

# Expanding intracranial parachiasmatic lesion simulating glaucoma: a case report and integrative literature review

Lesão expansiva intracraniana paraquiasmática simulando glaucoma: relato de caso e revisão integrativa de literatura

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## KEYWORDS:

Pituitary neoplasms; Glaucoma; Differential diagnosis.

## ABSTRACT

Glaucoma is a prevalent optic neuropathy that is characterized by the presence of increased optic disc cupping; however, other ophthalmologic pathologies may also be indicated by similar complications. Therefore, correct and early diagnosis of the etiology of changes in the eye is extremely important in the treatment of patients, especially in cases of potentially progressive and life-threatening diseases, including pituitary neoplasms. Herein, we report a case of misdiagnosis of glaucoma in a patient with a parachiasmatic intracranial expansive lesion, and briefly discuss some aspects that can help in the differential diagnosis of these conditions.

## PALAVRAS-CHAVE:

Neoplasias hipofisárias; Glaucoma; Diagnóstico Diferencial.

## RESUMO

Apesar de o glaucoma ser uma neuropatia óptica bastante prevalente e cursar com o achado característico de escavação do disco óptico aumentada, outras patologias oftalmológicas também podem cursar com quadro semelhante. Dessa forma, o diagnóstico correto e precoce da etiologia da alteração ocular é de extrema importância no tratamento dos pacientes, principalmente em casos de doenças potencialmente progressivas e ameaçadoras à vida, como em neoplasias hipofisárias. O presente artigo busca relatar um caso de equívoco no diagnóstico de glaucoma em paciente com lesão expansiva intracraniana paraquiasmática e realizar uma breve discussão sobre aspectos que auxiliam no diagnóstico diferencial dessas condições.

## INTRODUCTION

Glaucoma is a multifactorial degenerative optic neuropathy. It is the second highest cause of blindness, affecting an estimated 76 million people worldwide in 2020, and this number is estimated to increase to 111.8 million patients by 2040<sup>1</sup>. Glaucomatous optic neuropathy (GON) is the most common cause of

enlarged optic disc cupping, characterized by loss of neuroretinal rim, deep cupping, optic disc hemorrhages, peripapillary chorioretinal atrophy, decreased retinal arteriole diameter, diffuse or localized loss of nerve fiber layer (NFL), and typical changes in the visual field (VF)<sup>2,3</sup>. However, in up to 20% of cases, the finding of increased cupping may be attributable to other pathologies<sup>2,3</sup>.

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Regarding non-GONs that present with this finding, anterior ischemic optic neuropathy, hereditary neuropathies, and compressive injuries should be considered<sup>4</sup>. The latter includes sellar tumors which, through the compression of the anterior visual pathway, cause an increase in cupping prior to visual loss, as well as arcuate scotomas on VC examination, which could be confused with GON<sup>3,5-7</sup>.

Among pituitary tumors, 50%-80% are adenomas; these are very common benign neuroendocrine tumors and correspond to 15.5% of all tumors of the central nervous system (CNS)<sup>8,9</sup>. They can be classified by size as macroadenomas ( $\geq 10$  mm) or microadenomas ( $< 10$  mm), and as functional or non-functional, based on endocrine activity<sup>8-11</sup>. The incidence of adenomas is 3.4 cases per 100,000 inhabitants per year, with a bimodal peak between 25-45 years and 60-70 years<sup>9</sup>. Nonfunctioning tumors represent 33% of pituitary adenomas and, among those, macroadenomas are the most common, representing 75% of incidental diagnoses<sup>9,10</sup>. Notably other lesions can affect the chiasmatic region, including parasellar meningioma, craniopharyngioma, and internal carotid aneurysm<sup>12</sup>.

Visual impairment may occur as a result of a chronic increase in intracranial pressure, more specifically owing to compression or tumor invasion in the optic pathway<sup>11</sup>. Visual field defects-typically bitemporal hemianopsia-and low vision occur in 9%-32% and 4%-16% patients, respectively<sup>1</sup>. Other manifestations include headache, cranial neuropathy with and without ophthalmoplegia, ptosis, and endocrinopathy<sup>11</sup>.

Regarding diagnosis, magnetic resonance imaging (MRI) of the sellar region is the gold standard method for evaluating pituitary adenomas. The presence of visual damage, such as a visual field defect, is indicative of surgical treatment. However, in 10%-20% of cases, the visual field does not improve after surgery<sup>13</sup>.

The treatment of choice is transsphenoidal resection, which can be performed through the endoscopic or microscopic approach, whereas the transcranial approach is indicated for large tumors that extend to the middle fossa and cavernous sinus. Nonfunctioning adenoma has a recurrence rate of approximately 20%-30%; thus, an MRI is performed every 3-6 months after surgery, annually for 5 years, and biannually for an additional 6 years. The two most significant factors for recurrence are extension into the cavernous sinus and the presence of adenoma persisting after

surgery. Postoperative radiotherapy can help reduce recurrences from 40% to 10%; however, the associated adverse effects must be considered, including pituitary hormone deficiency, cerebrovascular event, optic nerve damage, and brain tumors<sup>9</sup>. Other options are drug suppression and radiotherapy<sup>8</sup>.

We aim to report a case of pituitary macroadenoma with significant visual function alteration simulating normal pressure glaucoma (NPG), with further discussion on the differential diagnosis between the two pathologies.

## CASE REPORT

A 46-years-old man, complained of visual loss in the right eye (OD) beginning 8 months before the study period. He was diagnosed with glaucoma at another Healthcare center, prescribed with a combination of 0.2% brimonidine and 0.5% timolol for 6 months, and presented with iridotomy in both eyes (OU) 4 months previously. Upon examination, corrected visual acuity (AVcc) in the OD was light perception ( $+1.00 -4.00 \times 60$ ) and in the left eye (OS) was 20/50 ( $+2.50 -4.50 \times 100$ ). Reduced direct photomotor reflex was observed in the OD in addition to relative afferent pupillary defect, biomicroscopy with iridotomy at 1 hour in OU, intraocular pressure (IOP) of 12/12 mmHg and funduscopy with increased cupping ( $0.8 \times 0.8$ ) in OU, as well as total pallor of the papilla and macula without changes in OU. Owing to the fact that the IOP was within the normal range and the presence of a previous acute glaucoma crisis was not indicated, the hypothesis of changes in the optic disc as a result of neurological or cardiovascular etiologies was considered. Cranial MRI, carotid Doppler, VF 30.2, optical coherence tomography (OCT), and retinography were prescribed. He returned with VF showing amaurosis on the right and loss of the temporal hemifield and lower nasal part on the left (Figure 1); retinography with bilateral optic atrophy and signs of grade 1 hypertensive retinopathy (Figure 2); OCT of the OD with cupping of  $0.77 \times 0.76$  and loss of superior and inferior NFL, and OS with cupping of  $0.78 \times 0.72$  and loss of superior and temporal NFL, as well as retinal ganglion cells with bilateral losses (Figure 3). Carotid Doppler with left carotid intima-media thickening, without stenosing plaques. Finally, MRI identified a parachiasmatic intracranial expansive lesion compressing the optic chiasm (Figure 4). The patient was then referred to the neurosurgery service.

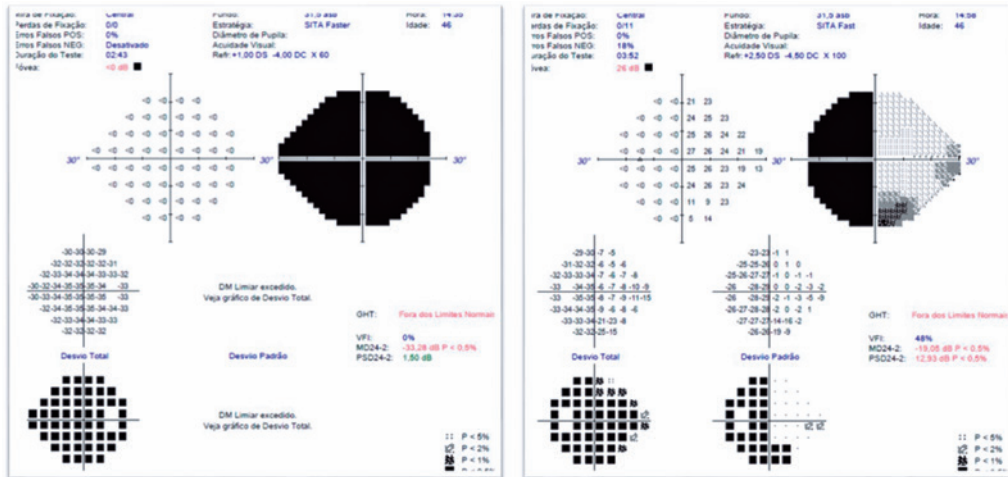


Figure 1. Visual field showing amaurosis on the right and loss of temporal hemifield and lower nasal part on the left.

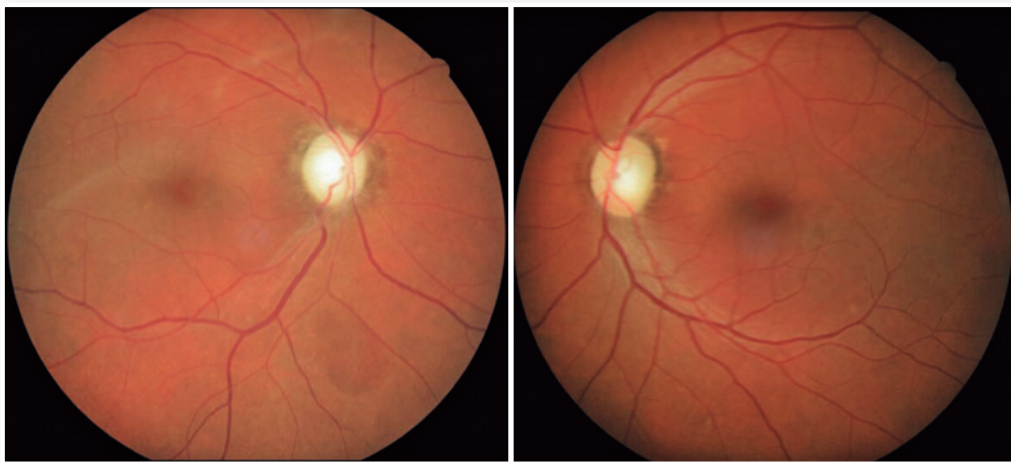


Figure 2. Retinography showing bilateral optic atrophy and signs of grade 1 hypertensive retinopathy.

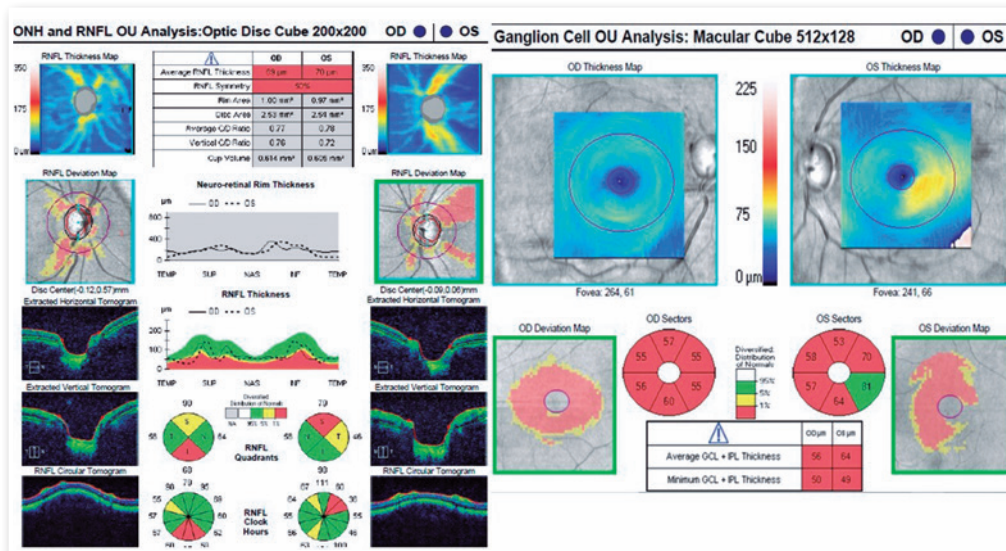
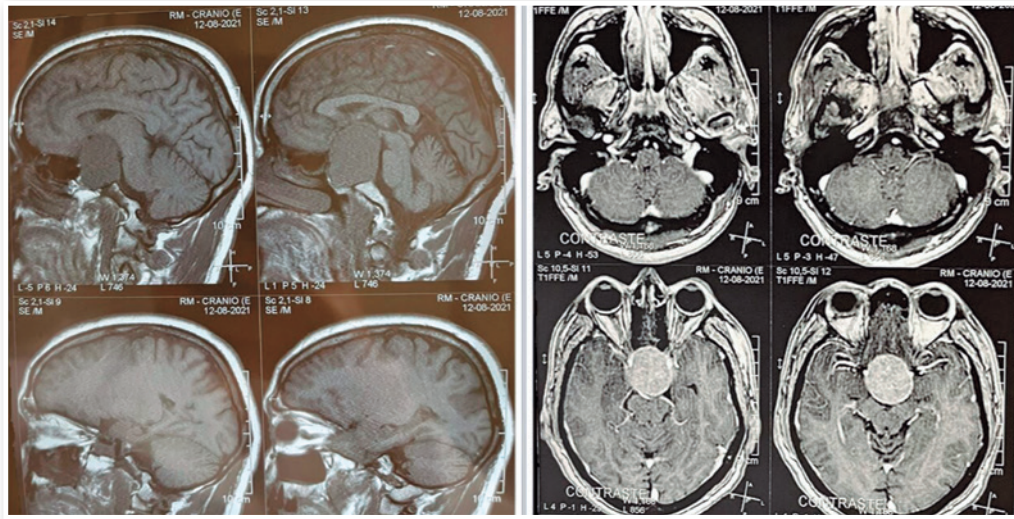


Figure 3. Optical coherence tomography showing increased cupping in both eyes, with a reduction in the superior and inferior nerve fiber layer in the right eye and superior and temporal in the left eye, as well as a diffuse reduction in the bilateral macular ganglion cell layer.





**Figure 4.** MRI of the brain and orbit showing expanding intracranial parachiasmatic lesion.

## DISCUSSION

Although optic neuropathy is most commonly associated with increased optic disc cupping, this finding is not a pathognomonic sign of GON, thereby highlighting the need to establish a differential diagnosis with other neuropathies. One such neuropathy, compressive optic neuropathy (CON), has been reported in this case<sup>2,3</sup>.

In many cases of CON, atrophy of the optic nerve is the only finding on funduscopic examination. Furthermore, a diagnostic confusion exists regarding NPG; one study observed a case series of 6.5% of patients previously diagnosed with NPG who had a compressive lesion<sup>14</sup>.

Another study, conducted in 2011, evaluated 501 patients with sellar tumors and identified an optic disc with a glaucomatous appearance significantly more frequently than in the control group. This finding was more closely associated with tumor location and size, i.e., the larger the tumor and the closer the internal opening of the optic canal, the more frequent the glaucomatous appearance. Among the hypotheses examined, sellar tumors located close to the ON are believed to block the passage of cerebrospinal fluid to the orbit through the ON channel. This would result in a collapse of the retrobulbar cerebrospinal fluid space, causing an increase in pressure gradient across the cribriform lamina. This situation would mimic what occurs in the case of normal retrobulbar cerebrospinal fluid; however, with increased IOP. Corroborating this hypothesis, recent studies have

suggested that some patients with NPG may have low cerebrospinal fluid pressure<sup>15</sup>.

However, although initially, the case reported was similar to that of NPG, glaucomatous patients have loss of neuroretinal rim and deep cupping. The rim is preserved in CON, albeit paler, which can be observed when evaluating the paler rim. on retinography<sup>7,14</sup>. Corroborating this fact, a study from 2016 showed that in early stages, eyes with CON have greater rim pallor than those with GON, despite having the same degree of nerve fiber involvement, reflecting different histopathological characteristics in relation to glial architecture in each pathology. However, in moderate to advanced stages, this relationship would not be maintained<sup>14</sup>.

Glaucomatous cavitation is understood to be associated with the death of ganglion cells, and is referred to as laminar or deep. Glaucomatous cupping is believed to have a greater anterior laminar depth than non-glaucomatous cupping<sup>5</sup>. Based on this, a 2020 study used the measurement of the minimum rim width at the opening of the Bruch's membrane (BMO-MRW)-a true anatomic edge of the neuroretinal rim-for quantitative assessment of cupping. The results demonstrated that patients with GON showed significantly lower values than those with non-GON<sup>5</sup>. When directly comparing CON and GON, another study, conducted in 2020, demonstrated that the ratio of nasal BMO-MRW to peripapillary NFL (N-BMO-MRW/pRNFL) with values  $\geq 4.24$  showed diagnostic sensitivity and specificity of 84%

and 72%, respectively. However, in patients with non-GON, MRW is preserved, despite the diffuse loss of NFL<sup>16</sup>. Although this assessment alone does not allow a differential diagnosis, it contributes to considering differential diagnoses of non-glaucomatous etiology, and to performing a more accurate clinical examination and thereby assessing the need for additional tests. Notably, this measurement does not specifically differentiate the type of non-GON<sup>5</sup>.

Another parameter used to differentiate GON and CON is the thickness of the macular ganglion cell layer, especially in the nasal subfields, which leads to a reduction in thickness prior to the appearance of changes in visual field in cases of CON<sup>7,17</sup>. Furthermore, it is a well-established fact that CON affects the NFL more temporally, in particular, and also nasally, whereas GON is more likely to affect the upper and lower regions of the NFL<sup>3,7,17</sup>. When compared to early glaucomas, loss of papillomacular bundle fibers is more common in non-GON, regardless of etiology<sup>17</sup>. However, when comparing advanced glaucomas, this differentiation is lost owing to an increased greater involvement of the NFL<sup>7</sup>.

Finally, some authors cited factors as indicative of non-GON and the need for neurological investigation. These factors include the following: young age (<50 years), low visual acuity (<20/40), vertical visual field defects in relation to the midline, pallor of the neuroretinal rim, asymmetrical loss of color vision, and relative afferent pupillary defect. Since the patient in the case reported had at least five of these factors, the initial diagnosis of glaucoma was considered as opposed to a non-glaucomatous etiology<sup>4,7</sup>.

Although glaucoma is a much more frequent neuropathy than CON, a detailed, critical clinical examination should always be performed, paying attention to the often subtle signs of non-glaucomatous etiology, such as those presented in this article. This will enable a more accurate diagnosis of the patient's real condition, as well as adequate treatment, resulting in a more favorable visual prognosis with the possibility of early treatment.

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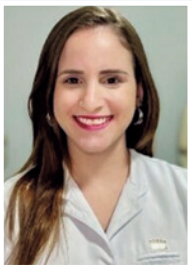
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