good alternative for postoperative FP, when the facial nerve is intact [9]. Another nonsurgical option is external eyelid weights, which can be fixed to the pretarsal skin with a double-sided adhesive tape [10]. Temporary tarsorrhaphy can be achieved with a simple suture or cyanoacrylate glue, but the main disadvantage consists in the reduction of the vision field.

If the recovery is absent or incomplete, a permanent surgical solution is required, and numerous techniques have been proposed, including permanent tarsorrhaphy, palpebral springs, eyelid lengthening, upper lid weights, or dynamic procedures, such as temporalis muscle transfer or cross-facial nerve grafting. The degree of inferior scleral show and the ability to achieve complete eye closure should be documented prior to any surgery. If there is no evidence of scleral show, only the upper lid needs to be addressed while, if significant scleral show occurs, both lids need to be treated [11].

In the past years, permanent tarsorrhaphy has been considered the first-choice treatment for lagophthalmos, because it ensures effective eye closure in 70-80% of cases [12]. Contrary to its name, it is not irreversible, and it can be considered a temporary procedure while waiting for a definitive treatment. The procedure is performed under local anaesthesia and consists in de-epithelization of both upper and lower eyelid margins, followed by mattress suture.

The main disadvantage is that it is aesthetically unpleasant and the risk of deformities and scarring after reversal is high. However, it is a treatment option for patients who cannot tolerate facial reanimation or with poor overall general health prognosis [4].
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Levator lengthening is indicated in young patients with no history of lid surgery. As a general principle, the width of scleral exposure is equal to the amount with which the levator needs to be lengthened - typically 5 mm [11]. Currently, upper lid loading is the most common technique used for treating long-term paralytic lagophthalmos. Although it is a simple procedure, several complications may occur, such as migration, extrusion, hypersensitivity reactions, foreign-body reactions, ptosis, trichiasis and astigmatism [13-15].

For many years, gold lid implants have been used due to their high density and malleability [16,17]. More recently, platinum was proposed for lid loading because it is denser than gold, which allows 10% smaller implants than the gold ones. Also, platinum is less reactive, reducing the risk of extrusion and adverse reactions to the metal [18]. There are several techniques described for locating the implant to make it less visible: fixation either to the tarsal plate or to the levator aponeurosis, including pretarsal, septal, postseptal and intraorbital placement [10,19]. Surgery can be performed under local anaesthesia, while preoperatively it is recommended that test weights should be attached to the upper lid, in order to determine the lightest ones. The most commonly selected weights are 1 g and 1.2 g and, for best results, they should be positioned at least 4 mm above the lid margin [11]. Regardless of the material or technique chosen, and due to the fact that the eyelid is pulled down by the gravity force, it may not close when the patient is in supine position. Thus, this residual lagophthalmos may leave patients with keratopathy or corneal abrasion. Studies of gold implants have shown improvement in lagophthalmos from 5.5-7.4 mm preoperatively to 1.2-2.3 mm postoperatively, the revision rate reported being around 20%, due to poor eyelid contour, allergy and extrusion [19-22]. In order to prevent extrusion, a few methods are available for implant covering, such as packing the weight with cartilage, temporal galeal fascia, human pericardium or orbital fat [23-25], but some authors believe that a proper fixation to the tarsal area and rounded edges of the implant are sufficient for preventing extrusion.

Palpebral spring is usually indicated in patients with significant lagophthalmos for at least 6 months, who are not expected to recover or who have failed conservative therapy or gold implants placement [4]. It consists of a wire implanted and secured to the superior orbital rim and pretarsal area. When the eye is open by contraction of the levator muscle, the spring is compressed and when the levator is relaxed, the spring expands and forces the eye to close. The advantage over the lid weights is that it does not rely on gravity, so it is functional in any position: upright, supine and even inverted. Also, studies show that palpebral springs are more effective in achieving blink and eye closure than gold implants [4,26]. However, the main disadvantages are the duration of surgery and the possible complications, especially the extrusion rate, reported to be 19% [26].

Dynamic correction of paralytic lagophthalmos frequently involves transfer of the temporalis muscle which can provide strong eyelid closure for a long period of time. A 1.5 cm strip of temporalis muscle is detached, rotated and secured to the medial canthal ligament. Closing of jaws activates muscle transfer and therefore blinking, keeping the cornea lubricated [27]. The only procedures than can restore spontaneous blink are cross-face facial nerve grafting, followed by a neurorrhaphy to the facial nerve branch for the orbicularis oculi muscle, in cases of recent paralysis [28], or to a motor nerve of a free muscular flap (platysma) in cases of complete palsy longer than 24 months [27]. However, these operations are complex and the results unpredictable.

4. CONCLUSIONS

Lagophthalmos is a serious complication frequently encountered in maxillofacial surgery, especially after parotid gland surgeries. Postoperative facial nerve disfunction is not only a cosmetic, but a functional problem, as well.

The surgical treatment of lagophthalmos is complex, so that an efficient management in early stages must be employed.
References


