

Sir,
Cutaneous vitiligo associated with choroidal hypopigmentation

We observed two vitiligo patients with extensive ocular involvement. In contrast to previous observations, our vitiligo patients show only hypopigmentation of the choroid, while the RPE cells are normal. Case 1 is a 59-year-old Chinese man who had vitiligo over 30 years and was referred to us because of visual problems. Except for vitiligo, he had no history of general or ocular disease. The ocular examination was normal except for presbyopia and an abnormal fundus appearance. He had no signs of past or present uveitis. Both fundi showed wedge-shaped areas of choroidal hypopigmentation (Figure 1). Fluorescein angiography showed no abnormalities (Figure 2). Additional Humphrey 30-2 and 30/60-2 visual fields and electrodiagnostic tests showed no evidence of retinal dysfunction. Case 2 is a 48-year-old Chinese woman with vitiligo who was referred to us because of an abnormal fundus appearance. She had no previous ocular disorders or complaints. The ocular examination was completely normal with the exception of the fundus. Her fundi showed large areas of choroidal hypopigmentation very similar to case 1. Humphrey 30-2 visual fields and electrodiagnostic tests were normal. Large studies suggest that choroidal pigmentary changes occurring in the presence of vitiligo can be ascribed to uveitis in many cases, although in some no evidence of inflammation could be found.¹⁻² Only a small number of cases have been reported with widespread hypopigmentation of both choroid and retinal pigment epithelium (RPE).¹⁻³ Our two patients with vitiligo had extensive choroidal hypopigmentation without signs or a previous history of ocular inflammation. They both had normal visual function, and additional fluorescein angiogram and electrodiagnostic tests in case 1 were normal. These findings suggest that the lack of pigment was confined to the choroid and that the RPE was not involved. The hypopigmentation of the choroid may be congenital and the coexistence of cutaneous vitiligo in our patients may have occurred by chance. Alternatively, this observation may be explained on the basis of the embryological origin of the affected cells. Both choroidal and skin melanocytes are derived from neural crest cells, differing in this respect from RPE cells that are derived from neuroectoderm.⁴ A disease related to cells of this origin could therefore affect dermal and choroidal melanocytes without involvement with the RPE cells. The pathogenesis of vitiligo is unclear but neural, autoimmune, and autocytotoxic mechanisms have been implicated. In favour of the first is the observation that the skin lesions sometimes follow the borders of



Figure 1 Case 1: Colour fundus photograph, right eye. Note the cone-shaped area of choroidal hypopigmentation in the area inferior to the optic disc. A similar area was seen superotemporal in the left eye.

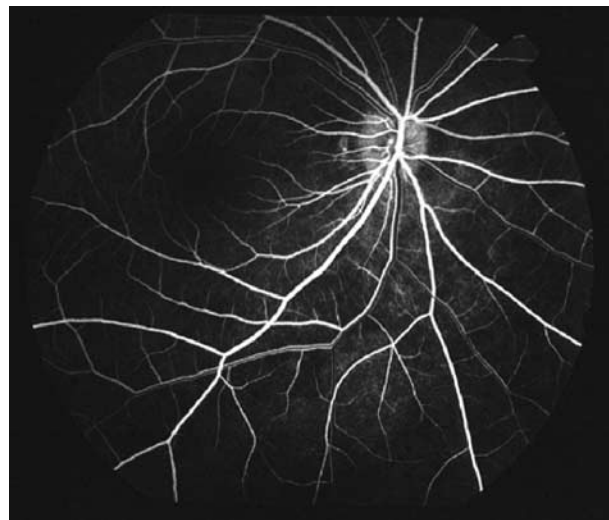


Figure 2 Case 1: Fluorescein angiogram of the same area as in Figure 1. The areas corresponding to choroidal hypopigmentation show no abnormalities.

dermatomes, and abnormalities of peripheral nerves have been described in areas of vitiligo.⁵ Thus, the innervation of the choroid may play a role in the pathogenesis of loss of choroidal pigment. Extensive choroidal involvement in vitiligo patients has rarely been reported, and is clearly uncommon.² However, it may not be detected since it may not always be evident clinically and it gives rise to no functional deficit. Owing to their Asian origin, the normal dark choroidal pigmentation could easily be distinguished from hypopigmented choroid.

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JR Vingerling^{1,2} S Owens¹, WI van der Meijden²,
CB Hoyng³ and AC Bird¹

¹Moorfields Eye Hospital, City Road
London EC1V 2PD, UK

²Erasmus Medical Center, Rotterdam
The Netherlands

³University Medical Center, Nijmegen
The Netherlands

Correspondence: JR Vingerling
Tel: + 31 10 4633691
Fax: + 31 10 4635105
E-mail: j.vingerling@erasmusmc.nl

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Sir,
An unusual case of tuberculous dacryocystitis

Dacryocystitis is usually a result of primary acquired nasolacrimal duct obstruction^{1,2} where chronic inflammation results in fibrosis, stenosis, and ultimately complete obstruction of the nasolacrimal duct. The inflamed lacrimal mucosa has increased numbers of mucin producing goblet cells and together with tear stagnation there is bacterial overgrowth and subsequent dacryocystitis.

We describe an unusual case of tuberculous (TB) dacryocystitis, a rare secondary cause of nasolacrimal duct obstruction,^{3,4} diagnosed with the aid of out-patient nasal endoscopy.

Case report

A 36-year-old homeless male Somali refugee had a 3-month history of chronic dacryocystitis with epiphora. The swelling burst and discharged spontaneously though a fistula. Nasal endoscopy performed in the Lacrimal Clinic by the ophthalmologist showed inflamed right nasal mucosa and he was referred to the otolaryngologists for their specialist opinion. He had an external dacryocystorhinostomy (Ext-DCR) with insertion of tubes, which were removed after 3 months, at which time the fistula had spontaneously healed but there was some residual epiphora. After 11 months, he reattended with recurrent chronic dacryocystitis and a second Ext-DCR was performed. He had failed to attend the otolaryngology clinic, as being homeless he did not receive the appropriate correspondence for this and he had also failed to attend the Lacrimal Clinic for removal of tubes as planned.

At 5 months after the second DCR, out-patient nasal endoscopy showed persistently inflamed and thickened nasal mucosa (Figure 1). The ophthalmologist performed an out-patient endoscopic biopsy of his nasal mucosa under topical anaesthesia. Histopathological analysis was equivocal for sarcoidosis and Wegener's granulomatosis. Ziehl–Neelsen stain for acid-fast bacilli was negative.

Following several months of repeated nonattendances, he returned with acute dacryocystitis with associated preseptal cellulitis. Following a course of oral antibiotics, an extensive exploratory (third) Ext-DCR was performed. Abnormal gelatinous tapioca-like tissue filling the ostium was curetted out and sent for histopathological analysis, alongside specimens of the lacrimal sac. This showed multinucleate Langhan giant cells and chronic caseating

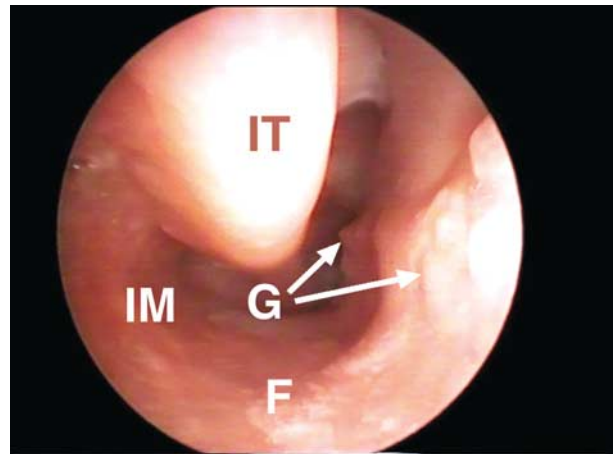


Figure 1 Endonasal photograph of right nasal space showing chronic granulomatous (lumpy) inflammation of the nasal septum. IM = inferior meatus, IT = inferior turbinate, F = floor, G = granuloma.