A 46-year-old woman was diagnosed with Graves’ disease 2 years prior to consultation and had undergone a total thyroidectomy 45 days before. She complained of progressive visual loss over the last 2 weeks. She had 20/100 visual acuity in the right eye and 20/20 in the left eye, with a relative afferent pupillary defect of 1.2 log in the right eye and a fundus without alterations. Magnetic resonance imaging of the orbits revealed hypertrophy of the extrinsic ocular muscles, which caused compression of the optic nerve at the orbital apex (Figure 1).

Dysthyroid optic neuropathy (DTON) is a serious complication of Graves’ orbitopathy that can result in profound and irreversible visual loss1,2. It affects about 5%-8% of patients with Graves’ orbitopathy and, in approximately 90% of cases, is related to compression of the optic nerve at the orbital apex by hypertrophic extraocular muscles (crowded orbital apex syndrome)2,3. In other cases, DTON is associated with optic nerve stretching secondary to proptosis3. Other factors may also be involved in the pathophysiology of DTON, including orbital congestion and hypertension, vascular insufficiency, and inflammation3. Risk factors for the development of DTON include advanced age, male sex, smoking, and diabetes mellitus3,4. Furthermore, the treatment for DTON includes intravenous systemic corticosteroid therapy and, for refractory cases, orbital decompression4,5.
REFERENCES


AUTHOR INFORMATION

» Luciano de Sousa Pereira
http://lattes.cnpq.br/9842717326456855
http://orcid.org/0009-0003-9783-3083