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ORIGINAL ARTICLE

Transnasal endoscopic orbital decompression: 15-year clinical experience in Southern Taiwan

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Background/Purpose: To present the results and evaluate the efficacy of endoscopic transnasal orbital decompression for dysthyroid orbitopathy.
Methods: Retrospective chart review of patients who underwent endoscopic transnasal orbital decompression from 1996 to 2010 in one institution. We included 42 orbits of 25 patients. Preoperative and postoperative examinations included visual acuity, Hertel exophthalmometry, tonometry, exposure keratitis, and diplopia. The measurements of outcome depend on proptosis reduction, intraocular pressure reduction, and visual acuity improvement of 42 orbits of 25 patients.
Results: There were no surgical complications for the 42 orbital decompressions except one patient experienced cerebrospinal fluid leak during the operation. Mean proptosis reduction in all orbits was 1.93 ± 0.25 (mean $\pm$ standard deviation, $p < 0.01$) after 1 month postoperatively and 2.07 ± 0.29 ($p < 0.01$) after 3 months postoperatively. An average reduction of intraocular pressure was 4.40 ± 0.72 ($p < 0.01$) and 4.38 ± 0.80 ($p < 0.01$) respectively after 1 and 3 months postoperatively. Visual acuity increased from a preoperative average of 0.45 ± 0.34 to 0.66 ± 0.36 and 0.70 ± 0.35 after 1 and 3 months postoperatively. In addition, postoperative relief of exposure keratitis is also noted.
Conclusion: The transnasal orbital decompression procedure has statistically significant improvements in proptosis, intraocular pressure, and visual acuity. The procedure has obvious benefit

Conflicts of interest: The authors have no conflicts of interest relevant to this article.

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in relieving exposure keratitis. Furthermore, there are favorable cosmetic results and rare complications.

Introduction

Dysthyroid orbitopathy is an autoimmune disorder that results in proptosis and compressive optic neuropathy. This is caused by an increase in volume of orbital contents, including the extraocular muscles and orbital fat. However, dysthyroid orbitopathy is often a self-limited disorder. Spontaneous resolution occurs in most cases. Only a minority of patients with severe disease (3—5%) require aggressive treatments.[1,2]

Treatment of dysthyroid orbitopathy consists of high-dose corticosteroids, retrobulbar radiotherapy, immunosuppressive agents, and orbital decompression. Systemic steroids need to be used for months at high doses that have undesirable side effects. In addition, the signs and symptoms may return when the medication is tapered.[3] Furthermore, different dose regimens of oral glucocorticoid use have not systematically been investigated.[4] External beam radiotherapy is effective for optic neuropathy in the acute phase of the disease, but it does not usually provide significant improvement of proptosis. Furthermore, the few available randomized controlled trials on the effects of radioiodine therapy on Graves ophthalmopathy show that a definite proportion of patients (approximately 15%) develop new eye disease or experience progression of preexisting Graves ophthalmopathy within 6 months after radioiodine administration.[4] Decompressive methods have evolved in concert with technical advances in surgical methods. Since 1911, when Dollinger[5] first described surgical orbital decompression, many different techniques and approaches were proposed. These methods involve removing one to four of the bony orbital walls to enable the orbital contents to expand into adjacent spaces. This is associated with improvement in proptosis, corneal exposure, and orbital neuropathy. In 1990, Kennedy et al.[6] first described the endoscopic transnasal medial and inferior wall decompression of thyrotoxic orbitopathy. The ability to perform this surgery via a transnasal route under precise endoscopic guidance without an external scar makes it a more attractive option, especially because of cosmetic reasons. We have adopted endoscopic surgery as our treatment of choice for the past 15 years. This retrospective study reviews and presents the results of 42 orbits of 25 patients who underwent endoscopic transnasal orbital decompression.

Materials and methods

Patients

Between July 1996 and May 2010, endoscopic transnasal orbital decompression was performed on 42 orbits of 25 patients with thyrotoxic orbitopathy at the ENT Department of the Kaohsiung Veterans General Hospital. General anesthesia was used in all cases. There were 14 male and 11 female patients with a mean age of 51.2 years (range, 28 to 81 years, Table 1). All patients had been previously examined at the ophthalmology department and all had received nonsurgical treatment with corticosteroids (10—60 mg prednisolone per day orally or under methylprednisolone pulse therapy, MTP, 250 mg every 6 hours) but in vain (Table 2). Postoperative examination was done at 1- and 3-month intervals. Preoperative and postoperative examinations included visual acuity, Hertel exophthalmometry, tonometry, exposure keratitis, and diplopia. Otolaryngologic assessment included nasal endoscopy to identify any sinus or septal pathology. The presence of sinusitis is a contraindication to endoscopic decompression. Computed tomography (CT) scans or magnetic resonance imaging (MRI) of the orbits and the paranasal sinuses were obtained preoperatively in all patients. This study was approved by the institutional review board of Kaohsiung Veterans General Hospital.

Seventeen patients had bilateral surgery and eight unilateral surgery. There were nine patients who received one-stage bilateral orbital decompressions and eight in whom surgery was performed in two stages (Table 2). The main reason for surgical decompression was severe/acute proptosis in 13 patients, rapidly decreasing vision caused by compressive optic neuropathy in seven patients, both proptosis and decreased vision in four patients, and severe exposure keratitis in one patient. All patients who received two-stage operations had symptoms in each eye that occurred at different times. No patient was operated on for cosmetic reasons (Table 2). In two cases, nasal septoplasty was performed concurrently.

Surgical technique

All patients were operated on according to the technique described by Kennedy et al.[6] The aim is to remove the entire medial wall and part of the floor of the orbit so that the orbital contents can prolapse into the ethmoidal and maxillary cavities.

After induction of general anesthesia, the patient was in the supine position on the operating table with the head slightly elevated to reduce venous pressure at the surgical site. The sinonasal mucosa was decongested and vasoconstricted with topical 4% cocaine by nasal packing. Submucosal injections of 1% lidocaine with epinephrine 1:50,000 were placed along the lateral nasal wall and middle turbinates.[7,8]

An uncinectomy preceded the formation of a generous middle meatal antrostomy to expose the medial and inferior orbital wall. The endoscopic transnasal approach was used with complete anterior and posterior ethmoidectomy. The ethmoid air cells were removed from the agger nasi cells to the face of the sphenoid. The medial wall over the anterior and posterior ethmoid sinuses was removed to the skull base superiorly and posteriorly to the optic nerve canal.