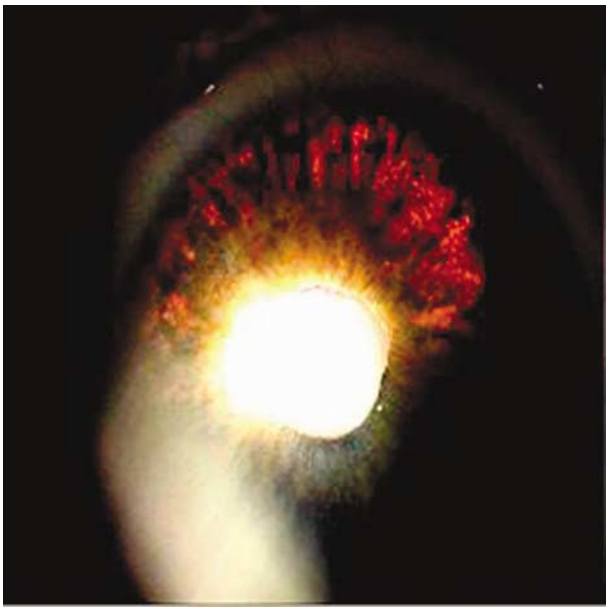

Figure 2 Superior iris transillumination defect.

depends on interference from other melanin-containing structures.[2,3] Hair follicles lie about 3 mm below the skin surface. However, thermal damage from the alexandrite laser can occur at several millimetres depending on user settings and skin type.

Before photo-epilation, this patient had her eyes closed and covered with damp cotton pads. Owing to the Bell's phenomenon, elevation of the globe with lid closure would have resulted in proximity of the superior iris to the area being treated. As the iris is a melanin-containing structure, exposure to the penetrating laser beam would have resulted in absorption of energy and subsequent damage with inflammation and pigment dispersion. The presence of superior iris atrophy is consistent with this mechanism.

At follow-up after 1 year, this patient was on no treatment to the affected eye and had normal VA and IOP. Long-term ocular complications from laser damage in this case are unknown. Recent guidelines have warned against the high potential for thermal damage to the iris and retina in patients who have photo-epilation close to the eye.[4] Strict safety measures, such as metal lenses, are required in these circumstances.

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Sir,

Evidence of Waite–Beetham lines in the corneas of diabetic patients as detected by *in vivo* confocal microscopy

Corneas of diabetic patients exhibit remarkable abnormalities of nerves, basement membranes, and cellular layers.[1] Confocal microscopy has become a popular technique to observe the structure of cornea *in vivo* in several disease states afflicting the cornea including diabetes mellitus.[2,3] Among the less common observations in the corneas of diabetic patients are the faint vertical lines at the level of Descemet's membrane and endothelium, initially described by Waite and Beetham[4] and Henkind and Wise.[5] The purpose of this paper was to report the characteristics and incidence of Waite–Beetham lines in the corneas of diabetic patients.

Case report

The study was carried out with approval from the Institutional Review Board. Clinical evaluation and data collection of all patients was performed by a single ophthalmologist (MI). *In vivo* confocal microscopy were performed using Confoscan 3.0 (Vigonza, Italy) attached to an immersion lens (Achroplan ×40/0.75W, Zeiss, Germany). The presence of vertical lines at the level of Descemet's membrane and endothelial cell counts were evaluated by a single observer (MCM) who was masked

to the patients' identities and disease status throughout the evaluation.

Thirty-five corneas of 35 type II diabetic patients (17 men and 18 women) with a mean age of 54.5±8.5 years and 24 corneas of 24 age-matched nondiabetic subjects (11 men and 13 women) with a mean age of 58.4±10.0 years were included in the analysis. Slit-lamp evaluation revealed very thin vertical lines at the Descemet's membrane in 13 (37.1%) diabetic patients and two (8.3%) normal subjects (*t*-test, $P=0.005$). Faint vertical lines at the level of Descemet's membrane (Figure 1a) were detected with the *in vivo* confocal microscope in 23 of 35 corneas (65.7%) compared to four of 24 corneas of control subjects (16.7%) (*t*-test, $P<0.001$). The lines were mostly vertical with occasional oblique or horizontal orientations. They were most prominent at the level of Descemet's membrane and the neighbouring posterior stroma (Figure 1b). Waite–Beetham lines observed clinically in 13 diabetic and two control subjects were also detected with the confocal microscope.

The mean age of diabetic patients who had the Waite–Beetham lines (59.5±9.7 years) was not significantly different from the mean age of diabetic patients who did not have them (56.3±10.8 years) (Mann–Whitney *U*-test, $P=0.404$). In addition, there was also no statistically significant difference between the mean ages of 23 diabetic patients (59.5±9.7 years) and four control (62.0±8.6 years) subjects who demonstrated evidence of the Waite–Beetham lines (Mann–Whitney *U*-test, $P=0.733$).

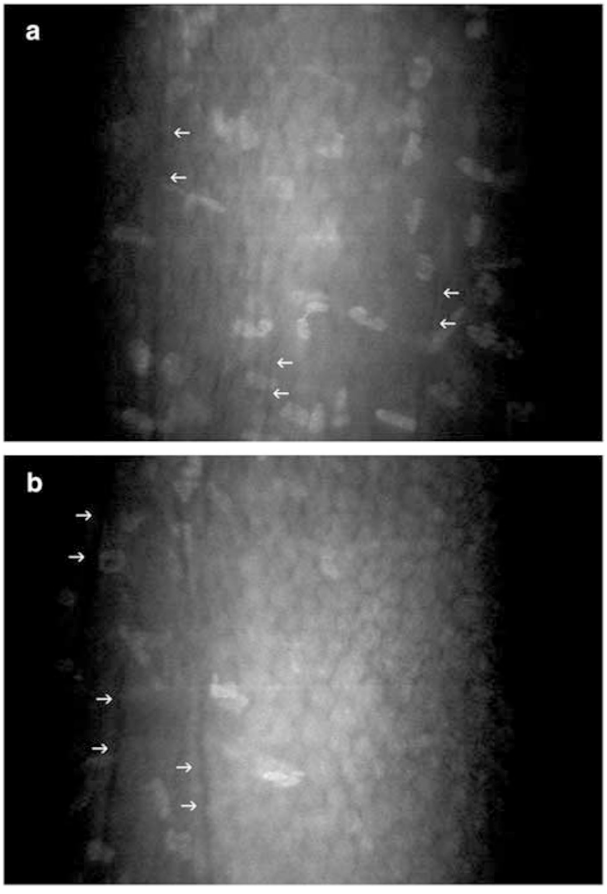

Figure 1 Faint (a) and prominent (b) vertical lines at the level of Descemet's membrane and the neighbouring posterior stroma of diabetic patients.

The mean endothelium cell count of diabetic patients (2678.0±507.6 cells/mm^2) was not significantly different from that of normal subjects (2631.8±417.0 cells/mm^2) (*t*-test, $P=0.714$). In addition, the mean endothelium cell count of diabetic patients who had Waite–Beetham lines (2713.1±542.0 cells/mm^2) appeared similar to that of diabetic patients who did not have them (2611.0±449.0 cells/mm^2) (Mann–Whitney U-test, $P=0.322$).

Comment

Folds at the level of endothelium and Descemet's membrane may be observed in the setting of contact lens-induced hypoxia in association with other signs of corneal hypoxia such as epithelial microcysts and oedema.[6] Although they may be regarded as a sign of corneal hypoxia, a recent study by O'Donnell *et al*[6] have observed such lines in the corneas of four out of 40 diabetic contact lens users with a mean age of 34.0±13.0 years and none of the age-matched nondiabetic contact lens users and have suggested that these folds are not indicative of hypoxia in all cases. Our results additionally suggest that these folds are not associated with loss of endothelial cells. However, ageing may be a risk factor in the development of the Waite–Beetham lines as they were detected in 16.7% of our normal subjects (mean age = 57.4 years) as well. In conclusion, the results of our study suggest that Waite–Beetham lines may be more common in the corneas of diabetic patients than can be clinically appreciated and may represent accelerated tissue ageing. The inciting stimulus for the development of Waite–Beetham lines, be it either metabolic or mechanical, continues to remain elusive.

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Sir,

Wound integrity and the conjunctiva in prevention of endophthalmitis following sutureless 25-gauge vitrectomy

I wish to thank Drs Taylor and Aylward for describing their case of presumed bacterial endophthalmitis following 25-gauge vitrectomy.[1] Discussion of complications or untoward outcomes ultimately leads to improved patient care.

It would be interesting to know whether scleral depression for examination of the periphery was performed during this case. When performed near the site of a 25-gauge cannula, this technique necessarily causes the cannula to be redirected anteriorly. If the cannula is plugged, this might not disrupt the sclerotomy, but if the cannula contains an instrument in active use such as the vitreous cutter, the manipulation of the instrument could be at odds with the anterior misdirection of the cannula. The consequence could be enlargement of the sclerotomy wound or at least distortion of the normal wound architechture. Similarly, scleral depression can tear the conjunctiva by pulling it posteriorly while it remains anchored at the 25-gauge cannula. From any of these scenarios, one could envision an increased risk of subclinical wound leak.

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Sir,

Reply to Dr Stewart: prevention of endophthalmitis following sutureless 25-gauge vitrectomy

I thank Dr Stewart for his interest in this case. We only performed scleral indentation at the end of the case to check the ora serrata near the entry sites, and during this procedure the cannula was plugged. The procedure was completely routine in every aspect, so we do not believe there were any case-specific risk factors for endophthalmitis. We remain concerned as to whether there is an increased risk of infection associated with the use of sutureless vitrectomy systems, and await further data from case reports and future trials.

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