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Meningioma or Masquerade

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Abstract

A forty-year old male presented with gradual, painless, progressive blurring of vision in his left eyes for the past four months. Old records brought by the patient showed that he was currently on topical steroids and cyclopedias for bilateral anterior non granulomatous uveitis. Examination revealed a pale optic disc with blurred margin in both the eyes. Magnetic Resonance Imaging (MRI) of his brain revealed a meningioma. Were meningioma and uveitis independent entities, or did meningioma typically masquerade as uveitis, the question remains. Masquerade syndromes are a group of disorders that mimic ocular inflammatory disorders that can be infectious or neoplastic

Keywords: Uveitis, Mengioma, Pale disc

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Introduction

An intracranial lesion can result in loss of vision and other ocular signs and symptoms. Pupillary reactions (relative afferent pupillary defect) and optic disc changes provide a valuable clue in this regard. This case report illustrates the importance of such clinical clues which guided the ophthalmologist to the diagnosis of an intracranial tumor.

Case Report

A 40-year-old male presented with four months history of gradual loss of vision of his left eye. Old records brought by the patient showed that he was currently on topical steroids and cyclopegics for bilateral anterior non granulomatous uveitis from some other hospital for the past four months plus he was HLA B-27 positive. There was no other significant medical, surgical, family, and traumatic or drug abuse history. Ocular examination was carried out and his visual acuity was 6/6 in the right eye and 6/36 in the left eye with no improvement on

pinhole. The pupils were already dilated in both his eyes as he was on atropine 1% eye drops. His slit lamp examination revealed a bilateral clear cornea, anterior chamber and crystalline lens. His ocular movements, color vision, and intraocular pressure were normal bilaterally. Fundus examination showed bilateral pale optic disc with blurred margins and surrounding "water marks". (Figure 1). Spontaneous venous pulsations were absent bilaterally. There were no neurological or endocrine symptoms. The laboratory findings (besides already diagnosed HLA B-27 positivity) revealed no abnormalities. His systemic examination was within normal limits.

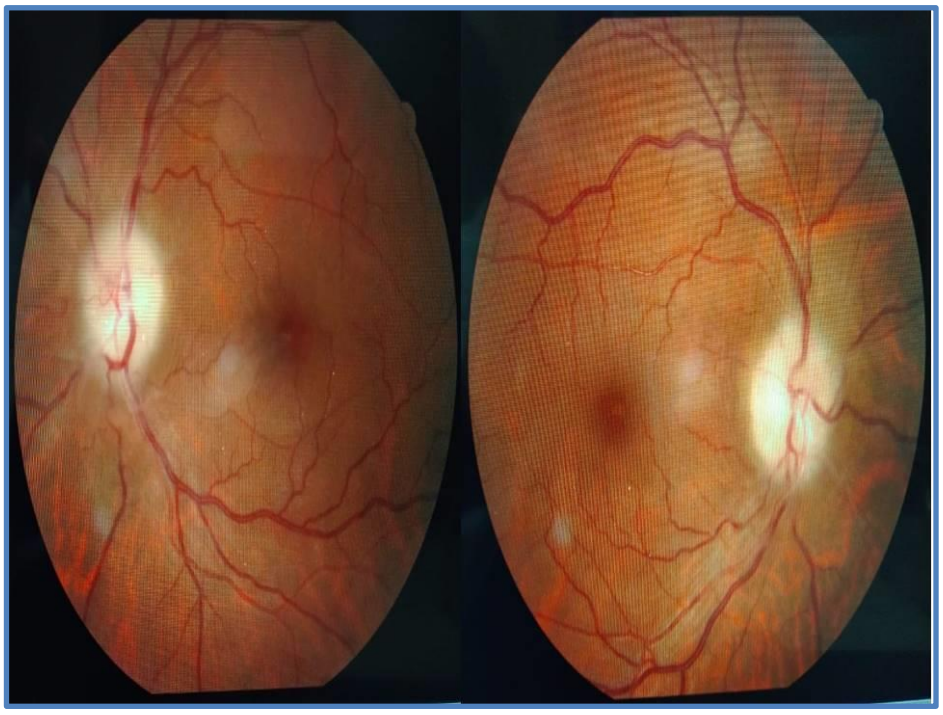

Figure 1: Fundus examination showed bilateral pale optic disc with blurred margins and surrounding "water marks"

An MRI of the brain and perimeter was planned. MRI revealed (Figure 2) "well defined extra axial mass of size about 42><40 mm is seen in left cerebello-pontine angle showing heterogeneous (hyper as well as isointense) signal intensity on T2WI/FLAIR, isointense on T1WI and showing multiple areas of blooming on gradient sequence. It is causing mass effect over brainstem and vermis, which however shows normal signal intensity. Both optic nerves show tortuous course with flattening of sclera. There is evidence of empty sella." The findings were highly

consistent with the diagnosis of calcified meningioma with benign intracranial hypertension. The patient was started on tablet acetazolamide 250 mg, three times a day and was referred to a higher centre for a neurosurgical consultation. Perimetry could not be done as the patient wanted a neurosurgical consultation before any further interventions.

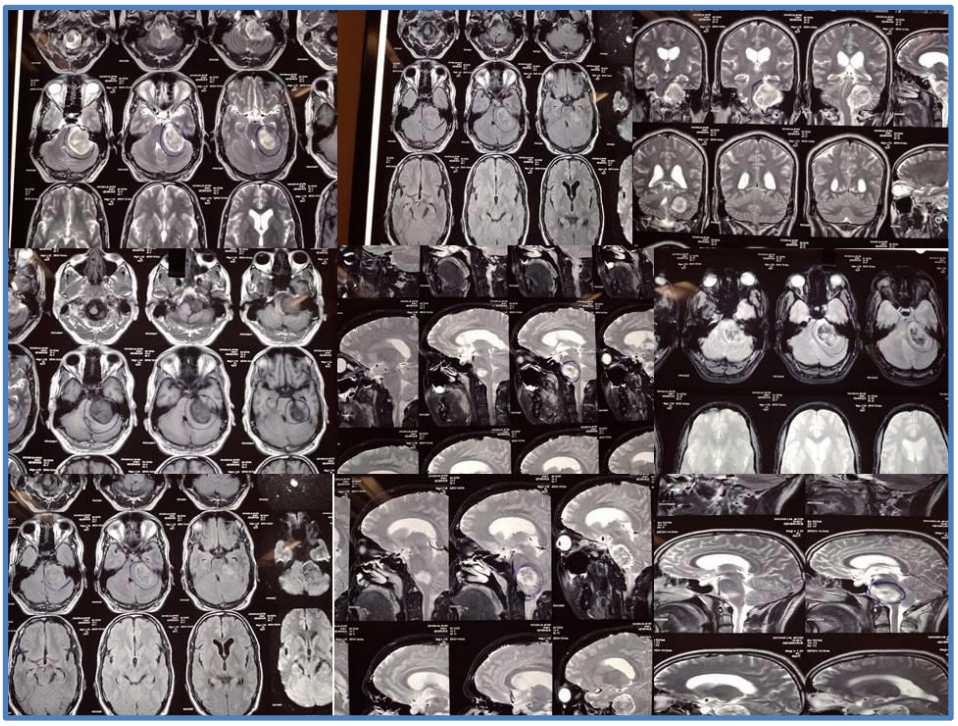

Figure 2: An MRI of the brain and perimetry.

Discussion

Meningiomas are the most common intracranial neoplasms. They are usually benign, more common in females and typically occur in the fourth to sixth decade of life with less than 10% demonstrating anaplastic features or distant metastases. Lung is the most common site for extra cranial distant metastases.[1] The commonest site for intracranial meningioma's is the parasagittal area.[2] These neoplasms occur with greater frequency in conditions such as type 2 neurofibromatosis or multiple endocrine neoplastic type 1.[3] Ophthalmic signs and symptoms in brain tumors include visual loss, double vision, nerve palsy, pupillary abnormalities, and optic nerve head defects.[4] Calcification, ossification, and bone invasion are often associated with these tumors. [5] These are often slow growing tumors. On MRI, the signal is hypo to isointense in T1, iso to hyperintense in

T2 with respect to the cortex. A heterogeneous signal due to tissue necrosis would be linked to a risk of malignancy. Spectroscopy reveals an increase in choline and alanine with a decrease in creatine and N-Acetyl-Aspartate.[6] The World Health Organization classification for meningiomas is based solely on histopathological characterizations of mitotic rate, cellular features of atypia, and local invasion. Treatment modalities include conservative management, surgery, radiation therapy, and stereotactic radiosurgery. Alpha interferon, somatostatin receptor agonists, and vascular endothelial growth factors inhibitors are the only classes of recommended drugs and have previously shown only modest benefit.[7] If calcification is present in a small meningioma, this signifies slow or absent tumor growth prompting the need to closely monitor the lesion without immediate surgical intervention.[8]

Conclusion

Uveitis and meningioma could be separate entities or may be co-related. To date, no such data proving there co-existence has been reported. Intracranial tumors especially lymphomas have been shown to masquerade uveitis. Further studies are warranted.

Conflicts of Interest

The authors declare that they have no competing interest.

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