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Chapter 10

Traumatic Neuroma of the Infraorbital Nerve Subsequent to Inferomedial Orbital Decompression for Graves’ Orbitopathy

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Abstract

**Purpose:** To present and discuss the occurrence of a traumatic neuroma subsequent to inferomedial orbital decompression surgery in Graves’ orbitopathy.

**Method:** Case report.

**Results:** Approximately 1 month after surgery, a patient who underwent bilateral rehabilitative inferomedial orbital decompression developed a mass with clinical and radiologic characteristics compatible with a traumatic neuroma of the left infraorbital nerve. The lesion, which was thought to be the result of unnoticed nerve trauma at the time of surgical dissection of the infraorbital canal, remained stable in shape and other imaging characteristics during the 39-month follow-up period. Symptoms of trigeminal neuralgia could be only partially controlled with medical therapy (oral pregabalin 75 mg 3 times daily).

**Conclusions:** The second branch of the trigeminal nerve may be damaged in the course of orbital floor removal decompression for Graves’ orbitopathy. This may potentially induce the formation of traumatic or amputation neuromas. Such lesions should be included in the potential complications of decompressions when counseling patients about to undergo this type of surgery, as they are difficult to treat and may cause persistent and disabling pain.
Introduction

Traumatic neuromas may be due to chronic nerve irritation or trauma that partially avulses or totally disrupts the nerve. In the case of chronic irritation, an undisrupted nerve gives rise to a focal fusiform swelling often referred to as “spindle neuroma.” Different degrees of nerve disruption and subsequent external axon growth, which represents an attempt at natural repair, leads to the formation of masses referred to as “lateral or terminal, traumatic neuromas,” the latter also referred to as “amputation neuromas.”

Traumatic neuromas may cause persistent pain and have been described in several anatomic locations including the orbit after enucleation. Orbital decompression surgery is a currently accepted, effective, and generally safe treatment for Graves’ orbitopathy. When decompression surgery entails removal of the orbital floor, the infraorbital nerve may be damaged by surgical maneuvers, by bone edges or fragments, or may be stretched by the soft orbital tissues which prolapse into the maxillary antrum. This generally causes hypoesthesia, rarely anesthesia or pain in the sensory distribution area of this nerve branch.

Herein we present and discuss a patient with Graves’ orbitopathy presenting with clinical and radiologic evidence of traumatic neuroma of the infraorbital nerve starting a few weeks after rehabilitative inferomedial orbital decompression, and who has been followed up at our department for 39 months afterwards.

We are unaware of previous reports that link traumatic neuromas of the infraorbital nerve to orbital decompression.

Case report

A 45-year-old Caucasian woman with stable inactive Graves’ orbitopathy for more than 5 years, on levothyroxine replacement therapy for 4 years following $^{131}$, was admitted for bilateral inferomedial rehabilitative orbital decompression in June 2005. On admission, she had best corrected visual acuity (pinhole) of 20/20 bilaterally, bilateral slight increase of the eyelid aperture with 3 mm upper lid retraction, moderate periorbital edema, consistent retroocular tension, and Hertel readings of 23 mm bilaterally, and did not have diplopia.

Decompression was carried out uneventfully by an anterior trans-inferior-fornix approach and included the complete removal of the orbital floor. The immediate postoperative period was unremarkable. The patient left the hospital 2 days after surgery without any medication.
Figure 1. Traumatic neuroma affecting the left infraorbital nerve in a patient with Graves' orbitopathy who had undergone bilateral inferomedial bone orbital decompression. (Top) Coronal T1-weighted magnetic resonance image demonstrates a low-signal-intensity mass (arrow). The encased infraorbital nerve can hardly be distinguished. (Bottom) Coronal contrast-enhanced fat-saturated T1-weighted magnetic resonance image shows enhancement of the mass encasing the infraorbital nerve (arrow).

except for artificial tears. As expected, moderate bilateral hypoesthesia of the areas innervated by the infraorbital nerve was present.

At the first postoperative control, carried out 1 month after surgery, exophthalmos had decreased to 17 mm bilaterally, periorbital aedema and retroocular tension had disappeared, and eyelid aperture had normalized. The patient did not have diplopia and best-corrected visual acuity was unchanged. For a few days prior to the examination, the patient had been experiencing symptoms compatible with trigeminal neuralgia in the distribution of the second left branch. An orbital computed tomography (CT) scan showed a round mass approximately 12 mm in diameter involving the left infraorbital nerve at the level of the inferior orbital fissure. The lesion was not present on the predecompression CT scan. Signal intensity, shape, and relation of the lesion with the parent nerve on postdecompression CT scan and on the follow-up magnetic resonance images performed afterwards were compatible with that described for traumatic neuromas. As in the case we described, traumatic neuromas have been reported to become evident at approximately 1 month after acute trauma. The patient we described started presenting symptoms of trigeminal neuralgia and evidence of the lesion on imaging a few weeks after surgery, and this does not permit us to include in the possible pathogenesis "chronic" causative mechanisms such as nerve irritation due to aggressive bone edges left at the level of the anterior margin of the inferior orbital fissure. Other trauma to the nerve such as stretching due to the mechanical action of the soft orbital tissues prolapsing into the maxillary sinus,
radiologic characteristics of the lesion remained constant until September 2008, when the patient attended her last follow-up examination. When the lesion was detected, the patient refused systemic glucocorticoids and truncular anesthesia of the infraorbital nerve. Persistent trigeminal neuralgia did not respond to medical treatment with oral gabapentin 600 mg 2 to 3 times daily, but could be partially controlled by oral pregabalin 75 mg 3 times daily until the present.

Discussion
Orbital decompression is mostly achieved by means of osteotomies which involve the lateral and the medial orbital walls, the orbital floor, and when necessary fat is removed possibly from the inferolateral orbital quadrant. The most effective and safest sequence of osteotomies and lipectomies to be used to gradually implement the effects of decompression surgery continues to be debated. Although orbital floor removal in course of orbital decompression is currently not favored in North America, a recent prospective survey of the European Group on Graves’ Orbitopathy (EUGOGO) showed that inferomedial bone decompression is still a widely used procedure in Europe. When clinical history and symptoms suggest the presence of a traumatic neuroma, the diagnosis can be further supported by radiologic imaging evidence of an abnormal bulbous appearance at the proximal end of or along a nerve which was respectively transected or damaged. CT and magnetic resonance imaging are optimal in detecting the location and shape of the lesion and its relation with the parent nerve, while their nonspecific signal intensity and variable enhancement after administration of contrast are less important for the diagnosis. Although we did not perform any confirmatory histopathology, in all likelihood the lesion we reported represents a traumatic neuroma, and we regard an unnoticed iatrogenic injury at the time of surgery as its most probable cause. As in the case we described, traumatic neuromas have been reported to become evident at approximately 1 month after acute trauma. The patient we described started presenting symptoms of trigeminal neuralgia and evidence of the lesion on imaging a few weeks after surgery, and this does not permit us to include in the possible pathogenesis “chronic” causative mechanisms such as nerve irritation due to aggressive bone edges left at the level of the anterior margin of the inferior orbital fissure. Other trauma to the nerve such as stretching due to the mechanical action of the soft orbital tissues prolapsing into the maxillary sinus,
which normally occurs after floor removal orbital decompression, are also unlikely to have been the cause of the lesion.

Traumatic neuromas of the infraorbital nerve, in fact, have never been described before, despite the elevated number of this type of surgery performed and reported in published series up until now. Although the rapid onset nature of the orbital mass might have been suggestive of an etiology ranging from inflammation to malignancy, with the patient’s consent, we elected not to perform incisional or excisional biopsies and histopathology on the lesion, but only to treat it medically and to closely follow it up with imaging. This choice, of course, implied accepting a diagnosis suggestive of traumatic neuroma and some risks, but surgery would have not been without risk either. Although medical therapy could offer only a partial control of the symptoms, we did not attempt to treat the lesion more aggressively, as more invasive treatment alternatives including intralesional injections of steroid or alcohol, phenolization, cryoapplications, and surgical resection do not offer any superior chance of pain relief.6

Common complications of orbital decompression are consecutive strabismus, infraorbital hypoesthesia and sinusitis, lower lid entropion, and eyeball dystopia, while leakage of cerebrospinal fluid, infections involving the central nervous system, damage to the eye and optic nerve or their vasculature, cerebral vasospasm, ischemia and infarction, and delayed decompression-related reactivation are rare events.7 Among these latter complications, traumatic neuromas deserve a special mention, because they can cause disabling trigeminal neuralgia with limited possibility of treatment. This retrospective case report was carried out following the tenets of the Declaration of Helsinki and of the Code of Conduct of the Dutch Federation of Biomedical Scientific Societies (http://www.federa.org/) as recommended by the Review Board of our institution. The Review Board of our institution declared that for retrospective studies its permission is not needed, nor is patient informed consent necessary if data are collected anonymously from the medical records used by the authors in their everyday practice.
Chapter 10

References


