CASE REPORT

Giant prolactinoma mimicking low-tension glaucoma at presentation

David Karl,^{1,2} Stewart Neil Gillan,^{1,2} Colin Goudie,² Roshini Sanders²¹Ophthalmology Department, NHS Tayside, Dundee, Angus, UK²Ophthalmology Department, Queen Margaret Hospital, Dunfermline, UK

Correspondence to

Dr David Karl,
david.karl@nhs.net

Accepted 19 January 2015

SUMMARY

We describe a case of a 68-year-old man, referred by his optometrist with suspected low-tension glaucoma, who presented with advanced cupped optic discs and field of vision loss that were subsequently found to be due to a giant prolactinoma. Failing vision in low-tension glaucoma suspects should have a low threshold for neuroimaging.

BACKGROUND

Pituitary adenomas comprise a heterogenic group of lesions, which account for 10–15% of all intracranial tumours, with an estimated prevalence rate of 16.7%. About 1 in 600 persons have a macroadenoma (≥ 10 mm, defined as giant if ≥ 40 mm) that can cause a wide spectrum of visual problems. Patients 'classically' present with bitemporal quadrantanopia or hemianopia due to compression of the optic chiasm.

Referral with suspected glaucoma is a very common ophthalmic presentation accounting for 20% of all referrals from community optometry to hospital eye services (HES). Low-tension glaucoma (LTG) is a subgroup of these referrals, accounting for about 30%.

We describe a patient referred with suspected LTG who presented with advanced cupped optic discs and progressive field depression, which were subsequently found to be due to a giant prolactinoma.

CASE PRESENTATION

A 68-year-old man was referred by his optometrist to HES with suspected LTG due to repeatedly irregular visual field test results, advanced optic disc cupping, normal intraocular pressures (IOPs) and a family history of glaucoma. The patient subjectively felt that vision in his 'good' left eye (LE), which normally had a visual acuity of 6/6 N5, started to

deteriorate 6 months earlier; at the point of referral it was best corrected to 6/7.5 N6. His right eye (RE) was known to be amblyopic with a visual acuity of 6/18 N12. His medical history included considerable risk factors for systemic vasculopathy, such as hypertension, hypercholesterolaemia, 50 pack-years of smoking and type 2 diabetes with no diabetic retinopathy. Despite detailed questioning, he denied any new systemic symptoms apart from experiencing increased lethargy.

Clinical examination at HES revealed advanced bilateral cupped optic discs with a cup-to-disc ratio right 0.9 (90%) and left 0.8 (80%; [figure 1](#)). Furthermore, the findings from the referring optometrist of equal and reactive pupils, normal eye movements and IOPs in mid-teens were confirmed. Computerised perimetry showed constricted visual fields in both eyes, albeit with considerably raised false-negative error rates in excess of 30% ([figure 2A](#)). Since this can be a result of poor concentration and attention span with testing compounded, especially with an amblyopic RE, the field test was repeated 4 weeks later with similar results ([figure 2B](#)).

At this visit, the patient experienced further deterioration of his 'good' LE. Visual acuities measured 6/24 (loss of one Snellen line) N36 in his amblyopic RE, and 6/18 (loss of two Snellen lines) N36 in his LE, which now also showed depressed colour vision. An urgent scan of the patient's head and orbit was requested to rule out a compressive lesion and blood analysis was carried out to screen for other causes of optic neuropathy including vitamin B12, folate levels and inflammatory markers.

INVESTIGATIONS

MRI revealed a large mass centred at the pituitary fossa measuring approximately 4.5 cm $\times$ 5.1 cm $\times$ 4.8 cm ([figure 3A–C](#)) extending into the suprasellar cistern and infrasellar sphenoid sinuses. The optic

Figure 1 Left and right optic disc at presentation: note the pallor and increased cup-to-disc ratio.

CrossMark

To cite: Karl D, Gillan SN, Goudie C, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2014-207634

Figure 2 Automated visual field analysis: (A) initial test, (B) repeat test and (C) after medical treatment.

Figure 3 MRI showing giant prolactinoma: (A) transverse, (B) sagittal and (C) coronal.

chiasm and both optic nerves were displaced superiorly and compressed on both sides. The mass showed heterogeneous T1 and T2 signals with multiple fluid levels most likely due to previous haemorrhage and low-signal components suggesting calcifications. The appearance was probably due to a craniopharyngioma or haemorrhagic pituitary adenoma.

TREATMENT

The patient was referred urgently to the neurosurgery unit and was found to have markedly raised prolactin levels of greater than 200 000 mIU/L, confirming the diagnosis of a giant prolactinoma. In cooperation with the local endocrinology service, he was started on cabergoline 500 µg and dexamethasone 8 mg.

OUTCOME AND FOLLOW-UP

Within a week this led to a dramatic improvement of the patient's visual fields and acuities to 6/18 RE and to 6/9 LE. His prolactin levels showed a similar dramatic response with levels coming down to 336 mIU/L. Three months later his visual acuity in his RE and LE were 6/18 and 6/5, respectively, and visual field examination showed striking recovery with almost normal results (figure 2C).

DISCUSSION

This case illustrates a common patient pathway from the high street optometrist to HES where patients are referred as glaucoma suspects depending on their visual field results, IOPs and optic disc appearance.

We present an adult patient whose initial symptom was slow gradual visual deterioration in his better eye. In the Mayo clinic series published in 1973, 9% of 1000 patients with adenomas had monocular visual field disturbances.¹ Optic disc cupping was misleading with an underlying diagnosis of giant pituitary adenoma after an initial diagnosis of LTG. In an observational case series, Ahmed *et al*² demonstrated that 6.5% of patients labelled with the diagnosis of LTG had clinically relevant compression of the anterior visual pathway. Our patient's visual field tests were complicated by the effect of amblyopia in the fellow eye and appeared initially to be compatible with advanced LTG, but were of low test reliability due to fixation losses and high false-negative rates. However, in hindsight, this was part of his underlying pathology and could not be attributed to poor cooperation.

Prolactinomas that derive from lactotroph cells are the most common and account for about 40% of all pituitary tumours.³ It is noteworthy that despite close questioning on any associated systemic symptoms the patient declined to admit to reduced libido, erectile dysfunction and depression, all of which he had suffered for several months before his ophthalmic presentation. There was no gynaecomastia. Men tend to present later with larger, rapidly growing, more aggressive prolactinomas than women, possibly because symptoms of hypogonadism are initially less manifest, whereas women tend to present earlier due to immediate irregularities of their menstrual cycle and galactorrhoea.⁴

In summary, our case emphasises the difficulty in diagnosing pituitary adenoma in a patient with cupped optic discs and gradual visual loss. A high index of suspicion needs to be maintained in all patients with a diagnosis of LTG and deterioration of visual acuity. Clinicians, general practitioners and optometrists need to be alert to the possibility of chiasmal compression

or pituitary apoplexy in cases of loss of vision, even if asymmetric or monocular. As was delineated in this case, our patient's sight recovered significantly on medical therapy, making surgical intervention unnecessary. However, any additional delay in definitive diagnosis may have had a detrimental effect on the long-term outcome for visual as well as for systemic health.

Patient's perspective

My vision in my good left eye started to blur and I therefore asked my optician to bring my annual test forward. My optometrist examined my eyes and organised a 3D scan of my optic nerves that apparently did not look normal. I was then referred to the eye clinic and underwent various tests for glaucoma, cataracts and diabetic retinopathy. I got worried and angry of these repeated tests, as I had no diagnosis for some time. At one point, I could hardly see out of my good eye and I was very depressed. I had an MRI scan and within an hour, I was being admitted immediately. At last, my wife and I had a diagnosis that I had a tumour on the pituitary gland, which was producing high levels of prolactin in my system. Good news it was not malignant and could be treated with medication. My vision started returning on the medicine.

Learning points

- ▶ Pituitary adenomas may cause variable visual field defects, optic disc atrophy and cupping by optic chiasm compression resembling spuriously chronic glaucoma.
- ▶ Gradual visual loss in one eye may be the only initial presenting symptom of patients with pituitary adenomas.
- ▶ 'Low-tension glaucoma' with failing vision should have a low threshold for neuroimaging to exclude compression of the optic chiasm.

Contributors DK wrote the report. All the authors contributed to the scientific content and proofreading of the paper. SNG and RS managed the patient. RS obtained consent.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Hollenhorst R, Younge B. Ocular manifestations produced by adenomas of the pituitary gland: analysis of 1000 cases. In: Kohler PO, Ross GT. eds. *Diagnosis and treatment of pituitary tumours*. Amsterdam: Excerpta Medica, 1973:53 (International Congress Series no. 303).
- 2 Ahmed II, Feldman F, Kucharczyk W, *et al*. Neuroradiologic screening in normal-pressure glaucoma: study results and literature review. *J Glaucoma* 2002;11:279–86.
- 3 Colao A. Pituitary tumours: the prolactinoma. *Best Pract Res Clin Endocrinol Metab* 2009;23:575–96.
- 4 Iglesias P, Diez JJ. Macroprolactinoma: a diagnostic and therapeutic update. *QJM* 2013;106:495–504.

Copyright 2015 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow