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Short Communication

Risk Factors of Thyroid Eye Disease Prognosis in Restrictive Myopathy

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DESCRIPTION

All periocular tissues become inflamed in Thyroid Eye Disease (TED), which can cause proptosis, eyelid retraction, strabismus, or compressive optic neuropathy [1]. Clinical symptoms might range in severity from asymptomatic or mild ocular and eyelid irritation to profound vision impairment [2]. Additionally, the clinical course of TED can occasionally be chronic and relapsing, necessitating follow-up therapy over an extended period of time. Generally speaking, autoactivated T-cells and autoantibodies activate orbital fibroblasts in the rectus muscles and orbital fat during the active phase of TED. Depending on their cell subtypes and kinds of cytokines, they interact with autoreactive T-cells when activated and develop into myofibroblasts or mature adipocytes. Hyaluronan can be produced by both myofibroblasts and adipocytes, which causes the soft tissues of the orbit to bulge edematously. This phase is characterised by a predominance of proinflammatory T-helper type 1 cytokines and an inflammatory response [3]. Therefore, immunosuppressive strategies have been employed to inhibit and shorten the active phase, including the injection of systemic steroids, immunosuppressants, and low-dose radiation. T-helper type 2 cytokines predominate throughout the chronic fibrotic phase. In the rectus muscles and soft tissues of the orbit, they cause fibrotic alterations. Surgery for rehabilitation may be used to fix structural issues brought on by a disease process. In about 20% of TED patients, the extraocular muscle develops a restrictive myopathy. During an active period, rectus muscle edoema may be the cause, and during a chronic phase, rectus muscle fibrosis may be the cause. Diplopia is the most upsetting symptom of restricted myopathy [4]. In addition to lowering quality of life, it also results in work incapacity.

Patients with TED who had normal ocular motility and a predominance of lipogenic change were classified as type I according to Nunery's classification of extraocular movements, while patients with significant restrictive myopathy and diplopia within 20° of the primary position were classified as type II. The type II disease, also known as TED with substantial restrictive myopathy, had a lower female-to-male ratio and a higher peak age of onset [5].

Patients with type II illness who had a lower orbital decompression outcome and a significant incidence of compressive optic neuropathy had a worse clinical prognosis.

Even though type II disease's epidemiology and natural history have been thoroughly explained, the clinical course of restricted myopathy in TED patients receiving immunosuppressive therapy has not been adequately documented. It is unknown what clinical circumstances would effect the muscle's recovery and the angle of strabismus during long-term follow-up.

CONCLUSION

In order to determine the predictive characteristics of restricted myopathy linked with TED, we examined the clinical and laboratory aspects in this study. Throughout the follow-up after treatment in this trial, the mean Limitation of the Extraocular Muscle Excursion (LOM) in patients with TED dramatically improved; however, a subset of patients exhibited no improvement in LOM. A worse prognosis for LOM recovery was linked to a greater baseline TSAb titer. The not-improved group's mean initial TSAb concentration was 66.4% greater than that of the improved group. Additionally, whereas the pre-treatment diplopia grade, pre-treatment LOM, and mean rectus muscle thickness did not substantially differ between the two groups, the diplopia grade at 1 year after treatment was significantly lower in the better group than in the not-improved group.

REFERENCES

1. Darin N, Kollberg G, Moslemi AR, Tulinius M, Holme E, Grönlund MA et al. Mitochondrial myopathy with exercise intolerance and retinal dystrophy in a sporadic patient with a G583A mutation in the mt tRNAphe gene. Neuromuscular Disorders. 2006;16(8):504-506.
2. Parulekar MV, Halliday W, Brent M, Kong K, Wong AM. Tubular aggregate myopathy associated with retinal degeneration. Can. J. Ophthalmol. 2008;43(3):361-363.
3. Goel R, Shah S, Sundar G, Arora R, Gupta S, Khullar T. Orbital and ocular perfusion in thyroid eye disease. Surv Ophthalmol. 2023 Jan 18.

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4. Vinson KB, Kirzhner M. Effects of teprotumumab on patients with long-standing, active thyroid eye disease. Am. J. Ophthalmol. 2022;26:101348.

5. Caroline YY, Keen JA, Shriver EM. Teprotumumab: A Major Advance in the Treatment of Thyroid Eye Disease Management. Advances in Ophthalmology and Optometry. 2022;7(1):383-400.