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Caso Clínico

Posterior orbital cellulitis as a complication of sphenoid sinusitis: a case report

Celulite orbitária posterior, uma complicação de

sinusite esfenoidal: relato de caso

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Abstract

Posterior orbital cellulitis is a clinical syndrome in which early severe visual loss dominates or precedes accompanying inflammatory orbital signs. This syndrome occurs as a complication of sphenoethmoiditis with involvement of the orbital apical segment of the optic nerve, and therefore it may be considered as a partial orbital apex syndrome. If a Posterior orbital cellulitis is suspected, an imaging diagnosis should be established and early treatment initiated to prevent irreversible blindness. We present a case of a 14-year-old patient, which fulfilled the criteria of a "posterior orbital cellulitis".

Correspondencia: Joana Pires Centro Hospitalar Universitário de Coimbra Correo electrónico: joana.sanchespires@gmail.com **Keywords:** sinusitis complications, optical neuritis, acute vision loss, sphenoid sinusitis, posterior orbital cellulitis.

Resumo

A celulite orbitária posterior é uma síndrome clínica caracterizada por uma perda de visão severa que precede ou domina eventuais sinais inflamatórios orbitários. Esta síndrome ocorre como uma complicação de uma esfeno-etmoidite com envolvimento do segmento apical orbitário do nervo óptico, e portanto pode ser considerada como uma síndrome parcial do ápex orbitário. Se houver suspeita de uma celulite orbitária posterior, um diagnóstico de imagem deve ser estabelecido e deve ser iniciado tratamento precoce de forma a prevenir uma amaurose irreversível. Apresentamos um caso de uma doente de 14 anos, que cumpriu os critérios de uma "celulite orbitária posterior".

<u>Palavras-chave:</u> complicações de sinusite, nevrite ótica, perda de visão súbita, sinusite esfenoidal, celulite orbitária posterior.

Introduction

Acute sinusitis is a common disorder in children, which in most cases is managed uneventfully. However, in a few number of cases, if left untreated, serious complications may occur, such as orbital involvement that accounts for 74 - 85% of sinusitis complications.¹ Acute visual loss is one of such complications². Acute vision loss in association with sinusitis may occur as a complication of orbital cellulitis or as a com-

ponent of orbital apex syndrome (OAS), an uncommon disorder mainly characterized by the presence of ophtalmoplegia (impairment of cranial nerves III, IV, VI) along with the visual loss (optic neuropathy).³ More rarely, some patients present with isolated acute vision loss, preceding any other orbital sign. This is associated to the sole involvement of the orbital apical segment of the optic nerve, and therefore it may be considered as a partial orbital apex syndrome. This pattern of presentation has been named "Posterior orbital cellulitis".²

We present a case of a 14-year-old patient with isolated acute vision loss in a context of sphenoiditis, which fulfilled the criteria of a "posterior orbital cellulitis".

Case Report

A 14-year-old female adolescent presented at the Emergency Room with a 3-day history of isolated progressive left eye vision loss. She denied other ocular symptoms (diplopia, ophthalmoplegia, ocular pain), any nasossinusal symptoms (nasal obstruction, anterior or posterior rhinorrhea, facial pain, rhino-allergic symptoms, anosmia), fever or other neurological symptoms. There was no epidemiologic context or a recent history of illness. The patient was otherwise healthy.

Ophthalmologic evaluation revealed a best-corrected visual acuity (BCVA) of 20/20 on the right eye, and absence of light perception on the left eye. A relative afferent pupillary defect was present on the left eye. Ocular motility was unaffected, with slight left side tenderness on the upward gaze. Ortophoric in both near and distance fixation, with no anisocoria, proptosis, ptosis or nystagmus. Anterior segment examination and intraocular pressure were unremarkable in both eyes. Left eye fundus examination revealed a hyperemic diffuse optic disc edema, more pronounced on the superior and inferior poles, without hemorrhages (Figure 1). Associated with a loss of the foveal reflex, there were diffusely dilated retinal veins with increased tortuosity. Right eye fundus examination was unremarkable. Neurological examination was normal.

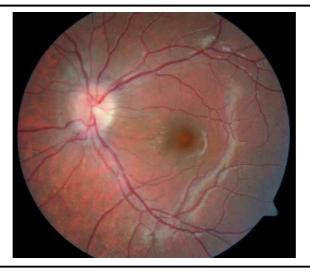


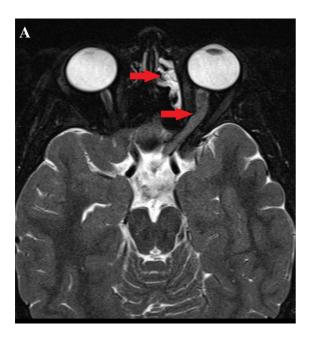
Figure 1: Left eye fundus examination revealed a hyperemic diffuse optic disc edema, more pronounced on the vertical poles, without associated peri-papillary hemorrhages.

Otolaryngologic examination revealed a deviation of the nasal septum to the right and inferior turbinate hypertrophy, mostly on the left side. There was no purulent nasal or pharyngeal drainage, periorbital edema or tenderness overlying sinuses.

An emergency head MRI demonstrated thickening and enhancement of the intraorbital, intracanal and pre-chiasmatic segments of the left optic nerve, with no evident alterations of the extraocular muscles or of the intra-orbital fat (Figure 2a and 2b). Besides these alterations, the MRI showed only a slight mucosal hypertrophy of the sphenoid sinus ant ethmoidal cells on the left side. Since there was no evident cause for such an obvious alteration of the optic nerve, a head CT scan was requested to exclude the hypothesis of a complicated sinusitis, associated to an eventual bone dehiscence.

The head CT scan showed a relatively exuberant hypertrophy of the left sphenoidal sinus, inclusively adjacent to the optic canal, where it could be admitted a slight demineralization of its inferior bone contour (Figure 3).

Given the association and proximity of these two conditions, it was assumed a probable diffuse left optic neuritis as a complication of an ipsilateral sphenoid sinusitis process.



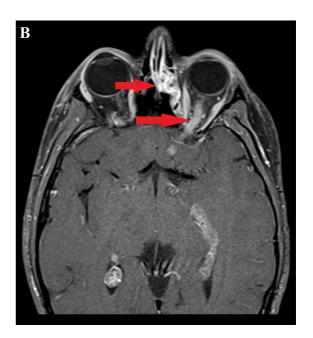


Figure 2a and 2b: Head MRI –day 1—Thickening and enhancement of the intraorbital, intracranial and pre-chiasmatic segments of the left optic nerve (red arrow). Exuberant inflammatory hypertrophy of the left spheno-ethmoidal sinus mucosa (red arrow).

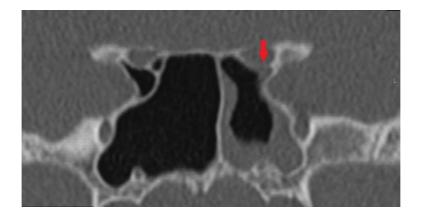


Figure 3: Head CT Scan –day 1 - Exuberant inflammatory hypertrophy of the left sphenoidal sinus mucosa associated to a Slight demineralization of the optic canal bone contour (red arrow).

As there were no signs suggestive of optic nerve compression that would require a surgical approach, the patient was admitted to the hospital for initiation of intravenous antibiotics (ceftriaxone 4g id) and corticosteroids (metilprednisolone 1g id). Intranasal steroid and decongestant treatment were initiated as well.

A thorough investigation for neurologic, auto-immune and specific infectious diseases was made, with no conclusive results.

During the hospital stay, left eye BCVA marginally improved to counting fingers at 1 meter, with a permanent relative afferent pupillary defect. Left eye fundus examination was unchanged during the acute stage. Multimodal imaging with optical coherence tomography revealed an increase in the retinal nerve fiber lay-

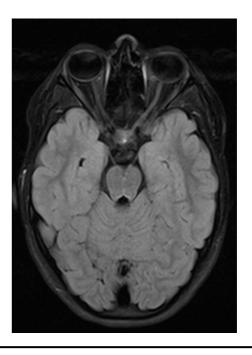
er due to edema, and a fluorescein angiography confirmed the optic disc edema with a late peripapillary leakage and no signs of retinal vasculitis. Visual evoked potentials performed at the 7th day revealed an absence of p100 wave on the left eye, contrasting with a normal exam on the right eye.

The patient was discharged on day 10 of disease, maintaining oral antibiotic (cefuroxime) and corticoid (prednisolone) treatment on an outpatient basis.

Throughout a 2-months follow-up period, BCVA slowly improved to 20/20, with a progressive decrease in retinal fiber layer thickness and resolution of the optic disc edema visible in the fundus examination.

A new head MRI executed on day 56 of disease, revealed a marked reduction of the thickening of the left optic nerve, corresponding to an imaging improvement of the optic neuritis process. An almost complete resolution of the ethmoid and sphenoid sinusitis phenomena was also verified (Figure 4).

Figure 4: Head MRI –day 57 - Imaging improvement of the left optic neuritis and almost complete resolution of the ethmoid and sphenoid sinusitis phenomena.



Discussion

The term orbital apex syndrome is classically applied when all the structures at the optic canal and superior orbital fissure are affected by a disease process.⁴ When the disease process affects only the optic nerve, it may be considered a partial orbital apex syndrome.⁴ Slavin and Glaser (1987) renamed this entity "posterior orbital cellulitis" and defined it as a clinical syndrome in which early severe visual loss overshadows or precedes accompanying inflammatory orbital signs.⁴ Meanwhile, this entity has been reported by many authors as an acute inflammatory optic neuropathy (retrobulbar optic neuritis), an uncommon complication of acute sinusitis.⁵

In the present case, the acute vision loss in the absence of the typical symptoms and signs of sinus or orbital inflammation makes this a representative case of a posterior orbital cellulitis syndrome. However, only after the imaging exams (Head CT and MRI scans) this diagnostic was considered. The exuberant inflammatory/infectious process of the left sphenoidal sinus associated to the erosion of the optic canal bone contour strongly suggested the process of a direct spread of the sinus infection and inflammation to the op-

tic nerve. This pathophysiologic process has been reported as probably the most obvious and most common pathway of optic neuritis.⁶ The invasion of the optic nerve by local bacteria and secondary inflammatory occlusive vasculitis would cause the optic neuritis.⁵

The exclusion of other and more common causes of optic neuritis in the pediatric age, along with the imaging and clinical resolution after antibiotic and corticoid therapy, contributed to establish the process of sphenoiditis as the most probable cause of this case of optic neuritis.

The incidence of permanent loss of vision in these cases is reported as high as 10.5%.⁷ An early diagnosis and treatment may help to prevent irreversible optic damage and the corresponding functional repercussions.⁶ Current treatment recommendations for acute sphenoiditis are intravenous broad-spectrum antibiotics for coverage of gram-positive organisms.^{7, 8} Depending on the degree of ophthalmologic, neurological and imaging findings, an immediate endoscopic sphenoidotomy may be the treatment of choice in order to release pressure on the optic nerve.⁷

A high index of suspicion of posterior orbital cellulitis should be kept up even in the absence of the common clinical signs of sinusitis. The presented case emphasizes the importance of neuroimaging for both diagnosis and treatment planning. As in most case reviews, it seems that these patients who have no gross evidence of orbital pathology, have a better visual prognosis.

Conflict of Interest: Without conflict of interests to declare.

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