

The objective was to present a clinical case of patient with Graves' disease and GO.

Clinical case: Patient V.P. (45 years) was hospitalized in the Department of Endocrinology on the 16.03.16, with clinical manifestations: painful feeling behind the globe, redness of the conjunctiva, hyperlacrimation, exophthalmos and diplopia. In July 2014, patient was diagnosed with Graves' disease, treatment with ATS was initiated. In April 2015, supports a viral infection that leads to worsening general appearance of exophthalmos, decreased eyes motility, sensation of "sand" in the eyes, hyperlacrimation and decreased visual acuity. Patient diagnosed with GO and oral Prednisone was given in decreasing doses: 30 mg for the first week, after the dose was tapered off by 5 mg per week and GO ameliorates. In September 2015, after a virosis, clinical signs of GO becomes more severe and the patient resumes treatment with Prednisone. As a result of recently appeared flu (2-3 weeks), GO worsens and patient is hospitalized for pulse therapy. Clinical activity score was appreciated according to CAS=6. Family history: patient's sister and brother have Graves disease with severe GO. Hormonal tests: **05.15** FT4-16,8 pmol/L (*normal values range* = 12-22 pmol/L); **07.15** FT4-33,2 pmol/L; TSH <0,005 mIU/L (*normal values range* 0,27- 4,2 mIU/L); **09.15** FT4-10,8 pmol/L; TSH 0,011 mIU/L; **11.15** FT4-12 pmol/L; TSH 0,185 mIU/L; **01.16** FT4-58,5 pmol/L; TSH <0,005 mIU/L; **03.16** TSH 0,011 mIU/L; FT4 11,24 pmol/L; FT3 4,32 pmol/L (*normal values range* 3,1-6,8). MRI of the orbit: diffuse thickening of: m.rectus inferior to 1,0 cm (*normal values range* 0,49-0,57 cm), m. rectus medial to 0,85 cm (*normal values range* 0,41-0,46 cm), m. rectus laterale to 0,7 cm (*normal values range* 0,29-0,35 cm), m. rectus superior to 0,75 cm (*normal values range* 0,38-0,45 cm) with signs of edema.

The CAS wasn't determinate before and after Prednisone treatment and we can't appreciate the success of suppressive treatment. In etiology an important role has genetic predisposition (20-60% of affected individuals have a positive family history of thyroid disease), 21 % of the risk for developing GD is attributable to environmental factors (infectious agents). To confirm the genetic predisposition it would be ideal to identify the cytokines: HLA-DR3, CTLA4, PTPN22, CD40, IL-2RA, FCRL3, and IL-23R. Also, we can't ignore the influence of other factors in the pathogenesis of GO, such as female gender and the age 45 years.

Conclusion:

1. It is important to appreciate the clinical activity score of Graves' ophthalmopathy before and after the suppressive treatment.
2. Environmental factors, like viral infections had an important role in the evolution and severity of Graves' ophthalmopathy.

Key words: Graves' ophthalmopathy, Graves' disease, score CAS.

24. HYPOPITUITARISM SECONDARY TO UNRUPTURED INTRACAVERNOUS CAROTID ANEURYSM WITH SELLAR EXTENSION ASSOCIATED WITH IPSILATERAL BRAIN ABSCESS: A CASE REPORT

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Introduction: The prevalence of intracranial aneurysms ranges from 0.4% to 3.6% in autopsy studies and from 3.7% to 6.0% in studies of patients undergoing cerebral angiography. Aneurysm projected into the sellar region account for 1% to 2% of all intracranial aneurysms and the hypopituitarism caused by them are very rare (0.17% cases).

Clinical case: A 73-years-old woman was admitted in the department of ophthalmology with headache, fever, painful proptosis of the right eye and visual loss. Her physical examination revealed nonpulsatile exophthalmos of the right eye with conjunctival hyperemia, complete loss of light perception and third nerve palsy. A head computed tomography showed a right-sided heterogeneous intracavernous mass extending to the sella. The patient's condition deteriorated due to hypotension accompanied by episodes of atrial fibrillation. She became comatose and was intubated emergently. Her hormonal investigations revealed hypopituitarism manifested of low serum free thyroxine and TSH, low ACTH and diminished basal cortisol. *Multiple sets of blood cultures confirmed Gram-negative bacteremia diagnosis.* She was placed on hormonal replacement therapy and empirical antibiotherapy.

The digital subtraction angiogram showed an unruptured intracavernous aneurysm (15 x 17 x 11 mm) of right internal carotid artery (ICA) with sellar extension. A repeat CT scan and MRI, discovered a brain abscess in the right temporal lobe (9 x 6 mm) *with perifocal edema.* Antibiotics were administered intravenously during the *hospitalisation* (3 weeks) followed by a course of oral antimicrobial therapy (3 weeks) adapted to culture results. After 24 days of hospitalization, the patient status improved and she was discharged home with treatment recommendations.

Discussion results: Most of the patients with intracavernous carotid aneurysms are asymptomatic. Symptomatic patients frequently present with the compression symptoms caused by the mass effect of an aneurysm (oculomotor palsy, trigeminal dysesthesia and optic nerve compression). Very rarely, unruptured intracavernous aneurysm with sellar extension may determine hypopituitarism due to mass-effect on the pituitary gland or the hypothalamo-pituitary axis. In our case, the initial diagnosis was difficult to establish due to its atypical features and presentation that may simulate other disorders. A *literature* review showed that hypopituitarism secondary to sellar ICA aneurysm is usually permanent in most of cases even after treatment of the aneurysm.

Conclusion: To the best of the authors' knowledge, this represents the first reported case in the literature of hypopituitarism secondary to unruptured intracavernous carotid aneurysm with sellar extension Associated with ipsilateral brain abscess.

Key Words: Hypopituitarism, internal carotid artery aneurysm, brain abscess.

25. CONGENITAL INFERIOR VENA CAVA HYPOPLASIA AND MULTIPLE VENOUS THROMBOSIS POSSIBLY CAUSED BY INHERITED COAGULOPATHIES DISORDERS.

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