■ CASE STUDIES

Reversible Retrobulbar Optic Neuritis Due to Sphenoidal Sinus Disorders: Two Case Studies

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Optic neuritis are clinically demonstrated by a temporary but severe loss of vision and can be caused by a wide variety of diseases. It is unusual for sphenoidal sinusitis to co-exist with acute optic neuritis, so the simultaneous appearance of both diseases would invite aetiological suspicion. We present 2 cases where the first clinical manifestation of infectious sphenoidal pathology was retrobulbar optic neuritis, which reverted with treatment, medical in one case and surgical in the other, of the sinusitis.

Key words: Optical neuritis. Sinusitis. Mucocele.

Neuritis óptica retrobulbar reversible por sinupatía esfenoidal: dos casos clínicos
Las neuritis ópticas pueden estar producidas por una gran variedad de enfermedades y se manifiestan clínicamente como una pérdida de visión temporal pero severa. Es raro que sinusitis esfenoidales coexistan con neuritis ópticas agudas, por lo que su relación invita a la sospecha etiológica. Presentamos 2 casos en los que la primera manifestación de una afección infecciosa esfenoidal fue una neuritis óptica retrobulbar que revirtió tras el tratamiento, médico, en un caso, y quirúrgico, en el otro, de la sinusitis.

Palabras clave: Neuritis óptica. Sinusitis. Mucocele.

INTRODUCTION

The term retrobulbar optic neuritis describes an inflammation of the optic nerve clinically characterized by loss of vision. Among the wide variety of conditions that may cause it, multiple sclerosis continues to be the most common. The relation between paranasal sinus involvement and optic neuritis is controversial, with the literature reporting anecdotal cases that corroborate it. Different mechanisms have been described by means of which a paranasal disease can come to provoke such involvement. The type of treatment applied will depend on the underlying cause; on occasion, surgical treatment may be required.

CASE STUDIES

Case 1

Twenty-one year old female without any history of interest comes to the emergency room due to symptomatology including decreased visual acuity with central scotoma in the right eye, lasting for 24 hours. She did not present dyschromatopsia, pain with eye movements, diplopia, or headache. The symptomatology remained stable from the time of onset. She had exhibited self-limiting paraesthesia in her right leg a few times over the course of the previous week that she related with changes in position. She had not displayed any gait disorder, motor deficit, or other associated neurological symptoms.

An emergency computerized tomography (CT) of the head was performed but was normal. The patient was admitted to the neurology department with suspected multiple sclerosis. The physical examination failed to reveal any finding of interest and examination of the *fundus oculi* was normal. During admission, magnetic resonance imaging (MRI) showed right sphenoid sinusitis as the sole finding of interest.

A study with visual evoked potentials was conducted with asymmetric latency responses, due to increased pathology on the right side. The neurophysiological examination revealed asymmetry, due to alteration on the right side, of the evoked visual responses in the scalp with macular stimulation, compatible with the clinical suspicion of right optic neuropathy.

Steroid-based treatment was initiated and symptoms improved, in the light of which the patient was discharged after 6 days and referred to our department for evaluation, where she reported having some difficulty breathing through her nose, often having a stuffy nose, no anterior or posterior

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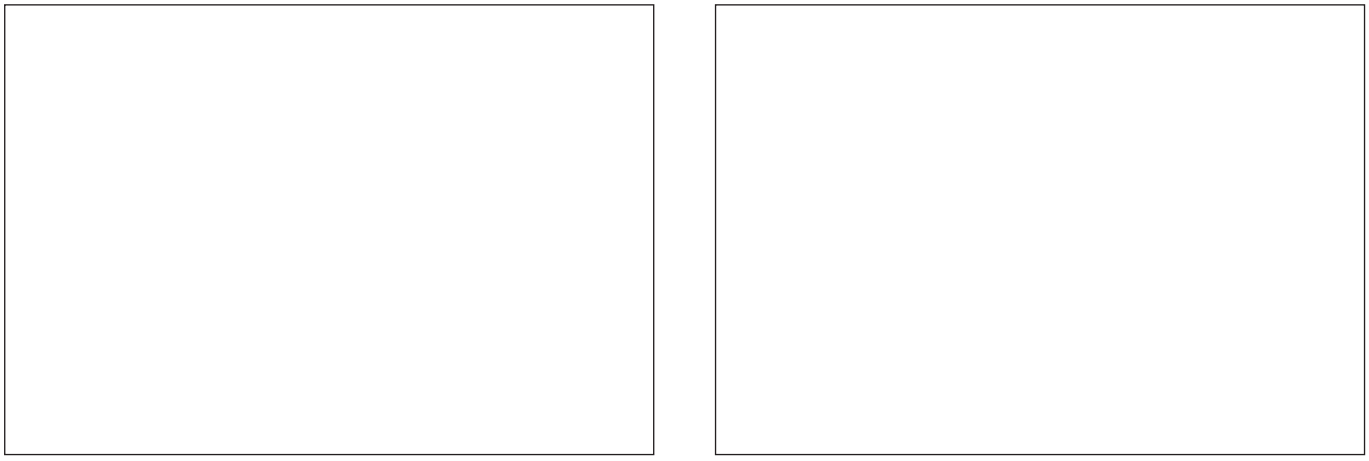

Figure 1. A: coronal slice of the computerized tomography (CT) in which occupation of the right sphenoid sinus and mucosal thickening is seen on the left. B: coronal slice of the CT of the same patient after medical treatment.

nosebleeds, or rhoncopathy. She did not have headache or hearing loss, although her voice was nasal. The rhinoscopic examination revealed only a slight septal deviation toward the left nostril, with compensating hypertrophy of the lower right turbinate, leaving little room for the passage of air. The CT of the paranasal sinuses revealed occupation of the right sphenoid sinus and thickening of the mucoperiostium of the left sphenoid sinus, with partial occupation of the anterior ethmoid cells (Figure 1A).

Oral and intranasal steroid treatment was initiated as a result of these findings, as well as oral antibiotic treatment with moxifloxacin (400 mg/24 h) for 1 month. Allergic testing was also ordered to typify possible allergic factors; all the tests performed were negative. The CT was repeated after 2 months, revealing that the previously described symptomatology had disappeared (Figure 1B); the patient was asymptomatic. A visual field study was requested for control purposes, with no scotomata and all parameters were within normal limits.

Case 2

Seventy-five year old male who came to the emergency department at his regional hospital due to loss of vision in his left eye; he reported that after having noticed non-specific discomfort in the same eye, he lost all sight, without displaying any other symptomatology. He was admitted to neurology where a CT was performed with the only finding worthy of note being occupation of the left sphenoid sinus (Figure 2), confirmed by MRI. The Doppler ultrasound scan of the supra-aortic trunks only indicated a single atheromatous plaque in both carotids. The visual evoked potentials confirmed left optic neuropathy. He was evaluated by ophthalmology where they found severe involvement of the visual field of the left eye with visual acuity of less than 1/10, increased papillary pallor in comparison to the right eye and physiological cupping of 0.5-0.6 in both eyes.

When brought to our department for evaluation, the loss of sight in his left eye persisted, with normal otorhinolaryngeal examination. The analyses performed on his arrival at our centre were normal, including a white blood cell count of 4600/µL with 77% neutrophils. The patient was admitted and intravenous antibiotic (amoxicillin-clavulanic acid) and steroid treatment begun.

Consultation with ophthalmology was ordered when the biomicroscopic examination was normal. The patient's uncorrected visual acuity was 0.6 in the right eye, and he was only capable of seeing fingers half a metre away with his left eye. The fundus of the right eye was normal, whereas the left eye presented a paler nerve ring than in the contralateral eye; the macula looked good and vascular trunks were normal.

Due to the limited visual acuity in the left eye, the visual field could not be quantified given the patient's inability to fix his gaze. These data support the diagnosis of a possible compression of the left optic nerve.

Four days after being admitted, the patient underwent endoscopic nasal surgery with a midline approach to the left sphenoid sinus, with abundant pus and a sphenoid cyst removed on opening. A communication was made between

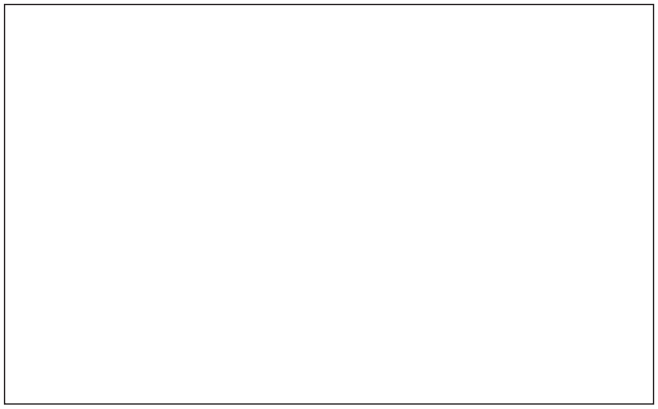

Figure 2. Coronal CT slice revealing a mucopyocele occupying almost the entire sphenoid sinus, with thinning of the bony walls.

both sphenoid sinuses by breaking the septum, as well as opening up the right ostium. No dehiscence of the left optic nerve was observed during surgery.

The culture of the pus was positive for *Enterobacter aerogenes* (inducible betalactamase-producing strain); consequently, treatment with ciprofloxacin was initiated.

The patient was re-evaluated by the ophthalmology department and this time he presented visual acuity in the left eye that enabled him to see fingers 1 m away. In the visual field, by comparison, he was seen to have recovered sight of the temporal area in that eye, which was consistent with the funduscopic examination in which the nasal neuroretinal ring was seen to have recovered its normal colouring with persistent pallor in the temporal area.

The patient gradually recovered his sight during his post-operative stay: he achieved greater corrected visual acuity of 0.4. His potential visual acuity is likely to have been better, but the intravenous steroid treatment caused a slight opaqueness of the crystalline lens. The visual field performed during this final check-up exhibited a residual nasal step with mean deviation of –1.56 dB. The patient was discharged 10 days after admission.

DISCUSSION AND CONCLUSIONS

Optic neuritis is a broad term describing inflammation, degeneration, or demyelinization of the optic nerve.[1] A wide variety of diseases may cause it, including retinitis, choroiditis, iridocyclitis, tuberculosis, syphilis, cellulitis, and orbital tumours, meningitis, polyneuritis, hyperthyroidism, etc. The most common cause of unilateral optic neuritis continues to be multiple sclerosis.[2] The term optic neuritis also encompasses retrobulbar neuritis, when the posterior aspect of the nerve is involved; hence, the ophthalmoscopic examination remains normal, except in the case of findings of optic atrophy. In clinical terms, it is characterized by severe loss of vision. In the typical case of idiopathic optic neuritis, visual acuity reaches its lowest point by the end of the first week; there may be pain in the ocular region, particularly with movements of the globus. Sight generally improves dramatically within 2 or 3 weeks without treatment. Half and two thirds of all patients have normal visual acuity at 1 month and 6 months, respectively.[3]

It has been calculated that more than one third of the fibres making up the optic nerve may become atrophied without obvious changes in visual acuity and the visual field or in the appearance of the optic disc, as we see in the first case, although scotomata, the most common defect, may be present in the central visual field. We can infer that, if we can resolve the underlying cause in the early stages, we will avoid irreversible optic damage with the corresponding functional repercussions. Most patients are between 20 and 50 years of age, with a slight predominance of females over males.

The relationship between involvement of the paranasal sinuses and acute optic neuritis has been investigated time and again but, after many anatomical and clinical studies, its aetiological role continues to be controversial. Onodi[4] in 1908 and Loeb[5] in 1909 reported the anatomical link between the optic channel and the posterior sinuses, emphasizing the contiguousness of the sphenoid and posterior ethmoid with the optic channel, with the possibility of direct spread of infections from these sinuses to the nerve.

There are various physiopathological routes by means of which sinusitis can give rise to optic neuritis: *a)* direct spread of the sinus infection and inflammation to the optic nerve, probably the most obvious and most common pathway, when pus passes through dehiscences of the posterior paranasal sinuses or by osteomyelitis of the walls of these sinuses[6,7]; it may be due to bacterial or fungal infections[8]; *b)* compressive optic neuropathy may be caused by ethmoid and/or sphenoid mucoceles or mucopyoceles[9]; *c)* by bacteraemias resulting from the passage of infection through the mucosa of the sinus[2]; *d)* vasculitis with thromboangeitis of the optic nerve[10,11]; and *e)* chronic allergic optic neuritis, reported in animal models, by intradermal injection of myelin in guinea pigs, by means of which said antigenic stimulation of the immune system might indirectly lead to demyelinization of the optic nerve.[12] In the cases reported here, the first 2 mechanisms might account for the neuritis: the direct spread of infection or osteomyelitis in the walls of the sphenoid sinus in the case 1 and compressive neuropathy in case 2.

With respect to treatment, it is worth noting that surgery of the paranasal sinuses should be reserved for patients with compressive optic neuropathy associated with mucoceles or pyoceles or for those cases in which there is a strong suspicion of suppurative sinusitis.[13] In spite of the fact that systemic corticoid therapy is frequently used, medical treatment for optic neuritis is controversial since the disease tends to improve spontaneously[2] if the sinusitis is resolved with antibiotic treatment.

Therefore, sphenoid sinus disease must be contemplated as a possible cause in all cases of retrobulbar optic neuritis.

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