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Case Report

Pituitary hyperplasia resulting in visual deficit

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Received : 09 December 2022
Accepted : 09 March 2023
Published : 24 March 2023

DOI
10.25259/SNI_1107_2022

Quick Response Code:

ABSTRACT

Background: Pituitary hyperplasia is an infrequent cause of visual disturbance and few such cases have been reported in the literature.

Case Description: We describe the case of a 16-year-old female who presented with a short history of progressive headache and visual blurring. Examination revealed markedly constricted visual fields. Imaging revealed an enlarged pituitary gland. Hormonal panel was normal. Following endoscopic endonasal transsphenoidal biopsy and decompression of the optic apparatus, an immediate improvement in vision was noted. Final histopathological examination revealed pituitary hyperplasia.

Conclusion: In patients with pituitary hyperplasia, visual deficit, and no identifiable reversible causes, surgical decompression can be considered to preserve vision.

Keywords: Hyperplasia, Pituitary, Transsphenoidal, Visual field

INTRODUCTION

Pituitary hyperplasia is an infrequent cause of visual disturbance and few such cases have been reported in the literature. While hyperplasia of the pituitary gland may occur due to pathology such as primary hypothyroidism or gonadal insufficiency,[2] it may also occur physiologically in the absence of such conditions.[1]

We describe the case of an adolescent female who presented with a short history of significant visual field loss due to pituitary hyperplasia. Following endoscopic endonasal transsphenoidal biopsy and decompression of the optic apparatus, an immediate improvement in vision was noted.

CASE PRESENTATION

History

A 16-year-old girl presented to the emergency department with a 10-day history of nonacute headache and associated binocular visual blurring. There was no associated nausea, vomiting, fatigue, or menstrual irregularity. Her medical history was significant for spina bifida, a lumbosacral dermoid tumor which had been operated twice in another institution, most recently 2 years before her current presentation, and hydrocephalus treated by ventriculoperitoneal shunt (now redundant), and subsequent endoscopic third ventriculostomy.

Examination

Neurological examination revealed a Glasgow coma score of 15/15 without any cranial nerve deficits, and no limb deficits apart from a pre-existing left lower limb weakness related to her previous spinal surgery. Formal ophthalmological examination revealed markedly constricted visual fields [Figure 1]. Visual acuity was 6/18 bilaterally. Fundoscopic examination revealed no evidence of papilloedema.

Investigations

Serum investigations revealed a mildly elevated prolactin of 551 mU/L and an elevated adrenocorticotropic hormone (ACTH) level to 24.4 pmol/L with an otherwise normal hormonal panel [Table 1].

Magnetic resonance imaging (MRI) revealed an enlarged pituitary gland measuring 11.5 mm (CC) × 7 mm (AP) × 13 mm (transverse) [Figure 2]. Homogenous enhancement of the pituitary gland and infundibulum was noted post-administration of intravenous gadolinium, with no focal pituitary mass seen. The optic chiasm was seen to be displaced upwards. No features of intracranial hypotension were noted.

Table 1: Hormonal panel

Hormone	Level	Reference ranges
ACTH (pmol/L)	24.4	1.1–13.2
Cortisol, AM (nmol/L)	124	77–452
TSH (mlU/L)	1.46	0.47–3.63
Free T4 (pmol/L)	11.5	8.64–15.74
IGF-1 (ug/L)	334	123–546
Prolactin (mU/L)	551	88–483
LH (IU/L)	5.2	0–13.1
FSH (IU/L)	2.7	0.26–7.7

TSH: Thyroid stimulating hormone, IGF-1: Insulin-like growth factor 1, LH: Luteinising hormone, FSH: Follicle stimulating hormone, ACTH: Adrenocorticotropic hormone

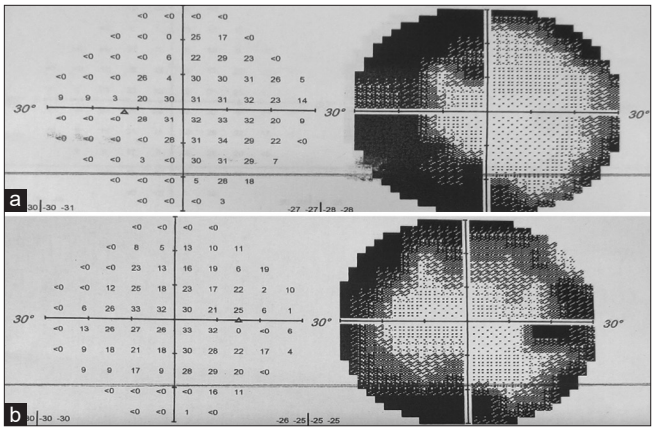

Figure 1: Preoperative visual field assessment. (a) Left eye; (b) Right eye.

Operation

Following input from endocrinology and neurology, the patient was counseled regarding her options and debulking surgery was recommended with the aim of preserving vision. The patient and her guardian consented to surgery.

Following induction of general anesthesia, debulking of this lesion was undertaken through an endoscopic endonasal transphenoidal approach. Following septostomy and sphenoidotomy, the sellar floor was drilled and the dura opened. A firm lesion was noted and biopsies yielded an intraoperative pathological diagnosis of pituitary hyperplasia. The hyperplastic pituitary gland was dissected from the cavernous sinuses and suprasellar arachnoid mater to decompress the optic apparatus. Minimal cerebrospinal fluid leak was noted. The skull base was repaired using synthetic dural graft, autologous fat, and tissue glue.

Pathology

Initial evaluation of tissue through frozen section during surgery did not identify adenoma.

Further examination of the paraffin embedded tissue [Figure 3] identified pituitary tissue with preserved acinar architecture; acini containing admixed eosinophilic, basophilic, and chromophoric cells in varying proportions. Immunostaining, particularly with the pituitary nuclear transcription factors Pit-1, Tpit, and SF-1, reiterated the morphologic impression

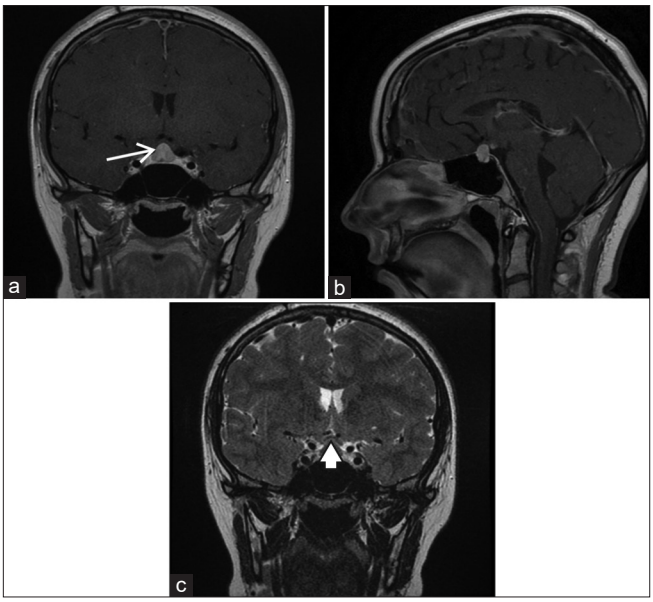

Figure 2: Preoperative magnetic resonance imaging. (a) Gadolinium-enhanced T1 coronal image showing enlarged pituitary gland displaying homogeneous enhancement (arrow); (b) gadolinium-enhanced T1 sagittal image; (c) T2-weighted coronal image showing upward displacement of the optic chiasm (arrowhead).

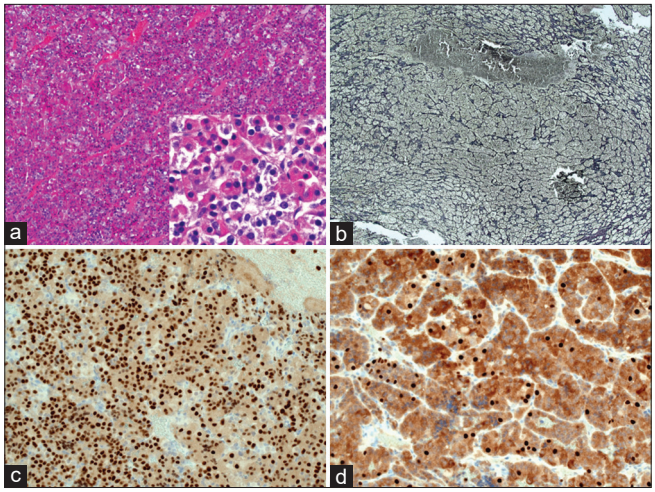

Figure 3: (a) Pituitary hyperplasia (inset) various cell types present include eosinophilic, basophilic, and chromophoric cells. (b) Reticulin histochemistry – acinar structures outlined in black (Gomori's modified reticulin stain) – expanded acini contrast with normally sized examples in the lower half of the photomicrograph. (c and d) Pit-1 immunostaining (Sigma polyclonal) - brown signal labels cell nuclei of Pit-lineage with more infrequent cell nuclei labelled with anti-SF-1 (ThermoFisher clone N1665).

of admixed anterior lobe cell types. Reticulin histochemistry further highlighted expansion of acini without destruction of normal acinar architecture. These appearances were deemed in keeping with pituitary hyperplasia. A pituitary adenoma or other mass forming lesion was not identified.

Postoperative course

Postoperative MRI revealed adequate decompression of the optic chiasm with residual normal pituitary gland [Figure 4]. The patient noted an immediate subjective improvement in her vision. This was confirmed with formal ophthalmological testing [Figure 5].

She required treatment with desmopressin for diabetes insipidus. Following endocrinology input, she was discharged home on maintenance desmopressin and hydrocortisone.

DISCUSSION

Within our unit, patients with pituitary adenomas who present with visual deficit and display evidence of mass effect on the optic apparatus are offered surgery, with the aim of surgical decompression and prevention of progressive visual deterioration. A clear management paradigm for patients with pituitary hyperplasia and visual deficit does not exist and there are few reports of such cases.

Raviv *et al.*[4] describe one such case, where a 24-year-old woman presented with a subacute history of visual deterioration and

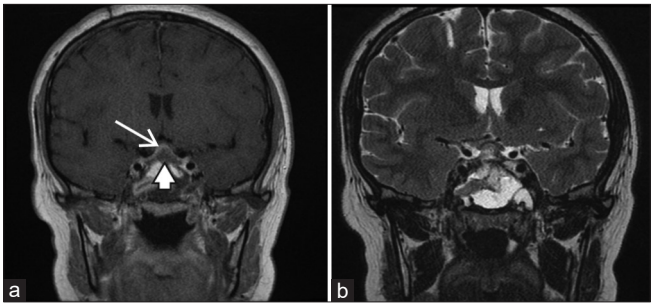

Figure 4: Postoperative magnetic resonance imaging. (a) Gadolinium-enhanced T1 coronal image revealing a central biopsy cavity (arrowhead) with surrounding pituitary tissue (arrow); (b) T2-weighted coronal image revealing reduction in degree of optic chiasmal compression.

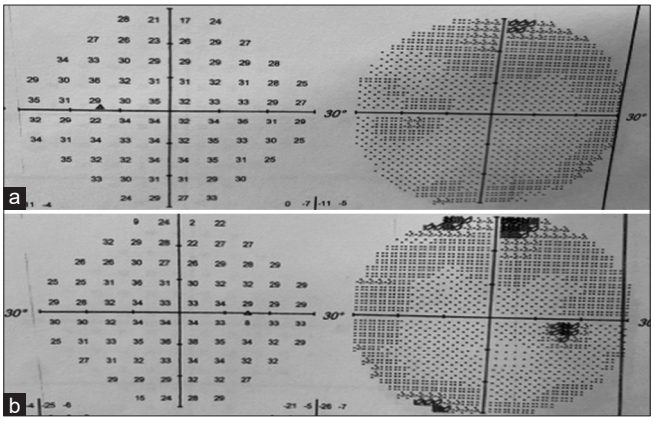

Figure 5: Postoperative visual field examination (a) Left eye; (b) Right eye.

was noted to have pituitary hyperplasia and a normal hormonal profile. Management was initially conservative but due to worsening visual deficit, surgical intervention was eventually undertaken with an immediate improvement in vision postoperatively.

Kinoshita *et al.*[3] describe two cases of pituitary hyperplasia causing visual disturbance in adolescent patients. In one case, a 15-year-old boy presented with initially progressive bitemporal field loss which subsequently improved without intervention after 6 months. The other case is of a 14-year-old girl with acute headache and visual disturbance whose MRI suggested pituitary hyperplasia. Her clinical course was consistent with pituitary apoplexy, and she underwent decompressive surgery with an immediate postoperative improvement in vision. Histopathological examination here revealed pituitary hyperplasia.

On MRI, a pituitary adenoma can often be differentiated from the normal pituitary gland on close examination. Pituitary hyperplasia, however, does not appear as a discrete lesion and instead is associated with an increased overall volume of the pituitary gland. Pituitary size may vary due to size and age. In

our patient, the pituitary gland volume was calculated to be 544 mm^3,[4] 2 standard deviations above the normal volume of the pituitary gland in a female of this age.[5] Pathological causes such as hypothyroidism or gonadal insufficiency were not noted. In the absence of potential reversible causes in a patient with such profound visual disturbance, we elected to proceed with surgical intervention with the aim of preserving visual function.

Notably, our patient's laboratory investigations revealed an elevated ACTH. Although the precise mechanism for this is uncertain, we hypothesize that her previous history of multiple courses of dexamethasone therapy as part of treatment of her lumbosacral dermoid tumor may have played a role; hypoadrenocorticism from steroid therapy is well described and has been known to cause elevated ACTH levels but normal levels of serum cortisol.[6]

To the best of our knowledge, this is the second reported case of profound visual disturbance related to physiological pituitary hyperplasia in which the visual deficit resolved with surgical decompression.

The previous authors have hypothesized that pituitary hyperplasia may be more likely to cause visual deficit than pituitary adenomas due to the relatively firm nature of the tissue,[4] or that certain patients may be more susceptible to visual deficits due to the morphology of the sella turcica.[3]

Given that little is known about the natural history of such presentations and the few reported cases in the literature, it is difficult to know how to optimally manage such patients. In both our case and in the case described by Raviv *et al*.,[4] the patient noted an immediate improvement in vision after surgical decompression. Notably, our patient developed diabetes insipidus in the early postoperative period which may possibly be attributed to the resection/biopsy of pituitary tissue. In our case, the neuropathologist was not certain if this was a pituitary adenoma or pituitary hyperplasia. Given this uncertainty, and the significant visual deterioration, partial debulking was achieved to confirm adequate decompression of the optic chiasm.

With this case, we have added to the currently small body of literature which suggests that in patients with pituitary hyperplasia, visual deficit, and no identifiable reversible causes, surgical decompression can be considered to preserve and/or improve vision. There is not yet sufficient experience to suggest the optimal surgical strategy; however, opening of the sellar bone and dura of the sellar floor with dissection of the pituitary from the surrounding dura and arachnoid layers have been seen to provide benefit in the cases described to date.

Written consent was obtained from the patient before the writing of this case report. All identifying information has been removed. Ethical approval was not deemed applicable in this case.

CONCLUSION

In patients with pituitary hyperplasia, visual deficit and no identifiable reversible causes, surgical decompression can be considered to preserve vision.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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How to cite this article: Altaib WS, Dablouk MO, Jansen M, Khan MH, Kamel M. Pituitary hyperplasia resulting in visual deficit. Surg Neurol Int 2023;14:104.