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Editorial

Orbital Decompression in the Biologic Era of Thyroid Eye Disease

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For decades, the surgical pathway in thyroid eye disease (TED) was relatively familiar. Medical treatment was used to control active inflammation, while orbital decompression was largely reserved for sight-threatening disease or for rehabilitation once the orbit had become inactive and stable. The arrival of targeted therapy, particularly teprotumumab, has made that sequence less predictable. This is not because surgery has lost its place, but because medical treatment can now improve a feature that was once considered predominantly surgical: proptosis. In the pivotal randomized phase 3 trial, teprotumumab produced clinically meaningful improvements in proptosis, disease activity, diplopia, and quality of life in patients with active, moderate-to-severe TED [1]. For orbital surgeons, the practical question has therefore changed. It is no longer simply whether decompression works, but when it adds value in a treatment landscape in which medical and surgical therapies increasingly address overlapping manifestations of the disease.

The first distinction must remain urgency. Dysthyroid optic neuropathy and severe corneal exposure are potentially sight-threatening conditions. In these settings, the availability of biologic therapy should not create therapeutic hesitation when vision remains threatened despite appropriate urgent management or when decompression is otherwise indicated. The recent WSOPRAS-ITEDS consensus statement provides a useful global framework, emphasizing that management should be adapted to disease severity and activity, available expertise, and local healthcare resources [2]. This is particularly relevant because access to teprotumumab remains uneven across countries and healthcare systems.

Outside the sight-threatening setting, decision-making is more nuanced. Teprotumumab can reduce proptosis substantially in selected patients, yet a good medical response does not necessarily remove the future need for surgery. In a retrospective single-center series, 25% of patients treated with teprotumumab subsequently underwent bony orbital decompression [3]. That figure should not be generalized to all patients with TED: referral patterns, baseline severity, treatment access, and local surgical practice inevitably influence who ultimately reaches the operating room. What the study does show is simpler and clinically important - decompression has not disappeared from the therapeutic pathway.

Perhaps the most useful way to think about the two treatments is not as competitors. They do different things. Teprotumumab is a monoclonal antibody targeting IGF-1R and can reduce inflammatory activity and orbital soft-tissue expansion [1]. Decompression changes the physical relationship between orbital contents and the bony orbit. It remains particularly relevant when persistent or recurrent proptosis, exposure, congestion, or disfigurement continues to trouble the patient after the disease has stabilized. Comparative observational data suggest that decompression may achieve a greater reduction in proptosis, whereas teprotumumab may have a more favorable effect on extraocular muscle motility [6]. These observations are clinically useful, but they should not be mistaken for evidence from randomized head-to-head trials.

The conversation with the patient therefore needs to move beyond a single millimeter measurement. Orbital decompression has well-recognized potential complications, and the possibility

of postoperative diplopia and further rehabilitative procedures should form part of preoperative counseling [2]. Medical therapy has its own burden. Prospective audiometric data have identified hearing changes in a subset of patients receiving teprotumumab, with pre-existing hearing dysfunction appearing to confer greater risk [4]. Cost, access, comorbidities, baseline ocular motility, expectations regarding appearance, and the possibility of further treatment all matter. A technically successful reduction in proptosis is not necessarily the same as a successful treatment pathway from the patient's perspective.

Timing is another area in which certainty remains limited. A multicenter retrospective study examined patients undergoing orbital, strabismus, or eyelid surgery at different intervals during or after teprotumumab treatment. Regression rates were not significantly different between patients operated before and after 180 days from the last infusion, although the clinical pattern of regression differed between groups [5]. These data are valuable, but they do not establish a universal waiting period. In elective cases, stability may be more informative than an arbitrary number of months: stable proptosis, ocular alignment, eyelid position, corneal status, and inflammatory activity together provide a more meaningful surgical target. Serial photographs and standardized measurements are therefore not merely documentation; they are part of surgical decision-making.

This changing landscape also challenges the traditional idea of a linear TED rehabilitation sequence. Some patients may achieve enough improvement with medical therapy to avoid decompression altogether. Others will have residual proptosis or exposure and still benefit from surgery. In selected patients, biologic treatment and decompression may be complementary rather than mutually exclusive. The question becomes less "medical therapy or surgery?" and more "what is the dominant problem now, and which intervention addresses it most directly?"

The next generation of studies should reflect that question. Proptosis remains important, but it is not enough. We need prospective comparisons of complete treatment pathways that include visual function, diplopia, exposure, appearance, quality of life, durability, adverse effects, additional procedures, and cost. We also need to understand which patients are most likely to need surgery after biologic therapy and whether treatment sequence meaningfully changes long-term outcomes.

The biologic era has not made orbital decompression obsolete. It has made its indication more precise. That is a welcome change. Surgery should neither be viewed as the inevitable final step for every patient with TED nor be displaced simply because an effective biologic treatment exists. The role of the orbital surgeon is increasingly to identify the problem that remains after the disease - and its treatment - has evolved, and to intervene when surgery offers a clear and durable benefit.

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