tissue disease[2] or with recent infections.[1,5] Dissection most commonly originates in the V3 segment,[6] followed by V1 and V2 segments.

The most common symptoms include vertigo, headache, and neck pain; the least frequent are ischaemic stroke, transient ischaemic attacks, and subarachnoid haemorrhage.[2] In rare cases it manifests as peripheral neuropathy and the most commonly affected nerve root is C5.

Arterial dissection occurs when blood enters the tunica media and the haematoma expands, causing the arterial wall to dissect. Compression of motor roots due to haematoma expansion within wall layers, or to impaired perfusion in the vasa nervorum causing radicular ischaemia, are the most widely accepted hypotheses to explain radiculopathy secondary to VAD.

Before non-invasive techniques such as MRI and Doppler ultrasound were used, arteriography was the technique of choice. MRI is currently a very sensitive method that enables identification of intramural haematoma, as well as the arterial lumen decreased by stenosis, in most cases.[7] In addition, Doppler ultrasound can be a very useful tool for measuring flow by means of high resolution images,[3] and to stratify the risk of ischaemic recurrence.[8]

Anticoagulants and antiplatelet drugs are the treatment of choice to prevent dissection progression and thromboembolic events associated with VAD.[9] When medical treatment fails, intravascular treatment represents a safe and effective therapeutic option.[10,11] The prognosis for radiculopathy secondary to VAD seems positive according to published studies[12]; resolution of symptoms and abnormalities on the vascular wall is frequent.

References

1. Engelter ST, Grond-Ginsbach C, Metso TM, Metso AJ, Kloss M, Debette S, et al. Cervical artery dissection: trauma and other potential mechanical trigger events. Neurology. 2013;80:1950—7.
2. Gottesman R, Sharma P, Robinson KA, Arnan M, Tsui M, Ladha K, et al. Clinical characteristics of symptomatic vertebral artery dissection. A systematic review. Neurologist. 2012;18:245—54.
3. Crum B, Mokri B, Fulgham J. Spinal manifestations of vertebral artery dissection. Neurology. 2000;55:304—6.
4. McGillion SF, Weston-Simons S, Harvey JR. Vertebral artery dissection presenting with multilevel combined sensorimotor radiculopathy: a case report and literature review. J Spinal Disord Tech. 2009;22:456—8.
5. Béjot Y, Aboa-Eboulé C, Debette S, Pezzini A, Tatlisumak T, Engelter S, et al. Characteristics and outcomes of patients with multiple cervical artery dissection. Stroke. 2014;45:37—41.
6. Arnold M, Bousser MG, Fahrni G, Fischer U, Georgiadis D, Gandjour J, et al. Vertebral artery dissection presenting findings and predictors of outcome. Stroke. 2006;37:2499—503.
7. Takano K, Yamashita S, Takemoto K, Inoue T, Kuwabara Y, Yoshimitsu K. MRI of intracranial vertebral artery dissection: evaluation of intramural haematoma using a black blood, variable-flip-angle 3D turbo spin-echo sequence. Neuroradiology. 2013;55:845—51.
8. Yamaoka Y, Ichiwaka Y, Kimura T, Sameshima T, Ochiai C, Morita A. A novel method for transcranial Doppler microembolic signal monitoring at the vertebrobasilar junction in vertebral artery dissection patients. J Neuroimaging. 2014;24:191—4.
9. Caplan LR. Antiplatelets vs anticoagulation for dissection: CADISS nonrandomized arm and meta-analysis. Neurology. 2013;80:970—1.
10. Hernández-Durán S, Ogilvy CS. Clinical outcomes of patients with vertebral artery dissection treated endovascularly: a meta-analysis. Neurosurg Rev. 2014 [Epub ahead of print].
11. Flis CM, Jäger HR, Sidhu PS. Carotid and vertebral artery dissections: clinical aspects, imaging features and endovascular treatment. Eur Radiol. 2007;17:820—34.
12. Lee VH, Brown RD Jr, Mandrekar JN, Mokri B. Incidence and outcome of cervical artery dissection: a population-based study. Neurology. 2006;67:1809—12.

L. Llull*, D. la Puma, N. Falgàs, A. Renú, A. Iranzo

Servicio de Neurología, Hospital Clínic de Barcelona, Barcelona, Spain

* Corresponding author.
E-mail address: blllull@clinic.ub.es (L. Llull).

Optic neuropathy after orbital decompression surgery☆

Neuropatía óptica tras la cirugía de descompresión orbitaria

Dear Editor:

Optic neuropathy is a possible, although very infrequent, complication of orbital decompression surgery.

We present the case of a 62-year-old woman with hyperthyroidism and long-standing thyroid-associated orbitopathy due to diffuse and nodular hyperplasia of the thyroid gland. The patient had been previously treated with steroids and total thyroidectomy to control symptoms. Exophthalmos was measured at 25 mm in the right eye (RE) and 23 mm in the left eye (LE) (Fig. 1). We also observed mild lid retraction and a bilateral supraduction deficit. At that time, our patient was being treated with topical ocular hypotensive medication for chronic bilateral open-angle glaucoma that had been stable for 4 years.

In her last examination before surgery, we recorded intraocular pressure (IOP) of 18 mmHg in both eyes, as well as optic disc cupping of 0.6 and 0.4 mm in the RE and LE, respectively. Results from both the campimetry study and the optical coherence tomography (OCT) performed before surgery were normal.

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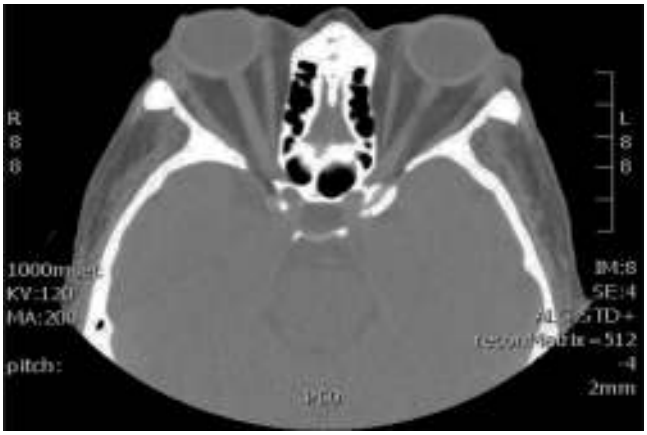

Figure 1 Computed tomography showing bilateral axial exophthalmos (asymmetrical, more pronounced in the RE) and elongation of the optic nerve.

The patient underwent orbital decompression surgery of the RE; the medial wall and orbital floor were removed endoscopically through the nasal cavity while the lateral wall was removed by the external approach. There were no complications.

One week after the surgery, the patient reported loss of visual acuity, but findings from the ophthalmological examination were identical to those from the previous one except for decreased IOP in the RE (14 mmHg). We performed tendency oriented perimetry (TOP) of the central 30° visual field, which revealed an altitudinal scotoma of the LE visual field. An OCT of the optic nerve fibre layer showed reduced thickness of that layer (Fig. 2). In light of these data, our diagnosis was ischaemic optic neuropathy possibly associated with the orbital decompression surgery.

Orbital decompression surgery is a therapeutic option for thyroid orbitopathy.[1] This procedure is generally performed after failure of corticosteroid treatment or radiotherapy during the active phase of the disease when vision is highly impaired, mainly due to compressive optic neuropathy. This procedure is also performed during the inactive phase to surgically correct sequelae, mainly consisting of keratitis due to corneal exposure, proptosis causing diplopia, and cosmetic disfigurement.[2,3] The procedure involves surgically opening orbital walls to create more space for the eye[4,5] and thus reduce proptosis and IOP.[6] Some of its more frequent complications are (generally) transient infraorbital nerve hypaesthesia,[2] sinusitis, epistaxis, oedema, and haematoma of the lids. New diplopia can occasionally develop and it usually resolves, although strabismus surgery may be required.[2]

Ischaemic optic neuropathy has been described as an infrequent complication of orbital decompression surgery.[6,7] Studies have suggested that this may be due to a direct intraoperative lesion to the optic nerve or to intraoperative increased IOP.[7]

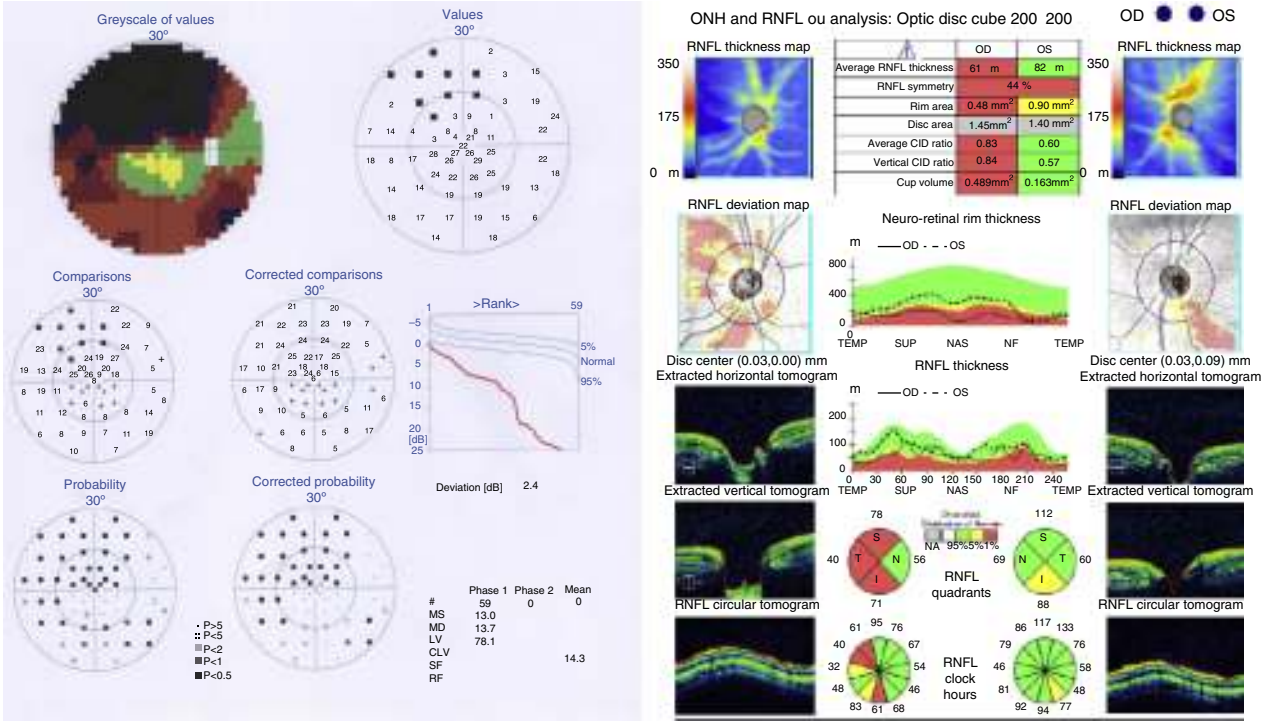

Figure 2 TOP of the 30° visual field and OCT of optic nerve fibre layers of the RE performed after orbital decompressive surgery. Studies respectively show a superior altitudinal defect and the corresponding decrease in thickness of the retinal nerve fibre layers in the lower sector.

References

1. Bartalena L, Pinchera A, Marcocci C. Management of graves ophthalmopathy: reality and perspectives. Endocr Rev. 2000;21:168—99.
2. Goh MS, McNab AA. Orbital decompression in Graves' orbitopathy: efficacy and safety. Intern Med J. 2005;35: 586—91.
3. Lal P, Thakar A, Tandon N. Endoscopic orbital decompression for Graves' orbitopathy. Indian J Endocrinol Metab. 2013;17: 265—70.
4. Pérez Moreiras JV, Coloma Bockos JE, Prada Sánchez MC. Orbitopatía tiroidea (fisiopatología, diagnóstico y tratamiento). Arch Soc Esp Oftalmol. 2003;78:407—31.
5. Saoers-Welch CV, Fatourechi V, Bartley GB, Beatty CW, Gorman CA, Bahn RS, et al. Optic neuropathy of Graves disease: results of transantral orbital decompression and long-term follow-up in 215 patients. Am J Ophthalmol. 2003;136:433—41.
6. Leong SC, White PS. Outcomes following surgical decompression for dysthyroid orbitophaty. Curr Opin Otolaryngol Head Neck Surg. 2010;18:37—43.
7. Eckstein A, Schittowski M, Esser J. Surgical treatment of Graves' ophthalmopathy. Best Pract Res Clin Endocrinol Metab. 2012;26:339—58.

A. Asorey-García*, R. Martínez-Chico, E. Santos-Bueso, J. García-Feijoo

Unidad de Neurooftalmología, Servicio de Oftalmología, Hospital Clínico San Carlos, Instituto de Investigación Sanitaria del Hospital Clínico, San Carlos (IdISSC), Madrid, Spain

*Corresponding author.
E-mail address: Almudena.asorey@gmail.com (A. Asorey-García).

Hemispatial neglect secondary to a traumatic brain injury☆

Heminegligencia espacial secundaria a traumatismo craneoencefálico

Dear Editor:

Hemispatial neglect is characterised by systematic omission of stimuli in the contralesional hemifield. This disorder affects such daily activities as getting dressed, eating, reading, or writing. Patients display a decreased ability to attend to, explore, and respond to novel or significant stimuli on the side opposite to the injury. This impairment cannot be attributed to presence of homonymous hemianopsia, which sometimes coexists with hemispatial neglect.[1] This frequent complication of right-sided vascular lesions[2] has also been described in patients with multiple sclerosis,[3] neurodegenerative diseases,[4] or head trauma.[5,6] We present a case of hemispatial neglect after severe head trauma.

Our patient is a 52-year-old right-handed man with no relevant medical history who experienced severe head trauma after falling in a suicide attempt. When the emergency medical services arrived on the scene, the patient had a Glasgow Coma Scale score of 6, a bleeding scalp wound, and numerous associated contusions. Brain CT performed when he was admitted to the emergency department displayed a comminuted fracture of the right parietal bone; the largest fragment was depressed. Subdural haematoma was identified in the right hemisphere, with a subarachnoid haemorrhage and the haemorrhagic contusion site in the parietal lobe. We found no midline shift, and the ventricular system was symmetrical and of normal size. Bone fragments were excised and the parenchyma of the right cerebral convexity was resected. A subsequent brain MRI (Fig. 1) displays a right frontal parenchymal lesion; a right subdural supratentorial haematoma with a discrete mass effect on the right hemisphere and right lateral ventricle and no midline shift; multiple microbleeds in the cortical and subcortical areas of both hemispheres, and around the lateral ventricles; and signal changes in the body and splenium of the corpus callosum.

One month after the traumatic injury, the patient was transferred to our hospital to undergo neurorehabilitation. Upon admission, he was disoriented and showed bradypsychia and psychomotor restlessness. Four months after head trauma, it was possible to conduct a formal neuropsychological examination, according to which the patient was oriented in time, space, and person and displayed preserved language skills. He showed verbal memory alterations (Rey Auditory Verbal Learning Test: short-term memory 35/75, long-term memory 2/15, and recognition 4/15) and executive dysfunction (Letter-Number Sequencing Test: 4/21; Wisconsin Card Sorting Test: number of categories 2/6, number of perseverative errors 32; Phonemic Verbal Fluency Test [3 min]: total number of words 19). During the examination, the patient was observed not paying attention to stimuli in the contralesional hemifield; homonymous hemianopsia had not been observed in the neurological examination. He was subsequently administered a specific neuropsychological testing protocol for assessing visuospatial attention (Bells Test, Ogden Figure Copying Task, Line Bisection Test, Baking Tray Task, and Reading Task); results confirmed presence of left hemispatial neglect. Ten days later, visuospatial tests were administered again; the patient showed slight improvements in task performance (Fig. 2).

Although hemispatial neglect commonly presents in patients with lesions in the right hemisphere, recurrence after head trauma is infrequent. Left hemispatial neglect has traditionally been associated with vascular lesions in the right parietal lobe.[7] At present, this condition is linked to right-hemisphere lesions, more specifically in the superior temporal gyrus, temporoparietal

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