Case Report

Pituitary Adenoma Misdiagnosed as Glaucoma in an Adult Nigerian Male

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Abstract

We report a case of pathologic disc cupping in a 45-year-old Nigerian male with pituitary adenoma, which was misdiagnosed and treated as glaucoma with resultant bilateral blindness. Pathologic cupping of the disc is most commonly associated with glaucoma. However, the clinician needs to be aware that there are non-glaucomatous causes of pathologic disc cupping, some of which may be life-threatening.

Keywords: Cupped disc, glaucoma, non-glaucomatous cupping, pituitary adenoma

Introduction

Pituitary adenoma is a tumour of the pituitary gland (which lies within the sellar turcica). Suprasellar extension of the tumour causes compression of the overlying optic chiasm with resultant ophthalmic manifestations such as progressive vision loss, visual field defects, colour dyschromatopsia and optic atrophy.[1] Although a pale pathologically cupped disc is most commonly associated with glaucoma, pathologic cupping of the disc in some cases of pituitary adenoma has been reported in the literature.[2-4]

We add to the literature the case of a pale and pathologically cupped disc in a Nigerian male with pituitary adenoma misdiagnosed and treated for glaucoma with resultant bilateral blindness. The aim of this article is to increase awareness that all pathologically cupped discs are not due to glaucoma and to discuss the evaluation of a patient with a pale cupped disc.

Case Report

A 45-year-old male presented with a history of gradual painless vision loss of 1-year duration in the left eye and 6-week duration in the right eye. He had no other ocular or neurologic symptoms. There was no history of galactorrhea, decreased libido, head injury or ocular surgery. He had been diagnosed and treated as a case of glaucoma prior to presentation and was placed on betaxolol and brimonidine eye drops for both eyes. He was neither a known diabetic nor hypertensive.

Visual acuity was no light perception in either eye. Colour vision and visual fields could not be assessed because the patient was blind. There was no proptosis of the globes and the extraocular motility was normal in both eyes. The lids and anterior segments were normal except for total afferent pupillary defect bilaterally. The right disc was pale and pathologically cupped with a cup to disc ratio (CDR) of 0.7 with thinning of the temporal neuroretinal rim, the CDR was 0.5 in the left eye and the neuroretinal rim in both eyes were pale [Figure 1]. The intraocular pressure was 11 mmHg and 15 mmHg in the right and left eye, respectively. Cranial CT scan (ordered due to the clinical features which were atypical of glaucoma) revealed a sellar mass with suprasellar extension suggestive of a pituitary adenoma [Figure 2]. The patient was subsequently referred to the neurosurgeon.

Discussion

A pale pathologically cupped disc is most commonly associated with glaucoma. However, disc pallor and cupping, which mimic glaucomatous disc changes with

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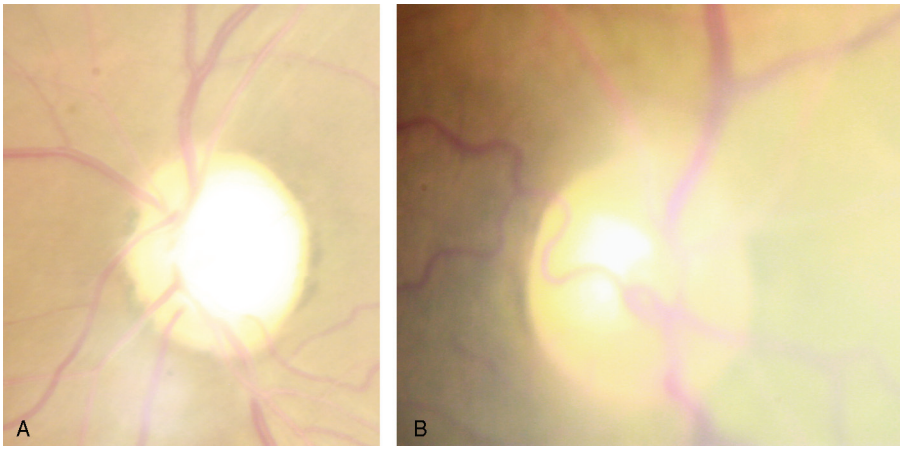

Figure 1: Fundus photographs showing (a) right eye: pale cupped disc with CDR of 0.7; (b) left eye: pale disc with CDR of 0.5

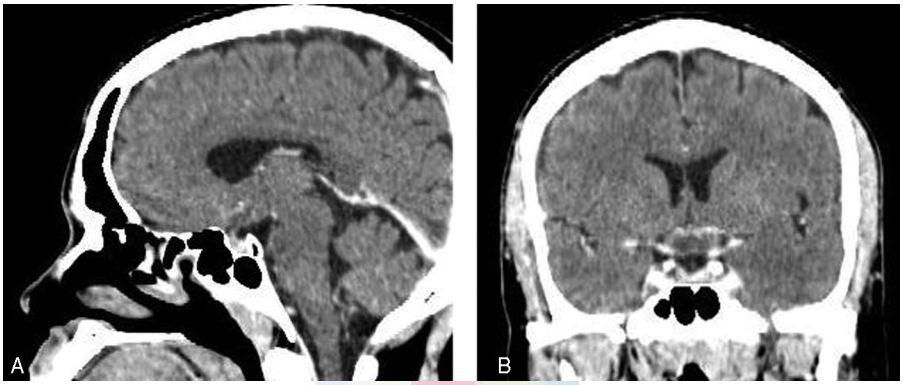

Figure 2: Cranial CT scan with contrast (a) sagittal view; (b) coronal view showing a sellar mass with suprasellar extension

normal or elevated intraocular pressures, have been reported in non-glaucomatous optic neuropathies such as intracranial tumours (including pituitary adenoma),[2-4] anterior ischaemic optic neuropathy,[5] posterior ischaemic optic atrophy,[6] methanol poisoning,[7] lebers hereditary optic neuropathy,[2] central retinal artery occlusion and central retinal vein occlusion[2] and dominant optic atrophy.[8]

Our patient had pituitary adenoma with pathologically cupped disc, which was misdiagnosed as glaucoma with resultant blindness. Vision loss arising from pituitary adenoma is a result of compressive optic neuropathy, which is reversible when it is diagnosed early and promptly treated.[9] The clinician needs to be aware that there are non-glaucomatous causes of pathologic disc cupping, some of which may be life-threatening. A thorough history and examination (including neurologic examination), looking out for atypical features of glaucoma, help to differentiate glaucomatous from non-glaucomatous disc cupping in many cases.[10] In pituitary adenoma, the patient may have symptoms of progressive vision loss, visual field loss, diplopia and headaches; prolactin secreting tumours may present with galactorrhea and amenorrhea in females and impotence and infertility in males.[11] In glaucoma, central visual acuity is preserved until the late stages of the disease after extensive optic disc cupping has occurred.[12,13] Similarly, colour vision loss and visual field defects also occur after extensive optic disc cupping in glaucoma and the remaining neuroretinal rim is pink; in non-glaucomatous optic disc cupping, there is substantial loss of central visual acuity, colour vision and visual field defects (even with mild disc cupping) as well as pallor of the neuroretinal rim.[12,13] Our patient had a history of progressive loss of vision in both eyes, even in the left eye with a presenting CDR of 0.5; the neuroretinal rims were also pale bilaterally. In a study on glaucomatous and non-glaucomatous optic disc cupping, Trobe *et al.*[14] analyzed four optic disc features – neuroretinal rim pallor, obliteration of the neuroretinal rim, thinning of the neuroretinal rim and lamina dots. Pallor of the neuroretinal rim was found to be 94% specific for non-glaucomatous optic disc excavation, whereas focal or diffuse obliteration (complete loss) of neuroretinal rim was 87% specific for glaucoma. Thinning (partial loss of thickness) of the neuroretinal rim was 47% specific for glaucoma, while lamina dot sign was not useful in making a distinction between glaucomatous and non-glaucomatous optic disc cupping.[14]

In glaucoma, the visual field defects respect the horizontal midline while in intracranial tumours affecting the visual pathway, the field defects commonly respect the vertical meridian except when the disease is advanced.[10] Visual field defects that are atypical for glaucoma include field defects that respect the vertical meridian, altitudinal

defects, central or caecocentral scotomas.[10] Confrontation visual field (CVF) testing, if done routinely, can help in detecting atypical visual field defects in the clinic. In a study comparing CVF test with automated perimetry, CVF test was found to have a high specificity (97%) and a high positive predictive value (96%), which implies that the identified visual field defects on CVF test are likely to be true.[15] Nevertheless, formal qualitative perimetry should also be documented. Limitation of eye movements (from compression of the 3rd, 4th or 6th cranial nerves) and corneal and facial hypoesthesia (from compression of the ophthalmic division of the trigeminal nerve) may result with tumour extension into the cavernous sinus. These findings are red flags to the fact that the aetiology of the disc cupping is not due to glaucoma.

In a patient with a pale cupped disc, the optic disc morphology should be evaluated and correlated with the amount of visual dysfunction (visual acuity, colour vision and visual field defects). Respect for the horizontal or vertical midline should be looked out for in the visual field defects. A complete neuro-ophthalmic evaluation should be done. Patients with clinical features atypical of glaucoma should have neuroimaging of the brain and orbits and appropriate laboratory work-up done.[10]

Conclusion

A pale cupped disc is not always due to glaucoma. It has been reported in other causes of optic neuropathy. Thorough history and examination help to differentiate non-glaucomatous from glaucomatous disc cupping in many cases. Those with clinical features atypical of glaucoma should have neuroimaging and appropriate laboratory test done.

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Conflicts of interest

There are no conflicts of interest.

References

1. Kanski JJ. Clinical Ophthalmology: A Systematic Approach. 6th ed. Philadephia: Elsevier Butterworth-Heinemann; 2007. p. 809-10.
2. Zhang YX, Huang HB, Wei SH. Clinical characteristics of nonglaucomatous optic disc cupping. Exp Ther Med 2014;7: 995-9.
3. Qu Y, Wang YX, Xu L, Zhang L, Zhang J, Zhang J, *et al.* Glaucoma-like optic neuropathy in patients with intracranial tumours. Acta Ophthalmol 2011;89:e428-33.
4. Choudhari NS, Neog A, Fudnawala V, George R. Cupped disc with normal intraocular pressure: The long road to avoid misdiagnosis. Indian J Ophthalmol 2011;59:491-7.
5. Sebag J, Thomas JV, Epstein DL, Grant WM. Optic disc cupping in arteritic anterior ischemic optic neuropathy resembles glaucomatous cupping. Ophthalmology 1986;93:357-61.
6. Sonty S, Schwartz B. Development of cupping and pallor in posterior ischemic optic neuropathy. Int Ophthalmol 1983;6:213-20.
7. Shin YW, Uhm KB. A case of optic nerve atrophy with severe disc cupping after methanol poisoning. Korean J Ophthalmol 2011;25:146-50.
8. Fournier AV, Damji KF, Epstein DL, Pollock SC. Disc excavation in dominant optic atrophy: Differentiation from normal tension glaucoma. Ophthalmology 2001;108:1595-602.
9. Elgamal EA, Osman E, El-Watidy S, Jamjoom Z, Hazem A, Al-Khawajah N, *et al.* Pituitary adenomas: Patterns of visual presentation and outcome after transsphenoidal surgery – An institutional experience. Internet J Ophthalmol Vis Sci 2007;4.
10. Moster ML, Kay MD. Glaucoma: The neuro-ophthalmologic differential diagnosis. J Curr Glaucoma Pract 2008;2:33-8.
11. Gittinger JW Jr. Tumours of the pituitary gland. In: Miller NR, Newman NJ, Biousse V, Kerrison JB, editors. Walsh and Hoyt's Clinical Neuro-ophthalmology. 6th ed. USA: Lippincott Williams and Wilkins; 2005. p. 1533.
12. Greenfield DS. Glaucomatous versus nonglaucomatous optic disc cupping: Clinical differentiation. Semin Ophthalmol 1999;14:95-108.
13. Sadun AA, Argawal MR. Topical diagnosis of acquired optic nerve disease. In: Miller NR, Newman NJ, Biousse V, Kerrison JB, editors. Walsh and Hoyt's Clinical Neuro-ophthalmology. 6th ed. USA: Lippincott Williams and Wilkins; 2005. p. 212-3.
14. Trobe JD, Glaser JS, Cassady J, Herschler J, Anderson DR. Nonglaucomatous excavation of the optic disc. Arch Ophthalmol 1980;98:1046-50.
15. Shahinfar S, Johnson LN, Madsen RW. Confrontation visual field loss as a function of decibel sensitivity loss on automated static perimetry. Implications on the accuracy of confrontation visual field testing. Ophthalmology 1995;102:872-7.