
LETTERS TO THE JOURNAL

Sir,

Sphenoidal Ridge Meningioma Masquerade: Glaucoma with a Sphenoidal Ridge Meningioma

Meningiomas of the sphenoidal ridge may produce field defects which resemble those produced in glaucoma.¹ This is particularly true in cases initially suspected of having normotensive glaucoma.² Patients with this tumour may therefore be misdiagnosed as having low pressure glaucoma, so that appropriate treatment is delayed. As far as we are aware there have been no reported cases of high pressure open angle glaucoma where the cause of loss of visual field was due to coexistent sphenoidal ridge meningioma.

Case Report

In 1988 a 62-year-old man was seen by his optician for a routine eye test, when he was noted to have high intraocular pressures. He was referred to an ophthalmologist and started on timolol eye drops twice a day. A few months later he was offered a job transfer and was referred to our eye unit for further management.

The patient was asymptomatic with no family history of glaucoma. His general health was good and he was on no systemic medications. On examination, each eye had a visual acuity of 6/5, with normal pupillary reactions, open angles, intraocular pressures of 18 mmHg, and normal-looking optic nerves with a cup/disc ratio of 0.3. He had a full visual field on threshold examination with the Octopus 2000 (Fig. 1a). The patient was therefore diagnosed as having ocular hypertension and the treatment with timolol was stopped, whereupon the intraocular pressure rose to 24 mmHg in each eye.

In 1992, during a routine follow-up visit to the eye clinic, he complained of a dull, intermittent aching pain on the right half of his face for the previous 2 months. There were no visual complaints at this stage. His visual acuity was 6/12 in the right eye and 6/5 in his left eye. The intraocular pressures were 22 mmHg in each eye and the optic discs appeared healthy. Threshold perimetry with the Octopus 2000 showed a diffuse loss of sensitivity in the right eye (Fig. 1b). Repeated intraocular pressure assessments, from 0900 to 2100 hours, showed the highest pressure

in both eyes to be 26 mmHg. The patient was diagnosed as suffering from primary open angle glaucoma and treatment with betaxolol 0.5% was initiated twice a day to the right eye.

On his follow-up appointment 2 months later the patient continued to complain of pain along the whole right half of his face; he also noted that vision in his right eye had deteriorated to 6/24. An afferent pupillary defect was noted and his colour vision showed a severe red-green defect (2/17 Ishihara colour plates) in the right eye. His intraocular pressure was 20 mmHg; the optic disc appearance remained unchanged (Fig. 2). Suprathreshold perimetry with the Henson's field analyser revealed a nasal field defect (Fig. 1c). Computed axial tomography revealed a sphenoidal ridge meningioma on the right side which was partially excised (Fig. 3).

The patient was seen 3 months after his surgery and his visual acuity had improved to 6/6; there continued to be a mild afferent pupillary defect with some recovery of his visual field loss (Fig. 1d). On repeated follow-up visits his intraocular pressures have ranged from 23 mmHg to 26 mmHg. A year later there has been no change in this patient's visual fields.

Comment

Sphenoidal ridge meningiomas have previously been misdiagnosed as low-tension glaucoma.² To our knowledge there are no documented cases of sphenoidal ridge meningioma mimicking primary open angle glaucoma. We were alerted to the possibility of intracranial disease by the development of an afferent pupillary defect with the progressive loss of visual field and loss of colour vision, despite the absence of any appreciable change in the optic disc.

Meningiomas arising from the middle third of the sphenoidal ridge produce no symptoms or signs for many years.³ They eventually invade the optic canal by bony extension of the tumour to cause a field defect. In this case there was no reason to suspect a tumour before the onset of deterioration in the patient's visual function.

The present case demonstrates that there should always be a high index of suspicion of intracranial

asb	0.02-	0.31-	1-	3.1-	10-	31-	100-	315-	1000
	0.25	0.8	2.5	8	25	80	250	800	
Symb.									
dB	47-	35-	30-	25-	20-	15-	10-	5-	0
	36	31	26	21	16	11	6	1	

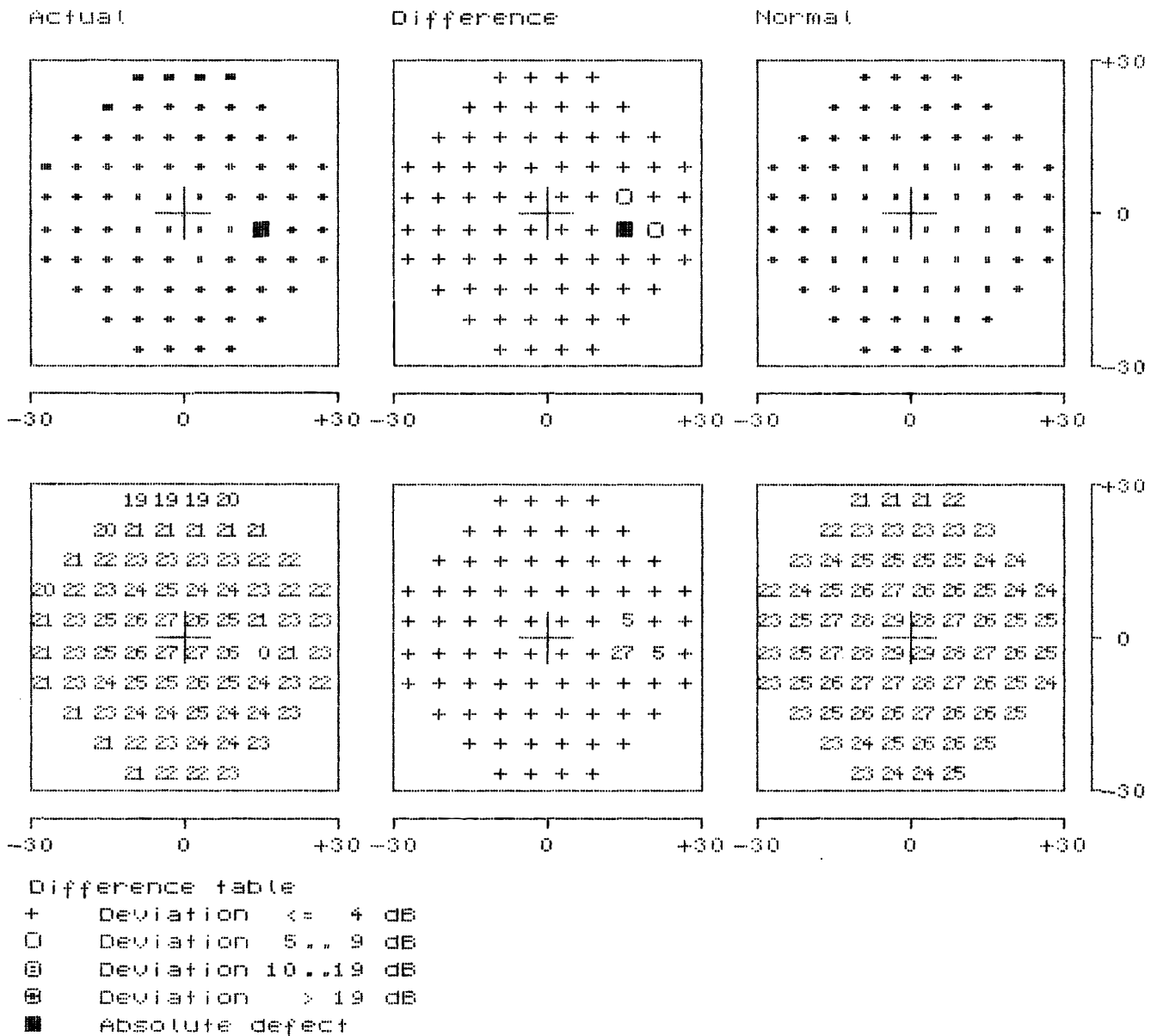


Fig. 1. (a) Normal right Octopus visual field at presentation.

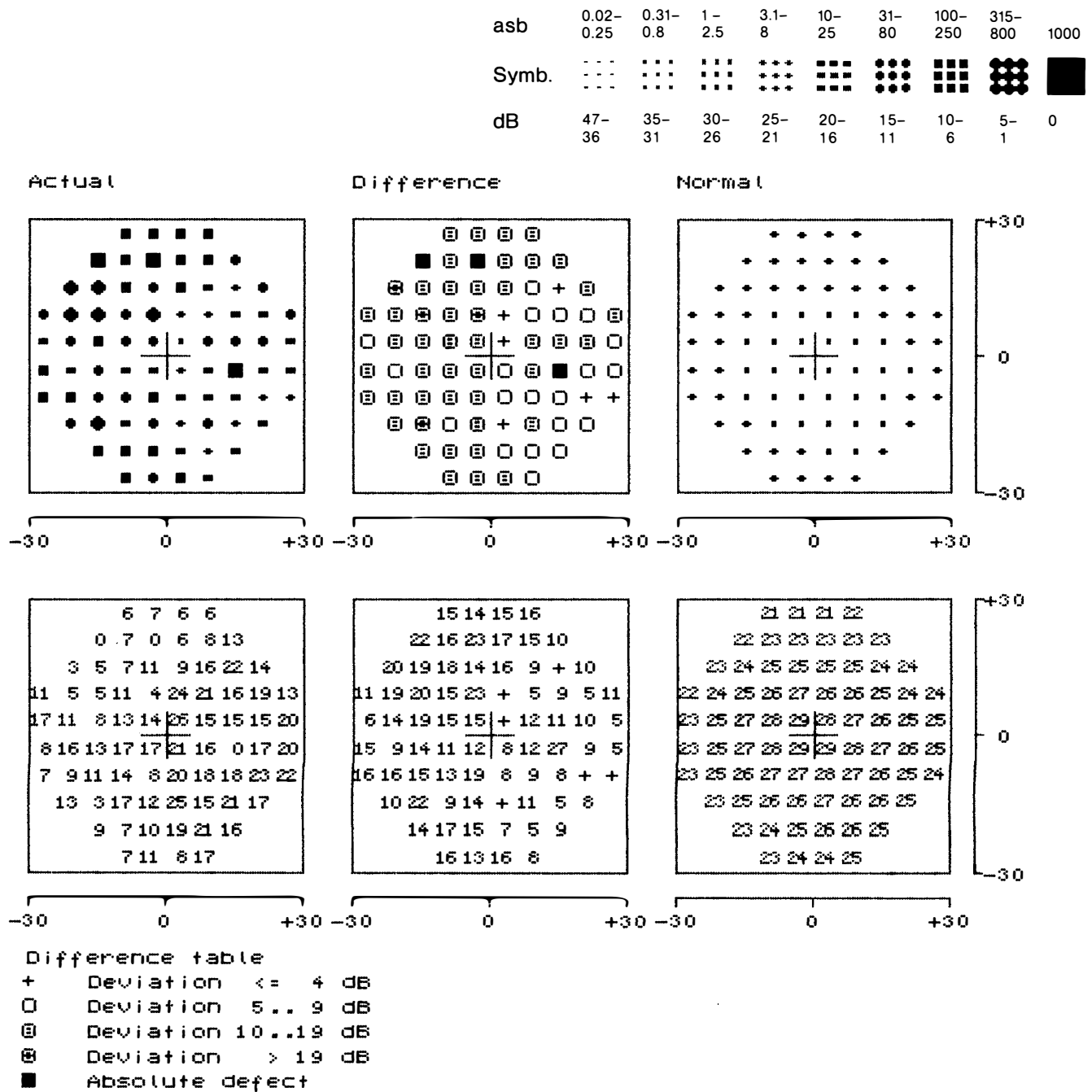


Fig. 1. (b) Diffuse reduction of threshold sensitivity of right Octopus visual field.

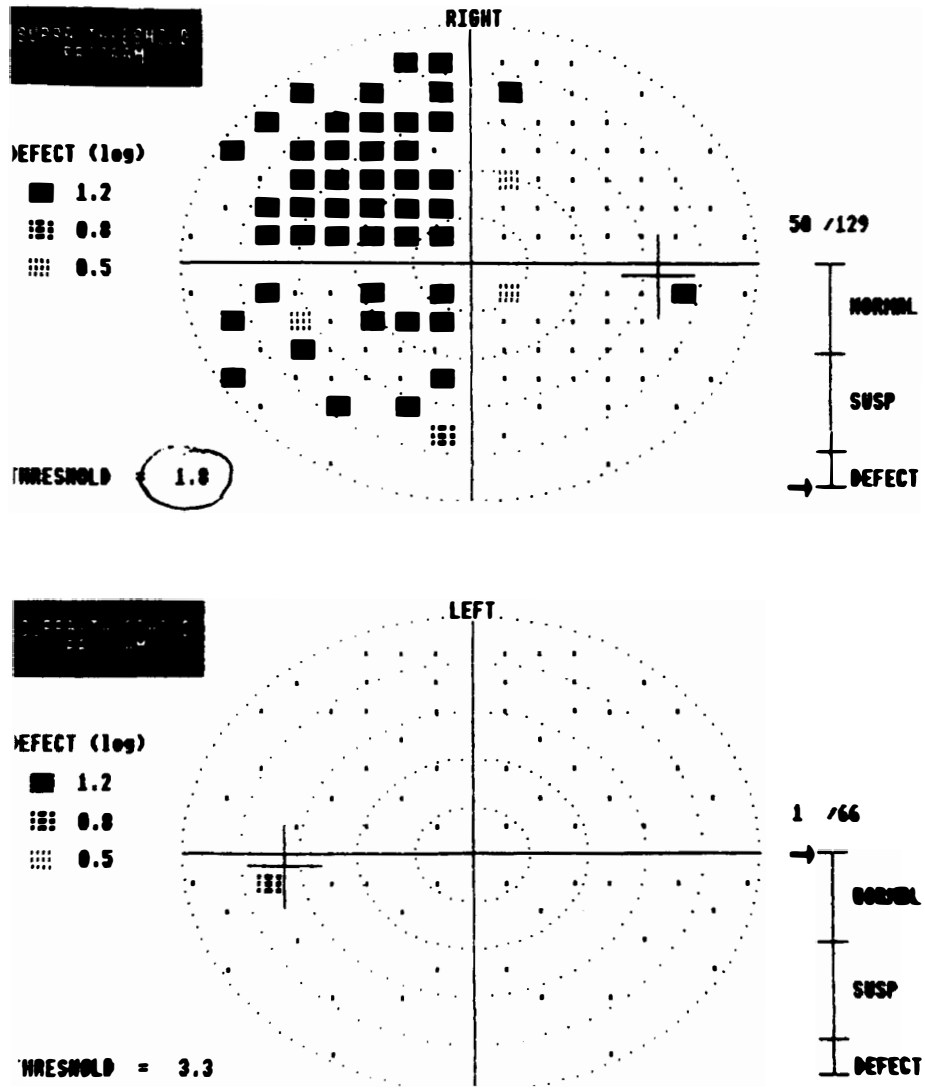


Fig. 1. (c) Nasal field defect right eye at the time of tumour diagnosis: suprathreshold field program.

asb	0.02- 0.25	0.31- 0.8	1- 2.5	3.1- 8	10- 25	31- 80	100- 250	315- 800	1000
Symb.									
dB	47- 36	35- 31	30- 26	25- 21	20- 16	15- 11	10- 6	5- 1	0

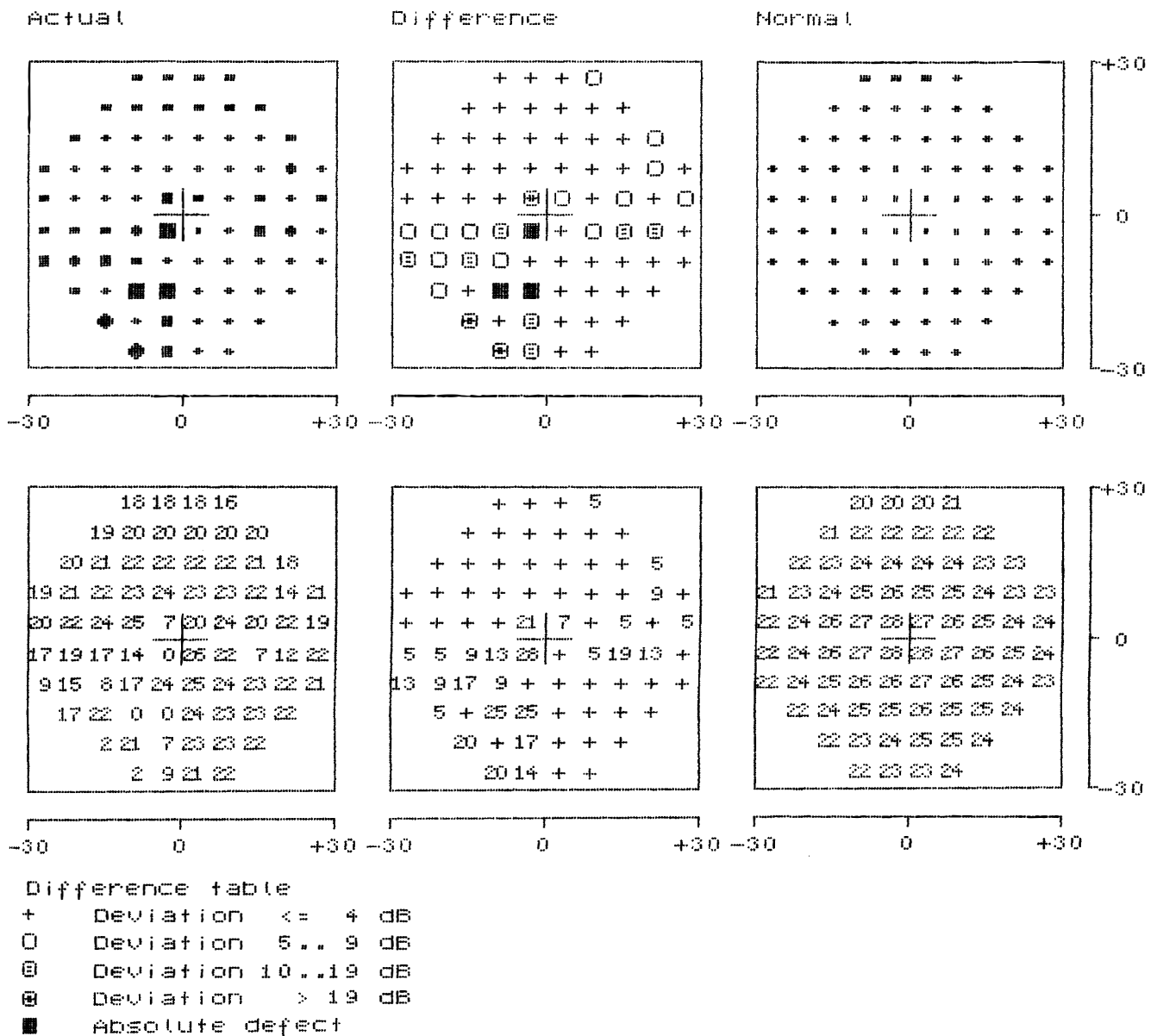


Fig. 1. (d) Recovery of right Octopus visual field defect post-operatively.



Fig. 2. Appearance of the right optic nerve head at the time the tumour was diagnosed.

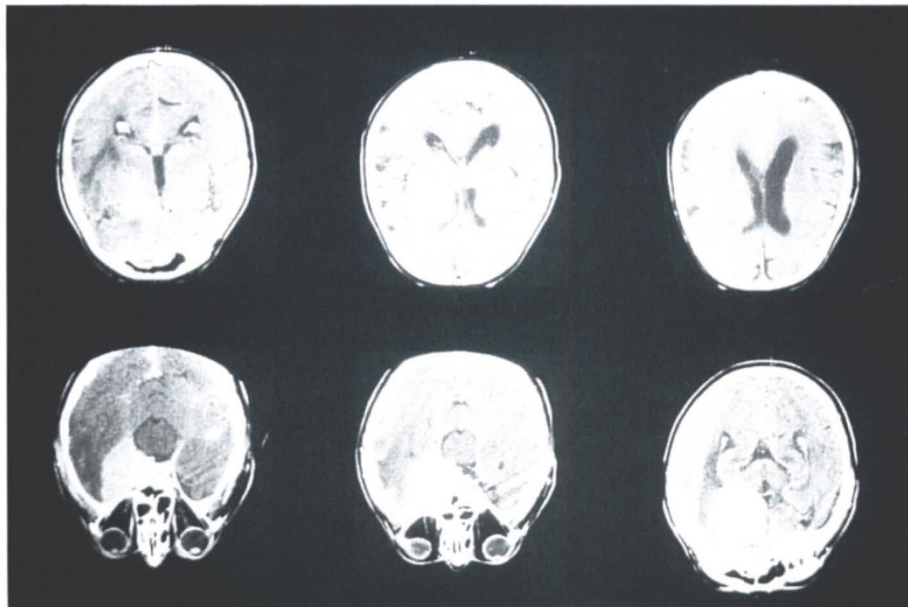


Fig. 3. CT scan of the brain and orbits, showing a large contrast-enhanced sphenoidal ridge meningioma on the right.

disease in all cases of suspected glaucoma whenever the optic disc appearances are not consistent with the visual field defect.

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References

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