the National Wilms Tumor Study Group Pathology Center. *Am J Surg Pathol* 2001;**25**:433–44.
8 **Gatalica Z**, Grujic S, Kovatich A, *et al*. Metanephric adenoma: histology, immunophenotype, cytogenetics, ultrastructure. *Mod Pathol* 1996;**9**:329–33.
9 **Tsuji M**, Murakami Y, Kanayama H, *et al*. A case of renal metanephric adenoma: histologic, immunohistochemical and cytogenetic analyses. *Int J Urol* 1999;**6**:203–7.
10 **Grignon DJ**, Eble JN. Papillary and metaephric adenomas of the kidney. *Semin Diagn Pathol* 1998;**15**:41–53.
11 **Renshaw AA**, Freyer DR, Hammers YA. Metastatic metanephric adenoma in a child. *Am J Surg Pathol* 2000;**24**:570–4.

Malignant melanoma of the ciliary body presenting as extraocular metastasis in the temporalis muscle

A man in his 30s presented with an 18-month history of a painless 3×4 cm mass in his left temporalis muscle which had grown rapidly in the previous month. There were no neurological or ocular symptoms. Past medical history was unremarkable. The mass was excised and showed a metastatic pigmented malignant melanoma. Immunocytochemistry using antibodies HMB45 and MelanA were both positive. Postoperatively, the patient underwent adjuvant radiotherapy.

In search for a primary source an in situ superficial spreading pigmented malignant melanoma of the left leg was removed but was not regarded as the primary lesion. Ophthalmological examination detected a pigmented mass in the inferotemporal quadrant of the left iris/ciliary body with pigmented cells seeding the vitreous. Intraocular pressure of the left eye was markedly raised, and the iris architecture was distorted. Fundoscopy showed multiple hyperpigmented choroidal foci involving the posterior pole of the left eye (fig 1A). CT scans of the neck, thorax, abdomen and pelvis were all normal.

The enucleated left globe confirmed a primary pigmented malignant melanoma of the ciliary body, with maximum thickness of 2 mm (fig 1B). It focally involved the full thickness of the ciliary body, spread into the lateral aspect of the iris and invaded the trabecular meshwork. Multi-focal satellite-type nodules were found in the choroid, each surrounded by a dense lymphocytic response, predominantly made up of CD8-positive T-cells with a smaller number of CD20-positive B-cells (fig 2A). Within the ciliary body there were two distinct patterns of melanocytes. One spindle and round population, which appeared benign, had a low cell proliferation index using MIB1 labelling using Ki 67 and was in keeping with melanocytoma. The other population, arranged in clusters and more epithelioid, had a higher MIB1 labelling using Ki 67 cell proliferation index and showed malignant features (fig 2B). These cells were similar to those present in the metastatic lesion of the left temporalis muscle (fig 3). Several nerve branches within the choroid showed perineural invasion by melanoma. At the angle deep to the ciliary body, a focus of vascular intrascleral invasion was also present. The optic disc and nerve as well as the meninges around the optic nerve were free of invasion.

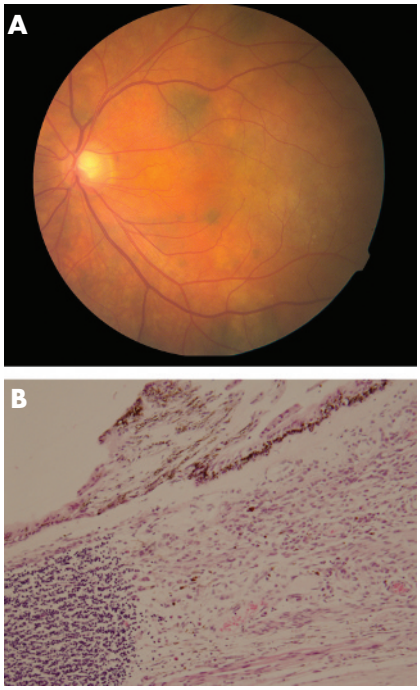

Figure 1 (A) Fundoscopy: note depigmented and pigmented nodules. (B) Ciliary body with underlying melanoma and lymphoid aggregate on left side (H&E ×200).

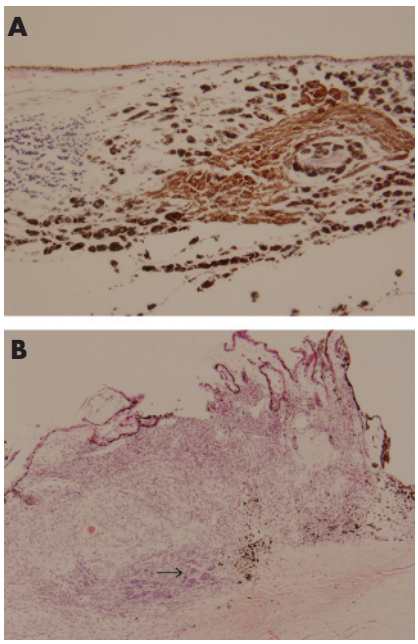

Figure 2 (A) Microscopy of the choroid showing pigmented satellite nodule with adjacent lymphoid aggregate (H&E ×100). (B) Full thickness of ciliary body showing partly pigmented malignant melanoma with epithelioid portion arrowed.

Discussion

This case illustrates an interesting histological presentation of a primary malignant melanoma of the ciliary body. Ciliary body melanomas comprise approximately 10% of uveal melanomas; they usually present with ocular symptoms and are amenable to local surgery. Of intraocular melanomas, iris melanomas have the best and ciliary body melanomas have the worst prognosis.[1] The median age at diagnosis ranges from 55 to 62 years.[1]

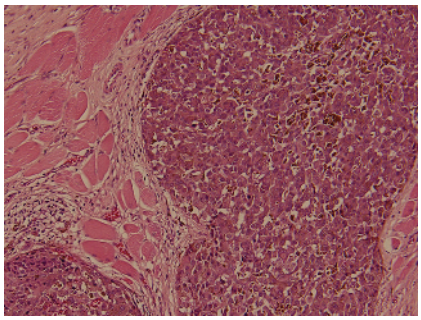

Figure 3 Epithelioid malignant melanoma (metastatic) in the temporalis muscle (H&E ×100).

In ocular melanoma, metastasis is usually haematogenous and in 95% of cases the initial site is the liver.[2 3] Liver metastasis is especially frequent with ciliary body melanomas.[4] In contrast to cutaneous melanomas, uveal melanomas do not have direct access to the lymphatics and thus cannot initially spread to regional lymph nodes.[5] In the present case, several nerve branches within the choroid showed perineural invasion by the tumour, and extraocular metastasis to the left temporalis muscle was found. This might have resulted from perineural invasion of the zygomaticotemporal branch of the maxillary nerve which exits the orbit through the zygomaticotemporal fossa and ends in the infratemporal fossa, the location of the metastasis.[6] Perineural spread of ocular melanoma has been well documented previously.[7 8] Vascular spread might also account for metastasis in this case.

Histologically, the multi-focal satellite spread in the choroid and the pronounced lymphocytic immune response around the tumour were highly unusual. Satellite metastasis and central regression is more frequently found in cutaneous melanomas.[9 10] A literature search has failed to identify previous reports of this type of ''satellite'' spread in ciliary body melanoma.

Malignant transformation of choroidal melanocytomas to malignant melanomas has been reported several times.[11–13] In all of these, the presenting features were due to the primary tumour, characterised by ocular symptoms and neurological deficits. In this case the metastatic lesion in the left temporalis muscle was the only clinical sign at presentation.

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References

1 **Singh AD**, Topham A. Incidence of uveal melanoma in the United States: 1973–1997. *Ophthalmology* 2003;**110**:956–61.
2 **Toivonen P**, Mäkitie T, Kujala E, *et al*. Microcirculation and tumor-infiltrating macrophages in choroidal and ciliary body melanoma and corresponding metastases. *Invest Ophthalmol Vis Sci* 2004;**45**(1):1–6.
3 **Yanoff M**, Fine B. Ocular melanocytic tumours. In: *Ocular pathology*, 5th edn. St Louis, USA: Mosby Inc, 2002:672–89.
4 **Frau E**, Lautier-Frau M, Labetoulle M, *et al*. Circumstances of choroid and ciliary body melanoma detection. *J Fr Ophtalmol* 2003;**26**:905–10.
5 **Lucas DR**, Greer CH. Tumours of the uveal tract. In: Chicago: *Greer's ocular pathology*, 4th edn. Blackwell Scientific Publications, 1989:113–30.
6 **Yanoff M**, Duker JS. Chapter 2: Orbital anatomy and imaging: clinical anatomy of the orbit. In: Dutton J, ed. *Ophthalmology*. St Louis, USA: Mosby International, 1999:2.1–2.7.
7 **Löffler K**, Tecklenborg H. Melanocytoma-like growth of a juxtapapillary malignant melanoma. *Retina* 1992;**12**:29–34.
8 **Wolter JR**. Orbital extension of choroidal melanoma: within a long posterior ciliary nerve. *Tr Am Ophth Soc* 1983;**81**:47–63.
9 **Leiter U**, Meier F, Schittek B, *et al*. The natural course of cutaneous melanoma. *J Surg Oncol* 2004;**86**:172–8.
10 **Meier F**, Will S, Ellwanger U, *et al*. Metastatic pathways and time courses in the orderly progression of cutaneous melanoma. *Br J Dermatol* 2002;**147**:62–70.
11 **Barker-Griffith AE**, McDonald PR, Green WR. Malignant melanoma arising in a choroidal magnacellular nevus (melanocytoma). *Can J Ophthalmol* 1976;**11**:140–6.
12 **Leidenix M**, Mamalis N, Goodart R, *et al*. Malignant transformation of a necrotic melanocytoma of the choroid in an amblyopic eye. *Ann Ophthalmol* 1994;**26**(2):42–6.
13 **Shetlar DJ**, Folberg R, Gass JD. Choroidal malignant melanoma associated with a melanocytoma. *Retina* 1999;**19**:346–9.

Sebaceous hyperplasia of the vulva: a clinicopathological case report with a review of the literature

Sebaceous hyperplasia consists of multiple asymptomatic small yellow papules with a central depression, occurring most commonly on the forehead and cheeks, but occasionally affecting the areola,[1] chest[2] or genital skin.[3] The lesions are sometimes mistaken clinically for basal-cell carcinoma. Sebaceous hyperplasia belongs to the group of epidermal tumours with sebaceous differentiation. Farina *et al*[4] suggested that hyperplasia in the sebaceous gland is analogous with trichofolliculoma. Although termed hyperplasia, they concluded that sebaceous hyperplasia is a benign neoplasm rather than hyperplasia as these lesions do not involute clinically. By contrast, the absence of significant enlargement of sebaceous lobules, and the sharp demarcation of the lesion distinguish it from hypertrophy. Thus, sebaceous hyperplasia is now considered as a hamartoma rather than a true neoplasm.[5 6]

A universally accepted definition of sebaceous hyperplasia is not yet available. However, Barnhill and Crowson[7] defined sebaceous hyperplasia by the presence of ⩾4 sebaceous lobules attached to the infundibulum of each pilosebaceous unit.

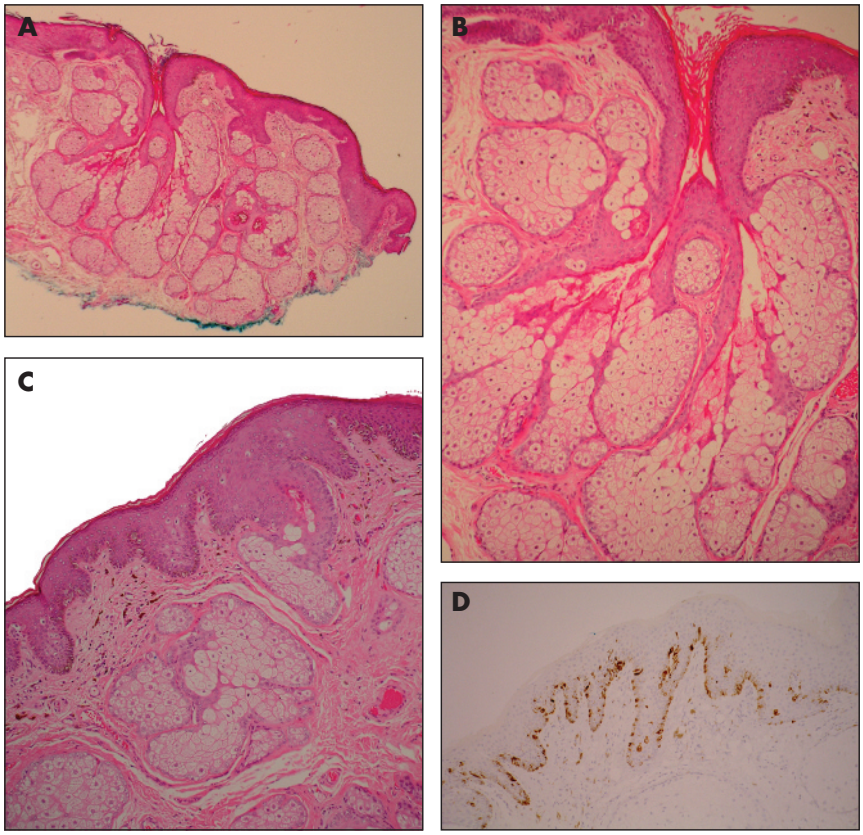

Figure 1 (A) Photomicrograph showing lobules of mature sebaceous glands attached to central hair follicle (original magnification ×70). (B) High-power view of sebaceous hyperplasia (original magnification ×250). (C) H&E section showing the overlying lentiginous hyperplasia of melanocytes, centrally within the lesion (original magnification ×250). (D) Melan A immunohistochemical stain demonstrating the melanocytic hyperplasia, (original magnification ×100).

Case report

A 31-year-old woman presented to the dermatology outpatients clinic with the appearance of two newly pigmented areas on the vulva, within the past 6 months. On examination, the lesions were darkly pigmented polypoidal papule (12 mm in diameter) and macule (5 mm in diameter) on the left labium minus and at the vestibule, respectively. They were non-tender without obvious textural change, and the surrounding skin was normal. Her gynaecological history was uncomplicated and she had had one normal pregnancy at the age of 20 years. Two biopsy specimens were taken. One was from the left vulva and the other one was from the mid-line region. Both lesions showed pigmented areas. The clinical diagnoses offered included melanocytic macule and benign tumour.

Methods

Sections were examined under routine light microscope using formalin-fixed, paraffin-wax-embedded tissue stained routinely with H&E. Immunohistochemical examination with a panel of stains, including antibodies to epithelial membrane antigen (1:50 dilution, Dako, Cambridgeshire, UK) and human milk fat globulin 1 (HMFG1; 1:25 dilution, Vector, Peterborough, UK) was performed on the paraffin wax blocks. Appropriate positive controls were run concurrently for all antibodies tested. Mouse non-immune sera were substituted for negative controls. Tissue submitted for ultrastructural examination was retrieved from the processed paraffin wax block. This involved dewaxing in xylene, hydrating through graded alcohols, osmicating, dehydrating through alcohol and propylene oxide, and infiltrating in TAAB EMIX epoxy resin (TAAB Laboratories, Aldermaston, Berkshire, UK). After polymerisation, sections were cut at 100 nm on a Reichert-Jung Ultracut E (Leica Microsystems, Buckinghamshire, UK) using a diamond knife; the sections were stained with alcoholic uranyl acetate and Reynold's lead citrate. Examination was on a Phillips 400T transmission electron microscope. Electron micrographs were taken using Kodak EM plate film (Agar Scientific Ltd, Stansted, UK).

Results

Microscopic examination of the first lesion showed an origin from sebaceous ducts and glands with a lobulated architecture. The lobules were composed of enlarged sebaceous glands (>4 around each pilosebaceous unit), which appeared to extend down into the dermis. The cells were predominantly mature sebocytes with a peripheral germinative layer, which was not prominent. Cytologically, the cells displayed a foamy, vesiculated cytoplasm and a central nucleolus with no atypical features (fig 1A,B). In addition, centrally, the