Hartzell was the first to publish (in 1904) a convincing photomicrograph of the condition known as desmoplastic trichilemmoma. This is a symmetrical, circumscribed lesion confined to the dermis. It classically consists of basaloid cells in columns and cords with the formation of infundibulo-cystic structures, and sometimes displays a biphasic appearance of squamous cells and basaloid cells in a sclerotic stroma. The latter mimics invasive carcinoma.[3] In fact, the main importance of desmoplastic trichilemmoma is its pathological appearance, which can mimic trichilemmal carcinoma, squamous carcinoma and morphoeic basal cell carcinoma. There is hence a major implication for treatment in the case of a pathological misdiagnosis. The present case was well-circumscribed and symmetrical with the typical biphasic appearance, had a basement membrane and lacked mitoses or any significant cytological atypia.

The histiogenesis of desmoplastic trichilemmoma is controversial but is thought to be the same as for the classical trichilemmoma. It is histomorphologically similar to lesions such as verrucae vulgaris and inverted follicular keratosis. This has influenced some to believe that the desmoplastic reaction represents an involuting verruca showing trichilemmomal differentiation.[4] Unfortunately there is no molecular or cytopathogenic evidence to support a human papilloma virus aetiology. Perhaps the stromal changes in desmoplastic trichilemmoma simply reflect the host response to superficial ulceration. As far as we are aware, this is the first report of desmoplastic trichilemmoma affecting the eyelid. The extent of its pseudocarcinomatous changes and general architecture may cause diagnostic confusion at both the macro- and microscopic levels. Tumour recurrence following excision has never been reported.[1] This has important implications for the patient since simple excision of this lesion effects cure. Recognition of this benign neoplasm as such will prevent misdiagnosis and unnecessary aggressive treatment.

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Sir,

An unusual chorioretinal dystrophy?

Chorioretinal atrophy is observed as a consequence of congenital, hereditary or acquired disorders which may be infective, inflammatory or degenerative in nature. Geographic location and patterns of atrophy are used to classify each type.[1] We report a case of chorioretinal atrophy with a striking symmetrical appearance not previously described.

Case report

A 68-year-old woman was incidentally noted by her optician to have bilateral chorioretinal scarring which was thought to represent inactive chorioretinitis. She had no significant past medical history other than hypertension, which was well controlled on atenolol 100 mg daily.

On examination, the unaided visual acuities were 6/6 bilaterally and her visual fields, tested by Goldmann perimetry, were full. No abnormality was noted in the anterior segments; vitreous syneresis was present but was unaccompanied by signs of an active uveitis. The striking abnormality was the presence of bilateral, symmetrical, discrete areas of chorioretinal atrophy (Fig. 1). These lay along the vascular arcades near the origin of the vortex veins. *Toxoplasma* serology was negative.

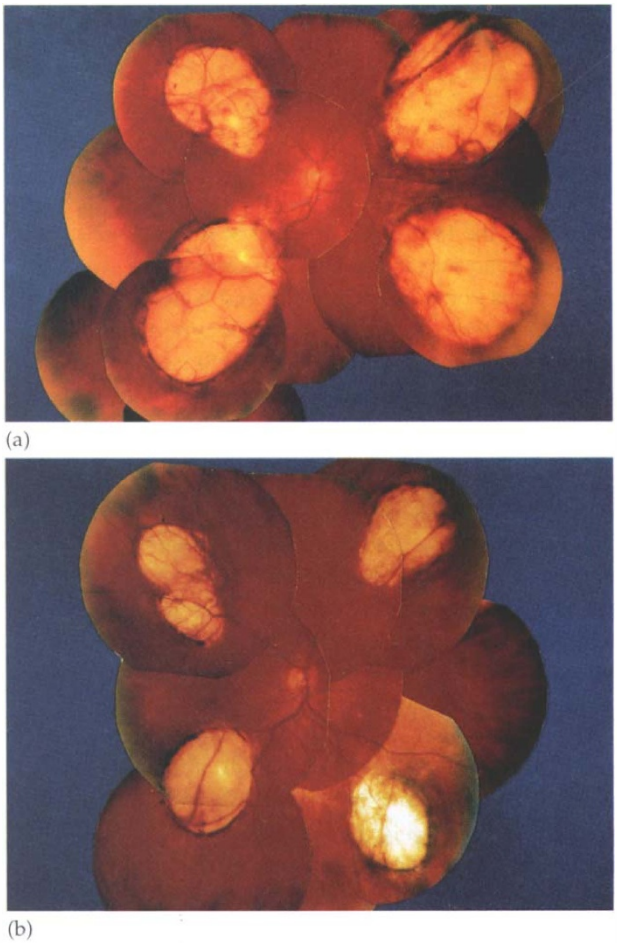

Fig. 1. *Symmetrical chorioretinal atrophy of the right (a) and left (b) fundi.*

The fundal appearances have remained unchanged since the patient was first seen in 1995. Both parents are deceased; fundal appearances of the patient's brother, sister, son and daughter were normal.

Comment

The chorioretinal atrophy observed could represent a congenital abnormality related to choroidal inflammation or an unusual chorioretinal dystrophy. The most common cause of well-defined chorioretinal scarring in the United Kingdom is inflammation caused by *Toxoplasma gondii*.[2] The absence of detectable (undiluted) serum antibodies suggests that ocular toxoplasmosis is highly improbable.[3] Well-defined atrophy such as this occurs in bifocal chorioretinal atrophy[4] and North Carolina macular dystrophy,[4] both of which are dominantly inherited, although the site is quite different from that described in this report. Whilst it is tempting to assume that these abnormalities may represent an unusual chorioretinal dystrophy no other living family members have been similarly affected. We are not aware of any reports documenting a similar phenotype and therefore are unable to establish the diagnosis.

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Sir,

Mitochondrial DNA disease masquerading as age-related macular degeneration

Pigmentary changes in the retinal periphery are well-recognised features of mitochondrial disease.[1] We report here a patient with the MELAS (mitochondrial encephalomyopathy with lactic acidosis and stroke-like episodes) syndrome[2] who presented with a pigmentary retinopathy characterised by predominant posterior pole and macular involvement.

Case report

The patient was first noted to have retinal pigmentary abnormalities aged 44 years during a routine visit to her optician. She was visually asymptomatic and had no parental history of any ophthalmic disease. Four years previously she had developed mild gradual-onset bilateral sensorineural deafness in the absence of other neurological disease. Past medical history was otherwise unremarkable and she was systemically well.

Visual acuity was 6/9 in both eyes. Lid position and function were normal and extraocular movements full. No pathology was detected on anterior segment examination. Indirect ophthalmoscopy revealed symmetrical irregular patches of retinal pigment epithelial atrophy and pigment aggregation predominantly involving the posterior pole and macular region (Fig. 1). This was confirmed on fluorescein angiography (Fig. 2). Both optic discs were pink and healthy. Electrophysiological studies, including flash electroretinograms and visual evoked potentials, were all within normal limits. The clinical picture was thought to be consistent with geographic age-related macular degeneration. In view of her relatively young age, periodic review was arranged. During the next 7 years there was no deterioration in her visual acuity or progression of her macular changes.

Seven years after her initial presentation the patient had a generalised seizure. Preliminary investigations revealed elevated fasting lactate levels and diabetes mellitus. A CT showed multiple occipito-parietal infarctions and calcification of her basal ganglia and posterior thalami. This clustering of clinical features and biochemical abnormalities suggested a diagnosis of mitochondrial cytopathy. A muscle biopsy was performed showing the presence of 2% ragged-red fibres. Subsequent molecular genetic studies confirmed the

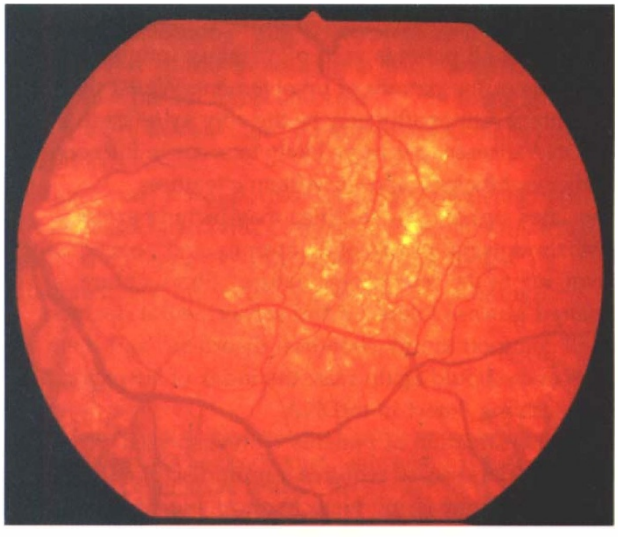

Fig. 1. *Fundal appearance at initial presentation.*