

MONOCULAR BLINDNESS SECONDARY TO GIANT SELLAR-SUPRASELLAR MASS: A CASE-REPORT

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Abstract

Background: Sellar–suprasellar tumors are of Ophthalmic importance due to their proximity to the visual pathway. They can present with progressive visual impairment from compressing the visual apparatus. Early recognition and prompt surgical treatment is critical to prevent irreversible blindness.

Case Presentation: A 38-year-old male presented with a two-year history of painless, progressive monocular vision loss in the left eye, accompanied by severe headaches, dizziness, and weight gain. Examination revealed no light perception and optic atrophy in the left eye, temporal hemianopia in the right eye, and reduced color vision. Hormonal evaluation showed hyperprolactinaemia (118 ng/ml) consistent with pituitary stalk compression. Neuroimaging demonstrated a giant invasive sellar–suprasellar mass with solid and cystic components, compressing the optic chiasm and encasing the internal carotid arteries. Differential diagnoses included pituitary macroadenoma and craniopharyngioma. The patient was referred for neurosurgical intervention but was lost to follow-up.

Discussion: This case highlights the need for wholistic evaluation of the visual pathway in patients presenting with progressive loss of vision. The clinical features of optic atrophy, temporal hemianopia and increased in prolactin level was a pointer to suprasellar mass. MRI aided in the diagnosis and remain the gold standard for evaluation of suprasellar mass. The ocular and endocrine manifestations demanded an urgent referral in addition to a suggestive radiological findings.

Conclusion: Ophthalmologists may be the first to attend to patients with Suprasellar mass . This demands a high index of suspicion, thorough clinical assessment and appropriate neuroimaging is needed to prevent missed diagnosis and irreversible vision loss and death. Timely multidisciplinary management involving Endocrinologists and Neurosurgeons is crucial.

Keywords: Monocular blindness, compressive optic neuropathy, suprasellar mass, pituitary macroadenoma, craniopharyngioma,

INTRODUCTION

Tumor or mass on the suprasellar region is a common pathology and is thought to be important, because it is adjacent to the vital anterior visual apparatus.¹ Various causes of tumors in this region have been noted including pituitary adenomas, meningiomas, craniopharyngiomas, epidermoid cysts, germinomas, chordomas, among others. Suprasellar region tumors can cause reduction in vision as a result of its compressive effect to optic nerve which leads to compressive optic neuropathy. Damage of the retinal ganglion cell (RGC) axon of the optic nerve is the result of mechanical compression of optic nerve associated with compressive optic neuropathy.² Suprasellar tumors comprise 25% of tumors in the chiasmal region. The three most common tumors are pituitary tumor (50%), craniopharyngioma (25%), and meningioma (10%).³

Ocular manifestations of sellar- supra sellar mass vary among patients, Uche et al reported impairment in visual acuity, headache and endocrinopathy as common manifestations.⁴ A study in Thailand reported common ocular manifestations to be vision loss, ocular pain, diplopia and ptosis, these symptoms were noted to be insidious but progressive.³ Other ocular manifestations that have been reported include blurred vision, visual field defect, loss of colour vision and optic atrophy.⁵ The resultant compressive optic neuropathy can result in permanent monocular or bilateral vision loss; therefore, prompt intervention plays a key role in averting this complication.⁶

Vision loss a common presentation of sellar-suprasellar tumors. The pattern of vision loss may help identify the mass. Lesions anterior to the chiasm can cause unilateral visual loss, while most posteriorly located masses can cause incongruous homonymous hemianopia and chiasmal lesions cause bitemporal hemianopia.⁷

Considering variability of symptoms in patients with sellar-suprasellar tumors, the fact that the ophthalmologist may be the first contact with these patients and the importance of early intervention, this paper hopes to emphasize the importance of high index of suspicion in managing patients who present with gradual onset blurring of vision. This is especially important in resource poor areas where neuro-ophthalmologists are few.

CASE PRESENTATION

The index case is a 38-year-old male patient who presented to the Ophthalmology outpatient clinic with a two-year history of painless, progressive loss of vision in the left eye with no associated history of eye pain, no preceding history of trauma, no double vision or ptosis. However, he reported accompanying severe left-sided headache, sometimes associated with nausea but had no vomiting. He also had dizziness sometimes but never lost consciousness. The patient also agreed to have developed progressive weight gain.

On general examination, he was found to be obese with BMI of 30.5 kg/m², he also had increased breast tissue.

Ocular Examination revealed a visual acuity of 6/9 on the right eye and 'no perception to light' on the left eye. The pupil was reactive on the right eye but was dilated and non-reactive on the left eye. The intraocular pressure was 12mmHg on both eyes. Fundus examination revealed a pale optic disc on the right with a vertical cup-disc ratio of 0.7 and an atrophic optic disc on the left with a vertical cup-disc ratio of 0.5. No papilloedema was noted. Based on the findings, a visual field was requested and the findings on the central visual field resulted in a request for a cranial CT scan and subsequent Brain MRI.

Ancillary Tests: Colour vision on the right eye was found to be reduced, Contrast Sensitivity (Pelli-Robson Chart) was also reduced (1.35 log units) in the right eye. Visual Field Perimetry demonstrated temporal hemianopia on the right eye (**Fig. 1**) while the left eye had no measurable vision.

The hormone profile shows normal Thyroid function indicating that the pituitary gland is producing sufficient TSH. Cortisol level was normal though the prolactin level was significantly elevated (Prolactin level= 118ng/ml); suggestive of Stalk effect.

Neuroimaging contrast-enhanced CT of the brain revealed a sellar and suprasellar mass lesion with widening and erosive remodelling of the pituitary fossa. A large cystic, rim-enhancing suprasellar component producing significant mass effect and rightward midline

shift (**Fig. 2a,2b**). These findings were suggestive of a pituitary macroadenoma with suprasellar extension and cystic degeneration.

Contrast-enhanced magnetic resonance imaging (MRI) of the brain demonstrated a large, well-defined, lobulated, predominantly extra-axial heterogeneous mass centred within the sellar and suprasellar region. The lesion exhibited both solid and cystic components with heterogeneous post-contrast enhancement and surrounding perilesional oedema. Inferiorly, the mass extended into the sphenoid sinus and clivus, causing marked erosion of the sellar floor. Superior extension resulted in compression of the optic chiasm and the floor of the third ventricle, while posteriorly the lesion exerted mass effect on the left cerebral peduncle, basal ganglia, and thalamus. The tumour encased both internal carotid arteries, more prominently on the left. Significant mass effect was evident, with effacement of adjacent brain parenchyma and a 13.3 mm midline shift associated with subfalcine herniation. The cerebral sulci, fourth ventricle, pons, medulla, and cerebellum were otherwise unremarkable. Overall, the imaging findings were suggestive of a giant invasive sellar–suprasellar tumour, with giant pituitary macroadenoma and craniopharyngioma considered the leading differential diagnoses.

The patient was referred to the Neurosurgeon for evaluation and consideration of surgical intervention (removal of the tumor) and subsequent histology. The patient was lost to follow -up.

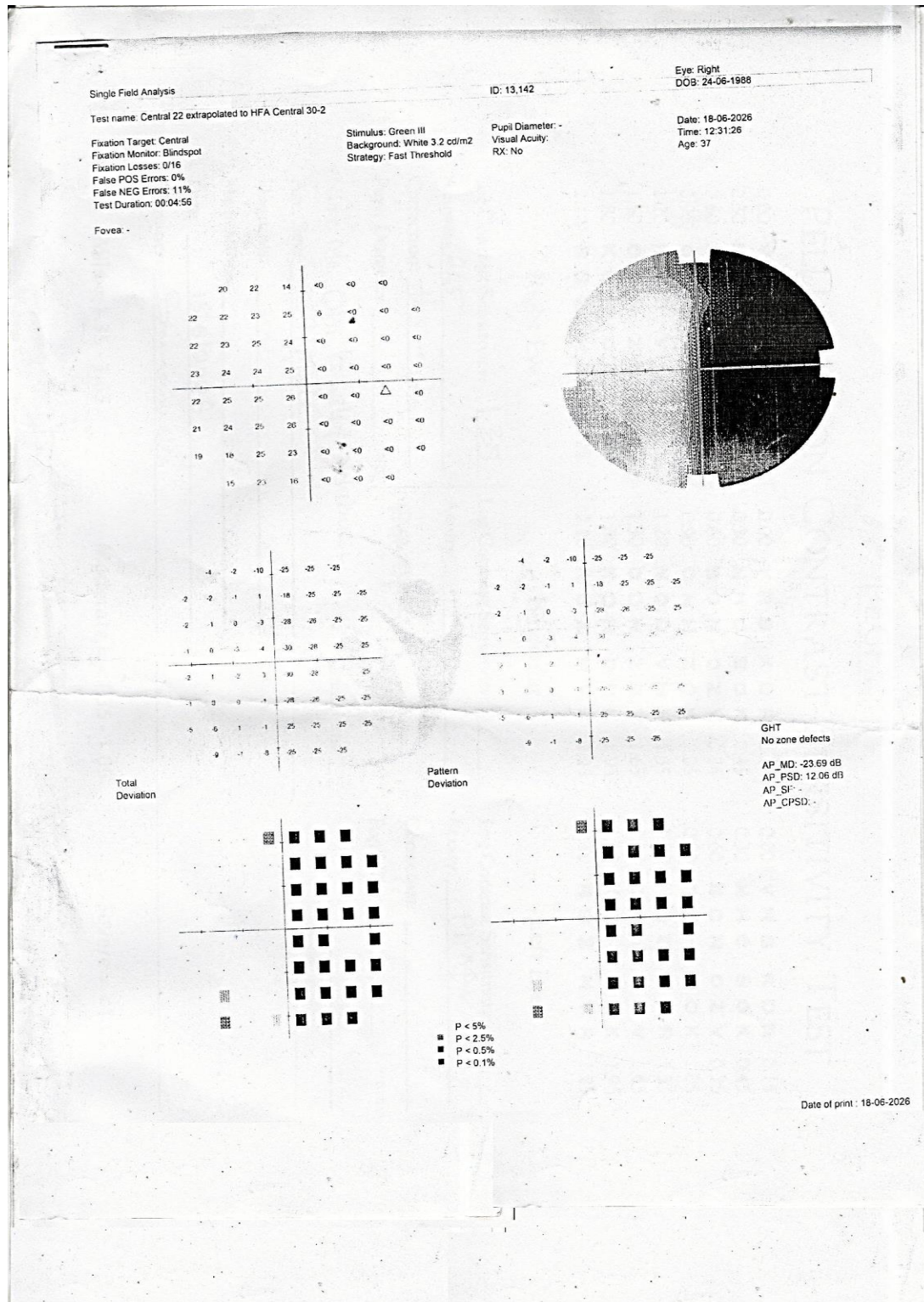
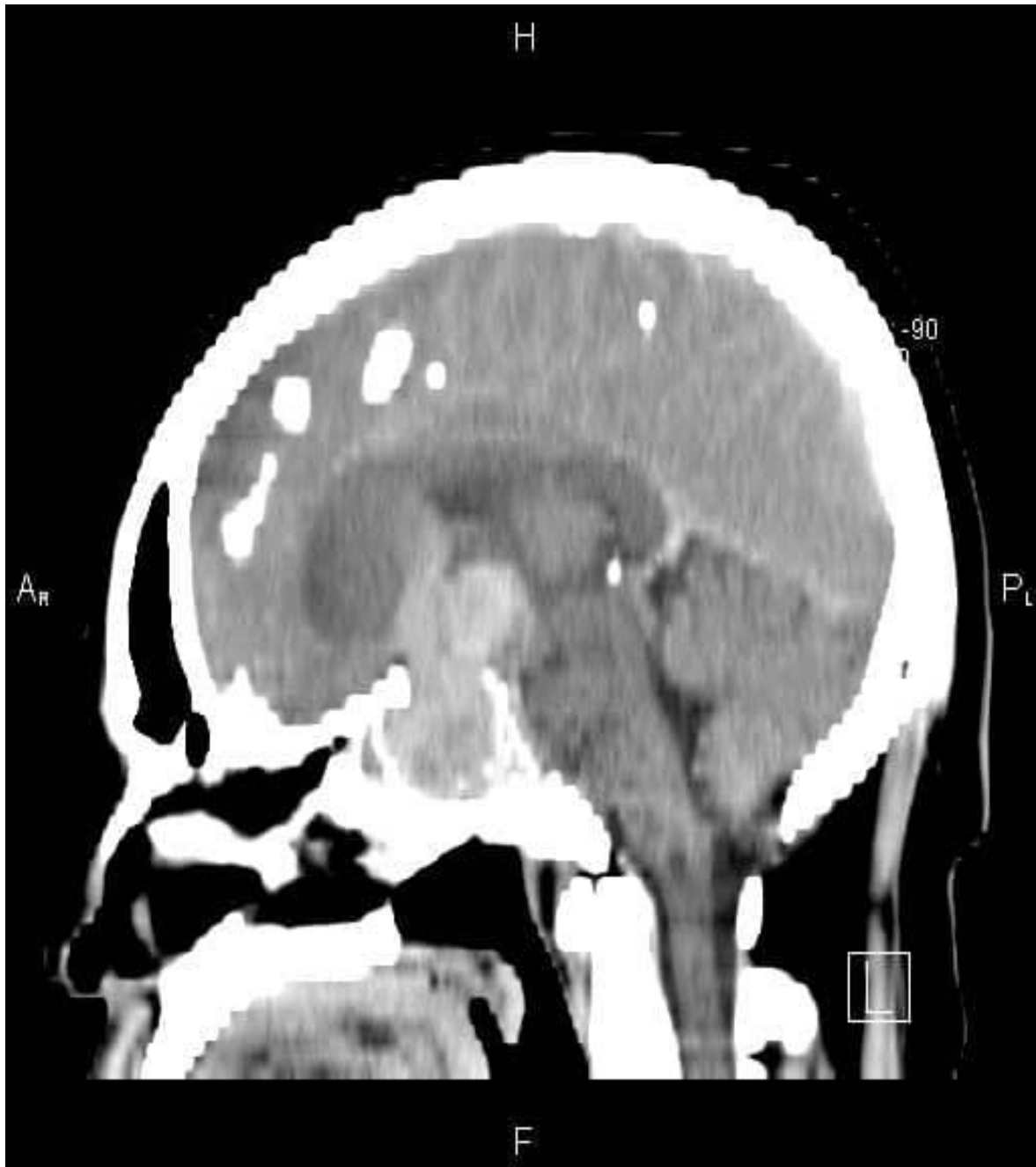


Fig. 1: Automated visual field analysis report showing right dense temporal hemianopic visual field loss, suggestive of chiasmal compression from a suprasellar mass.





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Fig. 2a. and Fig. 2b. Contrast -enhanced CT Brain images (axial and coronal views) demonstrating a giant sellar-suprasellar mass with cystic and solid components, widening

and erosive remodelling of the pituitary fossa, marked mass effect with midline shift to the right.

DISCUSSION

Making a diagnosis of an intracranial mass as an Ophthalmologist requires indebt knowledge of the ocular and non-ocular features of the mass. Our index patient had a large sellar- supra sellar mass with features in keeping with craniopharyngioma. Craniopharyngiomas are benign epithelial tumours derived from the suprasellar region of the brain. Due to their size and location, they can present with variable signs and symptoms which include: ocular features, features of increased intracranial pressure, features of pituitary dysfunction, hypothalamic dysfunction and disturbances in cognitive behaviour.⁸

In our index patient, the only ocular symptom he presented with was unilateral progressive reduction in vision. There was no report of ocular pain, diplopia and ptosis as noted in the study in Thailand.³ Furthermore, in our index patient, even though the ocular history did not tell us so much, objective ophthalmologic examinations revealed unilateral reduction in visual acuity, reduced colour vision and reduced contrast sensitivity. This highlights the need for wholistic ocular examinations in ophthalmic patients in order to avert missed diagnosis. Our patient had optic atrophy which is a recognized feature of large suprasellar masses.⁵ Visual field testing showed temporal hemianopia which signifies a midline mass affecting the chiasm.⁸

Patients with large suprasellar masses can present with features of raised intracranial pressure such as headache, nausea and vomiting, dizziness and loss of consciousness. Our patient presented with a left sided headache and occasional headache and no papiloedema on fundoscopy therefore ruling out the possibility of having developed raised intracranial pressure.⁹

These patients can also present with features pituitary dysfunction such as reduced libido, erectile dysfunction and weight gain.¹⁰ The hormone profile of our index patient showed normal thyroid function and preserved cortisol level, though a significant hyperprolactinaemia (118ng/ml) consistent with Pituitary gland compression- Stalk effect.

Additionally, it is well documented that a Prolactinoma - a Prolactin secreting Pituitary Adenoma is typically characterized by higher Prolactin levels $>200\text{ng/ml}$.

The mass was visualized by the CT scan, and further characterized by MRI highlighting the tumor compression of the left optic nerve and optic chiasm which account for visual loss in the left and temporal hemianopia in the right visual field. MRI remains gold standard for investigating and ruling out Supra- Sellar masses.¹¹

Our patient did not have features hypothalamic dysfunction such as sleep disturbances, diabetes insipidus, increased appetite among others or features of cognitive behaviour impairment such as poor memory, poor concentration and depression. These are also possibilities in patients who have large suprasellar masses.

CONCLUSION

Patients with large suprasellar masses may first present to ophthalmologists. A high index of suspicion, thorough ocular examination, and awareness of systemic features are crucial for timely diagnosis. Prompt neuroimaging and referral for neurosurgical and endocrinological co-management are essential to prevent irreversible vision loss.

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