actual lasering of setae *in vivo*. We are aware of this being attempted only once, and this involved vitreal hairs being treated with the argon laser.[10] It was noted that there was a subsequent reduction in vitreous activity immediately after treatment.

When the setae have entered the eye with some force, the risks of penetration are high[1,2,8] and subsequent damage so potentially serious that prophylactic lasering may be justified.

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*References*

1. Fraser SG, Dowd TC, Bosanquet RC. Intraocular caterpillar hairs (setae): clinical course and management. Eye 1994;8:596–8.
2. Cadera W, Pachtman MA, Fountain JA, Ellis FD, Wilson FM. Ocular lesions caused by caterpillar hairs (ophthalmia nodosa). Can J Ophthalmol 1984;19:40–4.
3. Becker B. Ein Fall von Ophthalmia pseudotuberculosa hervorgerufen durch Eindringen von Ramarhaaren. Berlin Klin Wochnschr 1892;29:529.
4. Villard H, Dejean CH. L'ophtalmie des chenilles. Arch Ophthalmol 1934;51:719–45.
5. Weiss L. Ein Fall von schwerer Regenbogenhautentzundung hervorgerufen durch in das Augeninnere eingedrungene Raupenhaare. Arch Augenheilkd 1889;20:341.
6. Steele C, Lucas DR, Ridgway AEA. Endophthalmitis due to caterpillar setae: surgical removal and electron microscopic appearances of the setae. Br J Ophthalmol 1984;68:284–8.
7. Corkey JA. Ophthalmia nodosa due to caterpillar hairs. Br J Ophthalmol 1955;39:301–6.
8. Gunderson T, Heath P, Garron LK. Ophthalmia nodosa. Trans Am Ophthalmol Soc 1945;48:151–67.
9. Watson PG, Sevel D. Ophthalmia nodosa. Br J Ophthalmol 1966;50:209–17.
10. Marti-Huguet T, Pujol O, Cabiro I, Oteyza, Roca G, Marsal J. Endophtalmie par poils de chenille intravitréens: traitement par photocoagulation directe au laser à l'Argon. J Fr Ophtalmol 1987;10:559–64.

Sir,

**The Eye and Adenocarcinoma of the Breast: Metastases and Meningiomas**

There have now been a number of reports highlighting the association between adenocarcinoma of the breast and meningioma.[1–3] Breast cancer is the commonest fatal malignant neoplasm of women,[4] and meningioma is one of the commoner intracranial tumours, accounting for 19% of all central nervous system tumours.[2] Carcinoma of the breast is also the second commonest source of intracranial metastases (after carcinoma of the bronchus)[3] and the most frequent source of choroidal metastases.[5,6] We present a patient with advanced carcinoma of the breast whose signs of intracranial mass with cranial nerve involvement were initially ascribed to a metastasis but subsequently found to be due to a meningioma. Detailed ophthalmic examination revealed the presence of an associated choroidal metastasis.

*Case Report*

A 71-year-old woman with known disseminated adenocarcinoma of the breast was referred to the eye unit by the radiotherapy department with a short history of diplopia. She had initially presented 5 years earlier with a large fungating tumour of the right breast and had subsequently received several courses of chemotherapy and radiotherapy for her metastatic disease, which included multiple bony secondaries and malignant pleural and pericardial effusions.

Her initial symptoms consisted of migrainous headaches in the morning, 'trouble focusing' and photopsia in the left eye. Two weeks later she developed diplopia on upgaze and was referred for ophthalmic assessment. Her vision was 6/6 in both eyes but she had a mild left ptosis, an enlarged and unresponsive left pupil and a small left hypotropia (7Δ) and exotropia (5Δ). Elevation of the left eye was limited, particularly in abduction. A Hess chart confirmed this and also revealed a small limitation of adduction supporting the clinical diagnosis of a partial third nerve palsy (Fig. 1). On dilated funduscopy, a pale elevated lesion approximately 3 disc diameters in size with indistinct borders was noted inferonasal to the optic disc in the left eye (Fig. 2). Fluorescein angiography (Fig. 3) showed a diffuse leak and this lesion was thought to be a choroidal metastatic deposit with an overlying serous retinal detachment.

An initial computed tomography (CT) scan of the head revealed a suspicious area adjacent to the pituitary which was thought to represent a secondary deposit. However, whilst the bone scan showed secondaries in the vault, there were no 'hot-spots' in the orbit or base of the skull. Therefore, a repeat CT scan with contrast was performed which showed

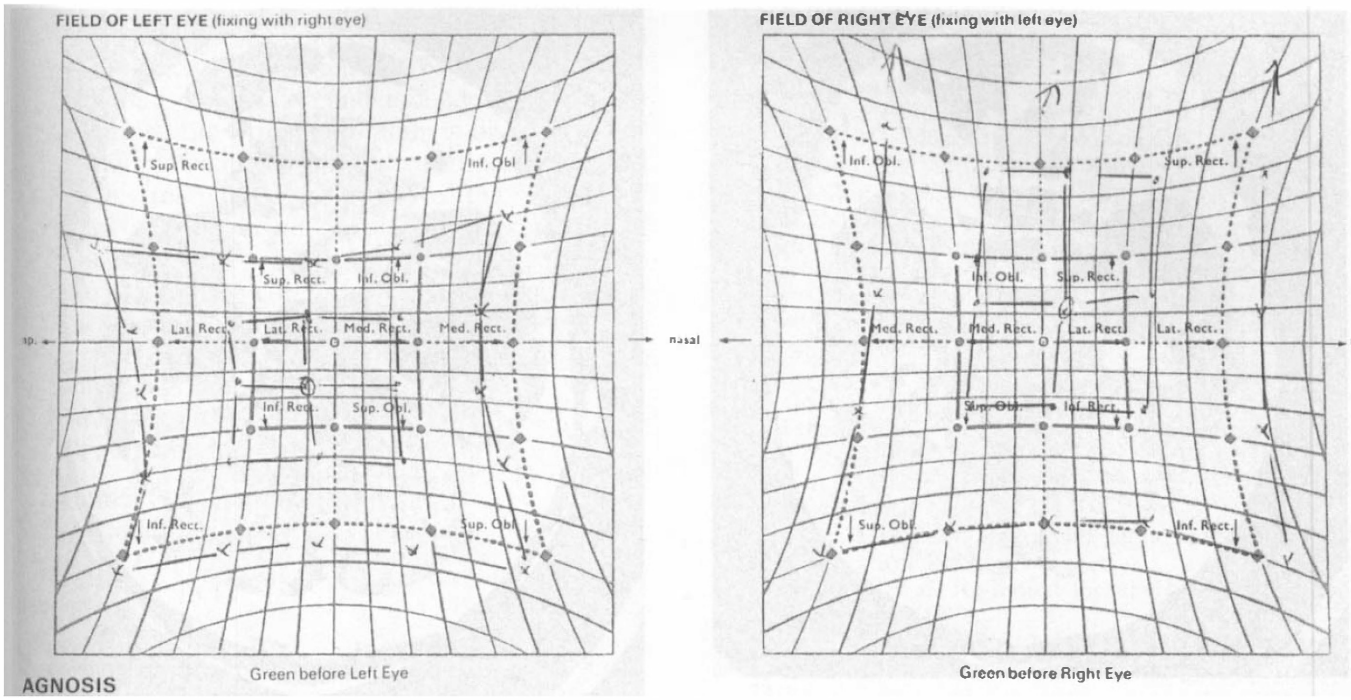


**Fig. 1.** *Hess chart confirming a left partial oculomotor nerve palsy shows left hypotropia and exotropia with reduced elevation and adduction.*

a well-defined, hyperdense mass which enhanced uniformly, characteristic of a meningioma of the right sphenoidal ridge (Figs. 4, 5).

Soon after the onset of diplopia, the patient also required decarubicin and then 3-M (mitomycin C, mitosantrone and methotrexate) chemotherapy as well as radiotherapy for progressive local and metastatic breast carcinoma. In the ensuing months her ophthalmoplegia remained stable and her ocular symptoms were satisfactorily controlled by a Fresnel prism alone. During this time there was no clinically obvious progression of the left choroidal metastasis. Unfortunately, her general condition continued to deteriorate and she died in a hospice 19 months after the onset of her eye problems.

*Discussion*

The unusually high rate of coexistence of breast carcinoma and intracranial meningioma was first reported by Schoenberg *et al.* in 1975[1] and since then a number of case reports have supported this.[2,3,7–9] It has been postulated that the link between the two neoplasms is a sensitivity to female sex hormones. The risk of developing breast cancer is known to be

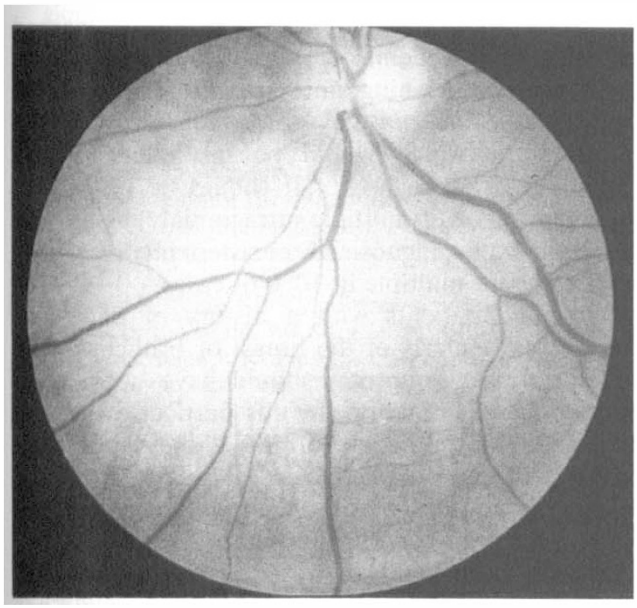

**Fig. 2.** *Fundus photography of the left eye reveals a pale elevated area inferonasal to the disc which represents a choroidal metastasis.*

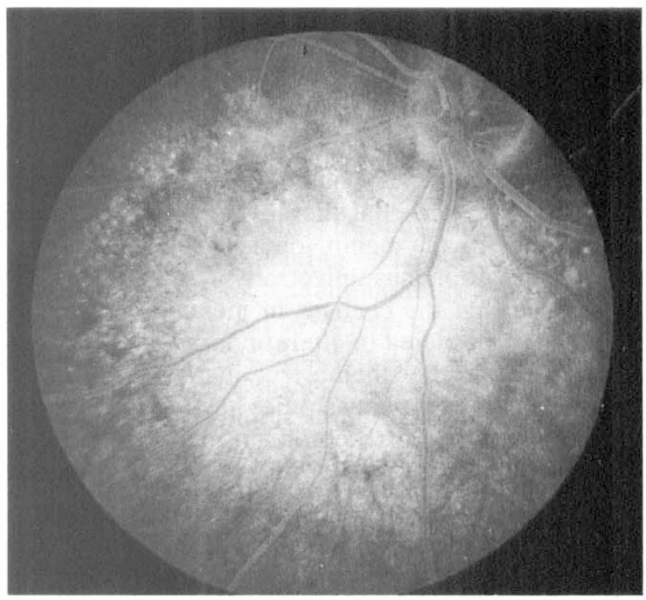

**Fig. 3.** *A fundus fluorescein angiogram of the left eye shows a diffuse leak of fluorescein over the choroidal metastasis.*

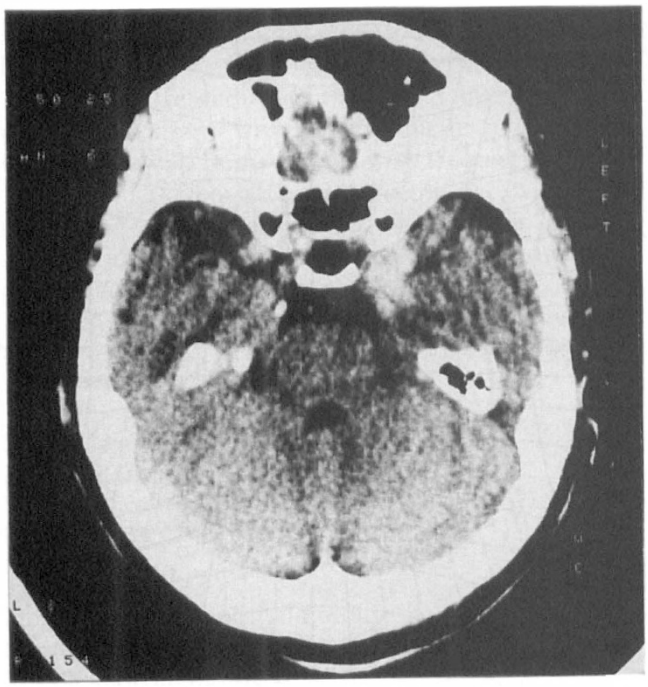

**Fig. 4.** *Computed tomography without contrast demonstrating a hyperdense mass on the right sphenoidal ridge.*

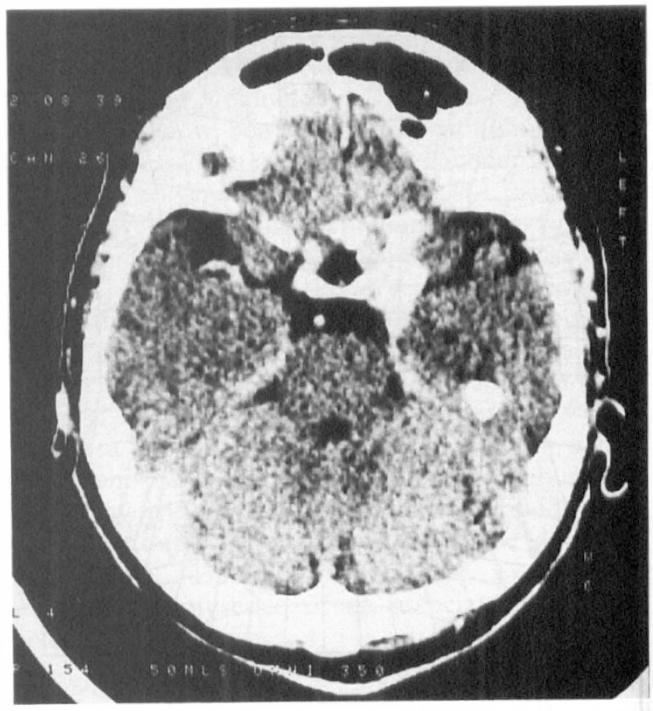

**Fig. 5.** *Computed tomography showing uniform enhancement of a meningioma of the right sphenoidal ridge after intravenous contrast.*

influenced by circulating levels of free oestradiol[10] and the presence of active oestrogen receptors is used as a guide to the likely responsiveness of the tumour to hormonal therapy.[11]

There are a number of persuasive reasons to believe that meningiomas are under hormonal influence. There is a female preponderance (women constitute two-thirds of patients with intracranial meningiomas)[2,12] and there are several published reports of the symptoms and the size of meningiomas being reversibly increased in pregnancy and during the follicular phase of the menstrual cycle.[13–15] The risk of meningioma is reduced in postmenopausal women[16] but is increased in severe obesity where oestrogen levels are raised.[11] Finally, the presence of oestrogen and progesterone receptors within tumour tissue has been documented[11,12] and there is preliminary evidence that hormonal therapy may be helpful in the treatment of meningiomas.[17] However, this theory is by no means universally accepted as there is evidence that the changes in meningioma size in pregnancy are related to alterations in water content or vascularity rather than a true increase in tumour mass.[15] In addition, many studies have failed to show significant oestrogen receptor numbers and activity in meningioma tissue[18,19] or any growth modulation with hormonal treatment.[20]

This case illustrates several important issues in the investigation and management of neurological or visual symptoms in patients with a history of carcinoma of the breast. Firstly, the assumption that an intracranial mass lesion equates with a cerebral secondary may not always be correct, particularly if there is no other evidence of disseminated carcinoma. Relatively benign or treatable space-occupying lesions, such as chronic subdural haematoma and astrocytoma,[8] as well as meningioma, need to be excluded by investigations such as CT with contrast, angiography and magnetic resonance imaging (MRI). Meningiomas have a characteristic appearance on CT, being well-defined, high-density and homogeneously enhancing lesions found in characteristic locations, whereas metastases show poor definition, low density, marked perilesional oedema and non-homogeneous enhancement.[9] Meningiomas are potentially curable by surgery alone and may not be responsive to the radiotherapy which would be instigated for the treatment of metastases.[21] It should be noted that the appearance of multiple intracranial masses does not exclude the diagnosis of coexistent meningiomata since they are multiple in 10% of cases.[3]

Secondly, patients with a history of neoplastic disease, particularly of the breast or bronchus, who develop visual symptoms should have a complete ophthalmic examination. In this particular case, an assessment of the ocular motility alone would have missed the choroidal metastasis. In retrospect the history of photopsia before the onset of diplopia may have been caused by the choroidal lesion and perhaps should have prompted an earlier ophthalmological referral, although the outcome would not have been affected in this patient. A high index of suspicion coupled with a thorough ophthalmic

examination including dilated funduscopy may reveal choroidal secondaries which can be the first sign of disseminated diseases[6,22] and indeed may even be noted before the primary tumour is diagnosed.[23,24] Choroidal metastases are usually radiosensitive and palliative treatment can improve the vision by shrinking the lesion and promoting absorption of subretinal fluid.[24,25] Such treatment may prevent further ophthalmic complications and improve the patient's quality of life during a terminal illness.

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*References*

1. Schoenberg BS, Christine BW, Whisnant JP. Nervous system neoplasms and primary malignancies of other sites: the unique association between meningiomas and breast cancer. Neurology 1975;25:705–12.
2. Burns PE, Jha N, Bain GO. Association of breast cancer with meningioma. Cancer 1986;58:1537–9.
3. Rubenstein AB, Schein M, Reichenthal E. The association of carcinoma of the breast with meningioma. Surg Gynecol Obstet 1989;169:334–6.
4. Donegan WL, Spratt JS. Cancer of the breast, 2nd ed. Philadelphia: WB Saunders, 1979:14–46.
5. Bloch RS, Gartner S. The incidence of ocular metastatic carcinoma. Arch Ophthalmol 1971;85:673–5.
6. Ferry AP, Font RL. Carcinoma metastatic to the eye and orbit. I. A clinicopathologic study of 227 cases. Arch Ophthalmol 1974;92:276–86.
7. Caruso G, *et al.* Meningioma associated with malignant glioma and adenocarcinoma of the breast. Presse Med 1991;20:222.
8. Raskind R, Weiss SR. Conditions simulating metastatic lesions of the brain. Int Surg 1970;53:40–3.
9. Metha D, Khatib R, Patel S. Carcinoma of the breast and meningioma. Cancer 1983;51:1937–40.
10. Franceschi S, *et al.* Breast cancer risk and history of selected medical conditions linked with female hormones. Eur J Cancer 1990;26:781–5.
11. Goffin J. Estrogen- and progesterone-receptors in meningiomas. Clin Neurol Neurosurg 1986;88:169–75.
12. Donnell MS, Meyer GA, Donegan WL. Estrogen-receptor protein in intracranial meningiomas. J Neurosurg 1979;50:498–501.
13. Kempers RD, Miller RH. Management of pregnancy associated with brain tumours. Am J Obstet Gynecol 1963;87:858–64.
14. Bickerstaff ER, Small JM, Guest IA. The relapsing course of certain meningiomas in relation to pregnancy and menstruation. J Neurol Neurosurg Psychiatry 1958;21:89–91.
15. Weyand RD, MacCarty CS, Wilson RB. The effect of pregnancy on intracranial meningiomas occurring about the optic chiasm. Surg Clin North Am 1951;31:1225–33.
16. Schlehofer B, Blettner M, Wahrendorf J. The association between brain tumours and menopausal status. J Natl Cancer Inst 1992;84:1346–9.
17. Blankenstein MA, *et al.* Hormonal dependency of meningiomas. Lancet 1989;1:1381.
18. Courriere P, Tremoulet M, Eche N, Armand JP. Hormonal steroid receptors in intracranial tumours and their relevance in hormone therapy. Eur J Cancer Clin Oncol 1985;21:711–4.
19. Schrell UMH, *et al.* Hormonal dependency of cerebral meningiomas. J Neurosurg 1990;73:743–9.
20. Schrell UMH, *et al.* Hormonal dependency of meningiomas. Lancet 1989;1:1381.
21. Wara WM, *et al.* Radiation therapy of meningiomas. AJR 1975;123:453–8.
22. Bullock JD, Yanes B. Ophthalmic manifestations of metastatic breast cancer. Ophthalmology 1980;87:961–73.
23. Kaiser-Kupfer ML. Role of the ophthalmologist in the therapy of breast carcinoma. Trans Ophthalmol Soc UK 1978;98:184–9.
24. Stephens RF, Shields JA. Diagnosis and management of cancer metastatic to the uvea: a study of 70 cases. Ophthalmology 1982;86:1336–49.
25. Maor M, Chan RC, Young SE. Radiotherapy of choroidal metastases. Cancer 1977;40:2081–6.

Sir,

**Adenocarcinoma Metastatic to the Choroid: Diagnosis by Trans-scleral Biopsy**

We report the use of trans-scleral choroidal biopsy in the diagnosis of a solitary, rapidly enlarging choroidal metastatic deposit, unresponsive to radiotherapy, in a patient with no evidence of systemic malignancy.

*Case Report*

A 68-year-old man in good general health was referred with a history of blurred vision in the inferior field of his left eye for 24 hours. There was no significant past medical or ophthalmic history. On examination, visual acuities were 6/9 right, 6/6 left. External and slit lamp examination of both eyes was normal, but funduscopy revealed a pale choroidal mass in the left eye (Fig. 1). The clinical features, fluorescein angiogram and ultrasound A-scan were suggestive of a metastatic deposit. Orbital CT showed thickening of the sclera, but no calcification or extraocular invasion (Fig. 2). A full general examination including chest radiograph, barium enema, isotope bone scan and abdominal ultrasonography revealed no evidence of systemic malignancy.

Over the following 2 months, the lesion showed rapid growth in all dimensions, and spread to involve the optic disc and macula (Fig. 3), despite external