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A Logical Approach to Bony Orbital Decompression Surgery for Thyroid Eye Disease

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Keywords

• Orbital • Decompression • Thyroid • Ophthalmopathy • Lateral • Medial • Dysthyroid • Neuropathy

Key points

- Patients with dysthyroid optic neuropathy initially should be managed with intravenous methyl prednisolone.
- Prompt decompression of the medial orbital wall and floor is indicated if early recovery of visual functions does not occur with medical treatment.
- In cases of mild to moderate degrees of exophthalmos (<~24 mm), deep lateral wall decompression may be sufficient to restore globe position.
- For greater degrees of exophthalmos (~25–29 mm), a balanced 2-wall decompression is preferred, which involves the lateral and medial orbital walls.
- Orbital decompression should be considered where there are congestive or hydraulic features.

INTRODUCTION

Thyroid eye disease (TED) is an idiopathic lymphocytic orbital inflammation that usually occurs in patients with autoimmune thyrotoxicosis (Graves' disease), but can occur in hypothyroid patients or those without any detectable abnormality of thyroid gland function. The underlying autoimmune mechanisms remain uncertain, but activation of orbital fibroblasts is thought to be central, with these cells displaying thyroid0stimulating hormone receptors

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and producing inflammatory molecules, and increasing orbital adipocytes and extracellular matrix [1]. The inflammatory phase of TED mainly affects the extraocular muscles and orbital adipose tissue, with increasing accrual of tissue edema, proinflammatory cytokines, and lymphocytes [2]. This expansion of orbital soft tissues can compromise blood flow at the orbital apex, resulting in optic nerve ischemia and secondary retardation of axonal flow (dysthyroid optic neuropathy [DON]). Where the orbital septum is noncompliant, forward expansion of the soft tissues is limited, and such patients are at greater risk of both DON and congestive orbitopathy. TED is manifest in about 50% of patients with Graves' disease and, although DON can be challenging to treat, fortunately only 5% of all patients with TED develop such severe disease [3].

The typical course of TED is one of an active inflammatory phase lasting 12 to 24 months, followed by an inactive fibrotic stage. Most patients suffer only mild disease. The cornerstones of management include controlling thyroid function, cessation of smoking, and addressing ocular surface inflammation and exposure [4]. In moderate acute disease, defined as severe enough to impair orbital functions, systemic immunosuppression is used to reduce the long-term sequelae of inflammation, with fractioned low-dose orbital radiotherapy advocated as a steroid-sparing measure. Surgical intervention during the active phase of moderate disease rarely is indicated. Suture tarsorrhaphy may be required to control ocular surface exposure and secondary keratitis. Early orbital decompression should be considered in the presence of severe congestive features, where it may limit progression to more severe disease. Severe acute TED poses a major risk of irreversible loss of vision owing to marked exposure keratopathy, hydraulic orbital congestion, and compressive optic neuropathy. When patients do not respond promptly (within 1–2 weeks) to high-dose corticosteroid treatment, urgent decompression of the deep medial orbital wall and floor is indicated, thus, rapidly relieving the congestive component and restoring optic nerve perfusion [5].

When the active inflammatory phase has abated, bony decompression is indicated to improve globe position, followed by surgery to improve ocular alignment–where there is troublesome diplopia–and upper lid retractor recession [6]. Such aesthetic orbital decompression, which also can include the removal of fat, has been used since the 1990s [7]. Various approaches are described, and include removal of the medial, lateral, and inferior bony walls, expanding the orbital space into paranasal sinuses or temporalis fossa [8]. However, despite extensive literature on the subject, no consensus exists on the most efficient intervention; certain authors advocate fat decompression alone, whereas others prefer bony decompression with a graded approach [8–10]. This review presents a practical approach to decompression with reference to surgical techniques.

HISTORY

Historically, orbital surgery has been performed by general, ophthalmic, and neuro-surgeons. Walter Dandy–a highly influential neurosurgeon in the