peer reviewed **CASE REPORT**

Suprasellar meningioma: a case report

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Abstract
This case report discusses a suprasellar meningioma diagnosed in a female patient in her late 30s. The clinical history, investigation, results, aetiology, epidemiology and, radiographic appearance of this pathology are discussed.

Keywords
Bilateral optic atrophy, visual loss, debulking, tumour.

Case report

A female patient in her late 30s was referred from the ophthalmology department to the neurology department at an academic hospital due to her clinical history of progressive loss of vision within the previous three months and severe headaches. She had bilateral optic atrophy and was known to be retroviral disease (RVD) positive and on highly active anti-retroviral treatment (HAART). She had no previous surgical history, and had a Glasgow Coma Scale (GSC) of 15/15 with no light perception. She was sent for an urgent magnetic resonance imaging (MRI) brain scan.

The sequences of the MRI were a fluid attenuation inversion recovery (FLAIR) axial- used for bleeds and tumours to demonstrate oedema surrounding them, T2 weighted axial and T1 weighted post gadolinium. The MRI images depicted an enhancing lobulated mass centred in the suprasellar region measuring 44 x 41 x 58 mm (transverse x anteroposterior x craniocaudal). The mass contained two components: a homogenously enhancing superior component with an inhomogeneously enhancing inferior component (Figure 1). The inferior component of the mass abutted the clivus. The sella did not appear expanded. The mass extended posteriorly to abut the pons. The vessels were not displaced by the mass, but were encased by it. There was no surrounding oedema related to the mass and no hydrocephalus. The fourth ventricle and basal cisterns were patent (Figure 2). The patient underwent bifrontal craniotomy and tumour debulking limited to the intracapsular space. A whitish grey capsule with bluish, soft and vascular tumour with fibrous strands was found. The patient was scheduled for chemotherapy treatment after the surgery.

Discussion

A suprasellar meningioma is a benign tumour of the meninges[1]. The term suprasellar meningioma refers to tumours centered on the region of the planum sphenoidale or tuberculum sellae. These tumours frequently invade the sella turcica (occasionally may be mistaken for a pituitary tumour), diaphragm sellae, optic canals, and medial aspect of the cavernous sinus. Such tumours can also encase the optic nerves within the optic canal and compress the optic nerves, chiasm, or tracts from below and, in some cases, even encase the intracranial portion of the nerve. The supracavernous portions of the carotid artery may be encased by the tumour, although an arachnoid plane frequently protects it[2-4]. Impaired vision is the most common symptom because of tumour compression of the optic nerve and optic chiasma, or the extension of tumour tissue into optic canals and fossa orbitalis[2]. Meningiomas are generally homogenously solid tumours. They may occasionally contain necrosis, scarring, cystic degeneration, and calcifications[5].

Suprasellar meningiomas account for 5-10 % of all intracranial meningiomas[3]. They are more frequent in women than men. Meningiomas can affect individuals at any age, but they usually arise in adults between the ages of 30 and 60 years[6]. Most of these tumours manifest with visual symptoms. It is often difficult to correlate the visual deficit (nerve, chiasm or optic tract) with the actual site of compression in tumours, which is more focal[4]. In this case the site of compression was the optic tract owing to diagnosed bilateral optic atrophy as stated on the patient's post-surgical notes. This inconsistency is due to the vascular involvement of the optic nerve rather than direct compression[4]. The optic nerve contains filaments that are ensheathed by oligodendroglia and not by Schwann cells. This makes the optic nerve very sensitive to injury and leaves it without regenerative potential in adult patients[4]. Suprasellar meningiomas are slow-growing tumours that usually arise from the tuberculum sellae or the chiasmatic sulcus. Due to the close proximity of the optic chiasm and the prechiasmal portion of the optic nerves, either one or the other is compressed, even while the tumour is still small[2]. Whether the chiasm or optic nerve is first affected depends on the clinical scenario and direction of growth of the meningioma, as well as the involvement of the chiasm. A suprasellar meningioma may initially involve the optic nerve rather than the chiasm.

The ophthalmologic signs of suprasellar meningioma are particularly important because of the general absence of other significant clinical features, at least in early cases. Pain or headaches are frequent and visual hallucinations are common[3]. Such tumours can also encase the optic nerves within the optic canal and compress the optic nerves, chiasm,

or tracts from below and, in some cases, even encase the intracranial portion of the nerve. The supracavernous portions of the carotid artery may be encased by the tumour, although an arachnoid plane frequently protects it[2-4]. In this case report the vessels were also encased by the mass (Figure1).

Meningiomas usually show as clear cut areas of increased density because such tumours frequently contain calcification. The extent of the meningioma is clearly visible after enhancement with an intravenous contrast agent. Non-enhanced MRI is not as sensitive as CT for detecting meningiomas because there is not a significant contrast difference between tumours and normal brain tissue[4]. The use of an intravenous gadolinium contrast agent clearly demonstrates meningioma on MRI examinations (Figure 2).

Surgical removal is the method of treatment for a symptomatic meningioma. However, if the meningioma is surgically inaccessible, stereotactic radiosurgery using a gamma knife may be required[6]. Radiosurgery is generally not preferred because of the proximity to the optic nerve and chiasm. When surgical removal is impossible because of the patient's medical condition then methods of shielding the optic nerve or of delivering fractionated radiotherapy are employed[4]. In this case report the patient underwent bifrontal craniotomy and tumour debulking limited to the intracapsular space. She was scheduled for oncology.

Conclusion

This patient underwent surgery and tumour debulking which was uncomplicated. At the time of writing up of this case report the patient was recovering well after surgery. Her headaches and vision had improved and she could walk very well with guidance. It is not always possible to remove the tumour completely due to awkward location and as a result patients often have to undergo radiation treatment as well as drug therapy. Follow-up care after treatment is also important to prevent recurrence[4]. Most of these tumours manifest with visual symptoms and have strong contrast enhancement on MRI scans. This imaging modality is an important diagnostic tool in the evaluation of intracranial tumours. Its effectiveness is due to its inherent high sensitivity to pathologic alterations of normal parenchymal water content,

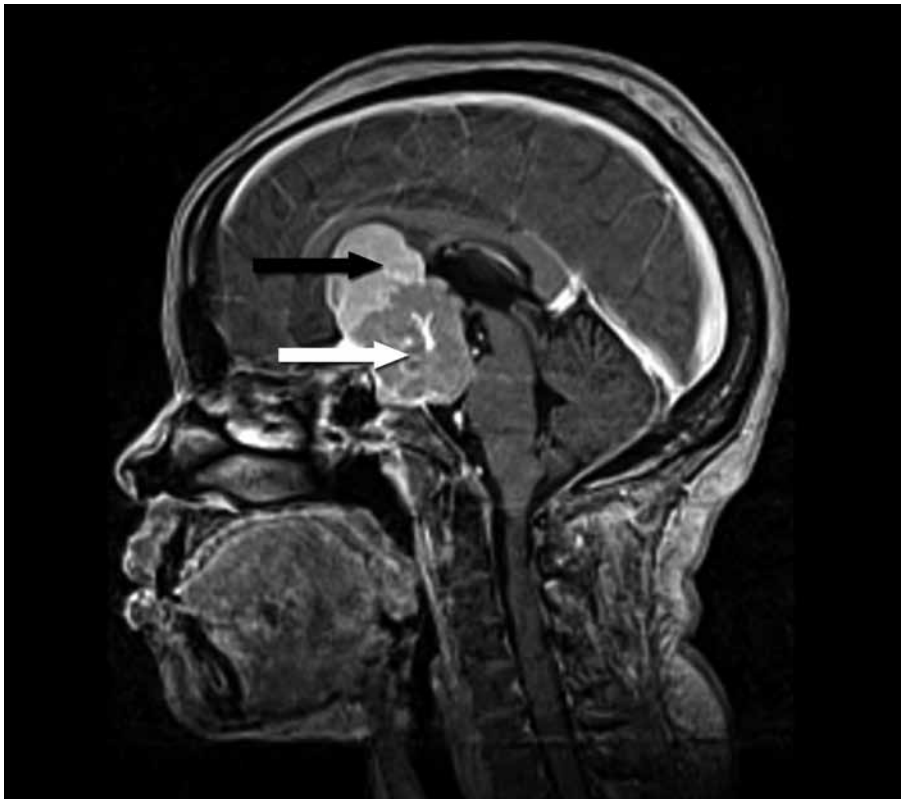

Figure 1: MRI T1 weighted mid-sagittal post gadolinium image, showing a homogeneously superior component (black arrow) with a more inhomogeneously enhancing inferior component (white arrow).

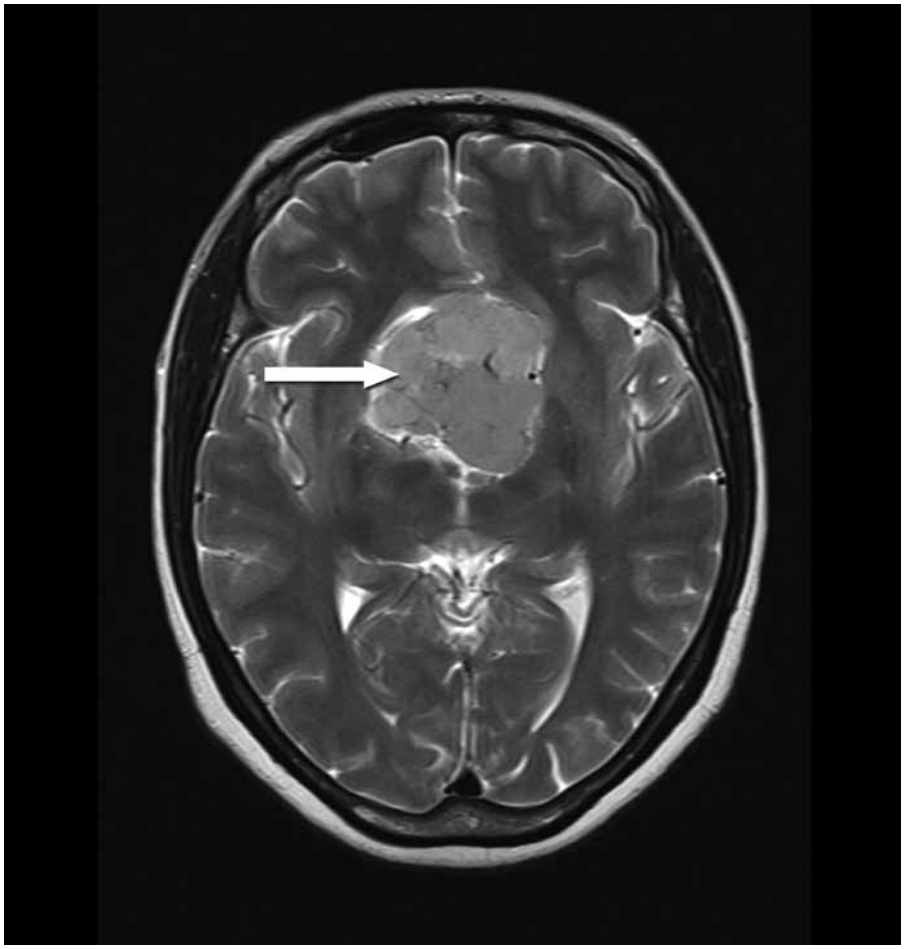

Figure 2: MRI T2 weighted axial view post gadolinium image, showing centrally located mass (white arrow).

as demonstrated by abnormal high and low signal intensity on T2 or T1-weighted images, respectively[7]. Compared to CT, MRI is good in demonstration of oedema of parenchyma which can be an early sign for tumour detection. It also allows accurate delectating extent of oedema and compression effect and better detection of mass effects and atrophy. MRI has a high neuroanatomical definition which is helpful for tissue differentiation and provides accurate detection of vascularity of tumour in various planes acquisition.

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