LETTERS TO THE EDITOR

Late dehiscence of healed corneal scars

EDITOR,—The stroma comprises about 90% of the total corneal thickness1 and is responsible for most of the corneal tensile strength. Presumably because of its avascularity, healing of corneal wounds is slower than in other connective tissues. Continued histopathological changes in human corneal laceration wounds have been observed years after injury,2 despite meticulous original suture closure.3 The wounds appear to undergo perpetual, dynamic remoulding, a fine balance between constructive and destructive processes. We report three cases of late, full-thickness corneal wound dehiscence occurring spontaneously or after minor blunt trauma 17–56 years after the original injury.

CASE REPORTS

Case 1

A 61 year old man was referred for spontaneous corneal perforation right eye. At age 5, he had sustained a full thickness corneal laceration in this eye which was repaired with sutures. The patient did well with an uncorrected visual acuity of 20/50 right eye until the time of his corneal perforation 56 years later whereupon he noted the sudden onset of eye pain and loss of vision in this eye upon awakening 2 days before referral. He denied any recent eye trauma or antecedent vigorous rubbing of the eye. He was in excellent health with no previous history of corneal melting disorders. At the time of his first visit to us, the best corrected visual acuities were light perception right eye and 20/20 left eye. There was an inferonasal, 3 mm linear, vertically oriented corneal scar in the right eye not involving the limbus. The iris was incarcerated and externalised along an inferior 1.0 mm long dehiscence in the corneal scar, dragging the entire pupil into the perforation. The Seidel test showed a slow, spontaneous leakage of aqueous humour from the dehiscence. An aqueous humour from the dehiscence further deprived the cornea of oxygen. The patient underwent elective extraction of the cataract in this eye. Subsequently, the best corrected visual acuity was 20/40 with a hard contact lens and spectacle overrefraction. Seventeen years after the injury, the corneal scar dehisced spontaneously in the absence of recent trauma. A peripheral tectonic penetrating keratoplasty was performed, completely excising the dehisced corneal scar. The patient later underwent a central, 8.0 mm, optical penetrating keratoplasty, combined with implantation of a posterior chamber intraocular lens by sulcus fixation. This resulted in a best corrected visual acuity of 20/20 in this eye.

COMMENT

These three cases illustrate the innate structural weakness of healed corneal scars. Two of the scars dehisced without any mechanical provocation. Although corneal scars become very slow to regain more than 50–70% of their original tensile strength,4 unsutured wounds, such as radial keratotomy incisions, are particularly weak.5 Even sutured full thickness wounds may not regain more than 50% of their original tensile strength.6 In penetrating keratoplasty, rupture after blunt trauma may occur many years after surgery and always occurs at the graft-host junction, the weakest point in the cornea.7 In full thickness corneal wounds, the discontinuity in Descemet’s membrane further deprives the cornea of another important source of structural strength. Ancillary factors which may further reduce wound integrity include diabetes mellitus, poor suturing technique, incarceration of uvea or vitreous, and entrapment of epithelium.

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Transpupillary thermotherapy of choroidal melanoma with or without brachytherapy: a dilemma

EDITOR,—Transpupillary thermotherapy (TTT) is a new approach to the treatment of choroidal melanoma with heat. The early treatment results seem favourable with regard to local tumour control and retained visual acuity; no early metastasis was found.1 The reported rate of complications is low.2 The heat treatment is performed with a temperature calculated at 65°C, induced by an infrared diode laser (810 nm) with a large beam diameter (3 mm) and a long exposure time of 1 minute per application. These factors promote a deep penetration of heat into the choroidal melanoma. The result is tumour necrosis up to 3.9 mm deep and occlusion of blood vessels in the treated area as shown on histopathology.3 Depending on the pigmentation of the tumour, the energy is raised or lowered according to the effect that is desired. The tumour is treated by overlapping applications extending beyond the margin of the tumour. The exposure time should not be shortened to less than 1 minute, as this takes 40 seconds to reach a temperature at subcoagulation level.

TTT is performed as an outpatient procedure and can be easily repeated. Treatment results can be evaluated in several ways. The outcome is favourable when the choroidal melanoma develops into a scar on ophthalmoscopic examination, substantial regression of the tumour thickness is seen on ultrasonography together with an increased reflectivity on the diagnostic A-scan, and hypofluorescence is visible in the treated area on the early and late phases of the fluorescein angiogram. TTT is not indicated in melanoma patients with severe media opacities, in patients with insufficient dilatation of the pupil, or in elevated, peripherally located melanomas.

Simultaneous use of TTT with brachytherapy in the management of melanomas is called “sandwich therapy”. This combined treatment has the advantage that TTT is maximal at the top of the tumour and brachytherapy efficiently treats the base of the tumour.4,5 Combining both treatments has several advantages. It enabled us to treat patients with tumours thicker than 5 mm, generally the maximum height for ruthenium-106 brachytherapy.6 Insufficient melanoma regression and/or recurrences after a combined treatment might be retreated with TTT alone. The combination might also lead to a decreased radiation dose of the isotope used in brachytherapy.

In principle, TTT can also be applied as the sole therapy. A satisfactory local tumour control is reported in the early years after TTT as sole therapy for small choroidal melanomas.7 TTT as sole therapy seems especially attractive in treating small choroidal melanomas in and around the posterior pole. Visual outcome may be better after TTT than after brachytherapy because the laser beam, but not the radiation beam delivered by the radioactive plaque, can be focused.8 As the macular capillaries are very radiosensitive to brachytherapy, this may finally result in radiation damage to the macula.
maculopathy with a considerable loss of central vision. However, after extramacular applications of sole TTT, macular complications such as transient cystoid macular oedema may be also seen. A dense scotoma is generally produced by TTT at the time of application, whereas a scotoma after brachytherapy usually does not develop for up to a year or several years. If heat treatment is performed as sole therapy, effective treatment of the sciera over the tumour is as important as treatment of the tumour itself because intra- and episcleral tumour invasion is present in, respectively, 55.7% and 8.2% of patients with medium and large tumours. In a group of 21 patients with enucleated small melanomas, 17 showed scleral ingrowth on histopathological examination. Intrasceral melanoma cells surviving treatment might be a focus for tumour recurrence, which is associated with a two to threefold higher risk of metastasis. This is evident from the results after photocoagulation treatment of choroidal melanomas showing a high rate of tumour regrowth. This might be attributed to the low density of photocoagulation penetration, insufficient to destroy the intrasceral melanoma cells. In TTT as sole therapy, the question arises as to whether the effect of the heat induced by TTT in the sclera, where the choroidal melanoma is identical to the effect of the heat to the possible melanoma cells in the sclera. In one eye, intentionally enucleated after experimental TTT, total tumour necrosis up to the sclera was associated with histopathological examination. Despite heat damage to the inner layers of the sclera which was evident from scleral oedema and necrosis of scleroocytes, a cluster of tumour cells with a viable aspect was located near the inner border of the sclera. A recent clinicopathological report on two cases of recurrent juxtapapillary choroidal melanoma after TTT revealed tumour cells appearing histopathologically intact and presumably viable. One patient was insufficiently treated with TTT because of cataract, another patient was treated with TTT and brachytherapy. Interpreting these data, we have to keep in mind that a pathologist is often unable to guess viability of melanoma cells in an enucleated eye after any treatment, especially in an undertreated eye. Unfortunately, no sensitive clinical technique exists to evaluate the effect of TTT on intra- and episcleral tumour cells, their destruction, and the early development of extrascleral recurrences. To reduce the risk of insufficient treatment of the sciera and because episcleral tumour recurrences rarely develop as a late complication of brachytherapy, one may consider combining TTT with brachytherapy (the sandwich therapy). TTT remains an investigative procedure until long-term results are available. In the meantime we have to choose between TTT as sole or combined treatment for patients with choroidal melanoma.

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Asymmetric diabetic retinopathy
associated with Fuchs' heterochromic cyclitis

Enron,—Diabetic retinopathy (DR), with its complications, is the leading cause of blindness among the working population in developed countries. Asymmetric diabetic retinopathy (DR) has been defined as proliferative disease in one eye and no or no retinopathy in the fellow eye, persisting for at least 2 years. Previous reports have described systemic and local factors associated with the development of asymmetric disease. These include unilateral carotid artery stenosis, choroidal scarring, complete posterior vitreous detachment, amblyopia, unilateral elevated intraocular pressure, optic atrophy, retinal pigment epithelial atrophy, myopia ≥5D, anisometropia >1D, concurrent retinal vascular disease, cataract extraction, vitreous loss, trauma, radiation, tumour, and unilateral recurrent panuvenitis. We observed a patient with proliferative DR in the right eye and no proliferative changes in the left eye which had Fuchs' heterochromic cyclitis (the sandwich therapy), one may consider combining TTT with brachytherapy (the sandwich therapy). TTT remains an investigative procedure until long-term results are available. In the meantime we have to choose between TTT as sole or combined treatment for patients with choroidal melanoma.

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Figure 1 June 1996: fundus fluorescein angiography. (A) Right eye, late venous phase. There is ischaemia of the superotemporal retina of the right eye. Superior panretinal laser scars and disc leakage from residual new vessels can be seen. (B) Left eye, late venous phase. The left eye also shows some areas of capillary non-perfusion. There is hyperfluorescence at the superotemporal aspect of the left disc but no characteristic leakage features of proliferative diabetic retinopathy. Note the small window defect inferonasal to the left fovea from an area of retinal pigment epithelial atrophy.

with clinically significant macular oedema at the baseline examination, or new onset proliferative diabetic retinopathy. Several authors have reported that the anterior lens capsule is more stable when in contact with an acrylic IOL (AcrySof) compared with PMMA and silicone lenses. To our knowledge this is the first case report of anterior capsular contraction following implantation of an AcrySof IOL.

**CASE REPORT**

A 77 year old white woman underwent uncomplicated phacoemulsification with insertion of a foldable acrylic (AcrySof, Alcon Laboratories, Hemel Hempstead) IOL (23.0 dioptre, 6.0 mm optical diameter, model number MA60BM) through a superiorly sited scleral tunnel (June 1998). The capsulohyaline diaphragm was 5.0 mm thick. No attempt was made to remove anterior lens epithelial cells (LECs) during irrigation aspiration of lens cortex. Ophthalmic history was negative for uveitis, pseudoxefoliation, and myopia. Postoperatively she made a good recovery, achieving an unaided visual acuity of 6/9. Three months later she presented again because of reduced vision (6/24 corrected). Dilated examination revealed fibrosis as well as marked phimosis of the anterior capsule opening (Fig 1A). The contraction was symmetrical and no decentration or tilting of the lens was noted. There was no posterior capsular capsular opacification of note. Nd:YAG radial anterior capsulotomy (193.5 mJ TE) was carried out (Fig 1B). She regained a visual acuity of 6/9 when reviewed 1 month later (Fig 1C).

**COMMENT**

The advent of CCC has led to the presentation of specific complications of this technique. These are capsule contraction syndrome, capsular bag distension, and LEC migration and proliferation onto the posterior capsule. It is now well recognised that capsule contraction syndrome is related to the gradual proliferation of residual anterior LECs that leads to fibrous metaplasia and eventual reduction of the capsular opening. Electron microscopy studies have revealed these to be cells resembling fibroblasts surrounded by a dense collagen matrix.

Vision can be impaired not only because of opacification of the media but also because of tilting, decentration, and buckling (foldable only) of the IOL. In severe cases the zonular traction may lead to IOL dislocation and retinal detachment. Anterior capsular shrinkage occurs more rapidly in the first 6 weeks postoperatively but continues slowly thereafter.

Sickenberg et al have claimed that the stronger centrifugal force of the AcrySof IOL's

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Figure 2 April 1998: fundus fluorescein angiogram. (A) Right eye, late phase. After multiple sessions of panretinal laser the right eye shows regression of disc new vessels with no residual leakage. (B) Left eye, mid venous phase. Following cataract surgery the hyperfluorescence shows regression of disc new vessels with no residual leakage.
Central serous retinopathy complicated by massive bilateral subretinal haemorrhage

Editor,—Central serous retinopathy (CSR) is a common cause of visual disturbance in the younger age group. Spontaneous visual recovery occurs in the majority of patients. A minority of patients, however, suffer permanent visual loss commonly caused by chronic retinal pigment epithelial changes. We report a devastating complication of untreated CSR, with bilateral massive subretinal and vitreous haemorrhages.

A 43 year old Asian man presented with a 3 week history of blurred central vision and metamorphopsia affecting his left eye. Ocular examination revealed best corrected visual acuity of 6/9 in both eyes. Funduscopy revealed a neurosensory retinal detachment overlying the left fovea, with bilateral multiple retinal pigment epithelial changes. Fundus fluorescein angiography (FFA) and indocyanine green angiography (ICGA) confirmed the diagnosis of bilateral CSR (Figs 1 and 2).

The neurosensory retinal detachment in the left eye resolved spontaneously. Seven months after his initial presentation, he developed sudden loss of vision in his left eye to counting fingers at 1 metre. Ocular examination revealed a large macular subretinal haemorrhage, which progressed to a dense vitreous haemorrhage after a week. Ultrasoundography revealed no evidence of a retinal detachment. The vitreous haemorrhage subsequently cleared spontaneously leaving an atrophic macular scar.

Figure 1 (A) Slit lamp photograph showing anterior capsule phimosis and fibrosis. (B) Appearance of anterior capsule immediately after Nd:YAG radial anterior capsulotomy. (C) Appearance of anterior capsule 3 weeks after laser.

Figure 2 (A) Fluorescein angiography of the right macula reveals multiple areas of leakage. (B) ICGA reveals more extensive disease with diffuse hyperfluorescence representing multiple areas of choroidal hyperpermeability. (C) Late phase ICGA demonstrates severe retinal pigment epithelial detachments with characteristic central hyperfluorescence and hyperfluorescent staining of the surrounding ring. (arrows).

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Figure 1 Fluorescein angiography of the left eye reveals pinpoint hyperfluorescence superior to the fovea (A) which shows progressive leakage in the later phase (arrows) (B), confirming the diagnosis of central serous retinopathy.

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COMMENT
Massive subretinal macular haemorrhage can occur secondary to a number of causes such as choroidal neovascularisation (CNV), retinal artery macroaneurysm, idiopathic polypoidal choroidal vasculopathy, blood dyscrasia, or trauma. Histopathological analysis of patients with age related CNV complicated by massive subretinal haemorrhage may be associated with rupture of a large choroidal blood vessel.1 CNV is known to occur infrequently in patients with CSR complicated by laser photoagulation.2 In only two previous cases has CNV developed spontaneously in patients with CSR.1 Massive subretinal haemorrhage, however, was not the feature in these two reported cases. In our case, the use of ICGA had helped to understand the mechanical events (Figs 2 and 3) but the underlying aetiology remains unclear.

The pathogenesis of CSR has been disputed. Recent studies with ICG suggest focal choroidal hyperpermeability as the possible initial event, leading to the formation of serous retinal pigment epithelial detachment. Excessive fluid accumulation then leads to pressure on the retinal pigment epithelium, resulting in either mechanical disruption or retinal pigment epithelial decompensation.3 The chronic secondary retinal pigment epithelial changes, if extended, could predispose to the development of CNV. In our patient, the sudden onset of haemorrhage in both eyes may in part be explained by the presence of disorganised and dysfunctional choroidal blood vessels. The latter leads to an initial increase in choroidal hyperpermeability (hence the CSR) and later, the tendency to rupture suddenly resulting in massive haemorrhage (as illustrated by our case).

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CASE REPORTS
The family tree is shown in Figure 1A. Case I-1 is a 52 year old man with visual impairment and nystagmus. He had cataract from an early age (the detail was unknown). His vision was right eye finger counting at 20 cm and left eye 0.01; eye examination revealed bilateral partial aniridia, dense cataract, corneal pannus, and foveal hypoplasia (Fig 2A, B). His cataracts were removed and posterior chamber intraocular lenses were implanted at age 52. One son, II-1, is 16 years old with nystagmus, visual acuities of right eye 0.3 and left eye 0.2, bilateral corneal pannus, iritis, and mild foveal hypoplasia; his lenses were clear (Fig 2 C, D). Another son, II-2, is 14 years old with nystagmus, visual acuities of right eye 0.2 and left eye 0.2, bilateral iris thinning, corneal pannus, and mild foveal hypoplasia; his lenses were clear (Fig 2 E, F).

In our case, the PAX6 mutation is predicted to result in amino acid substitutions from the 339th position and a truncation at the 344th position, resulting in a truncated protein product that ultimately excludes part of the PST domain. Truncated proteins generally tend to be degraded, thus haploinsufficiency of PAX6 was believed to cause aniridia phenotype. However, biochemical assay recently indicated that truncation mutations in the PST transactivation region of PAX6 result in dominant negative mutants.8 A truncated protein of nearly normal size may function for a short time.

The affected individuals in this family had a wide variation of phenotypes, including thinning and the absence of the iris, various features of cataract, and foveal hypoplasia. Hittner et al19 11 described suspected familial aniridia cases in which expressivity was variable as was seen in our cases. Probably many modifiers affect a variety of phenotypic expression in a pedigree with the same PAX6 mutation.

The human PAX6 gene is 422 amino acids long and has paired box and homeobox DNA binding domains. These are separated by a 78 amino acid linker segment and followed by a 152 amino acid C-terminal region rich in proline, serine, and threonine. The C-terminal region is less conserved and contains at least 14 exons for the PAX6 gene that are amplified by polymerase chain reaction (PCR) and subjected to SSCP analyses.

A normal pattern for exon 11 was identified indicating a heterozygous mutation in all affected family members. Genomic DNA representing 14 exons for the PAX6 gene was amplified by polymerase chain reaction (PCR) and subjected to SSCP analyses.

An abnormal pattern was detected in the deletion of a single nucleotide at the 1434th position (in this study, the nucleotide and amino acid were based on the sequence of GenBank Accession No M93650) (Fig 1B). No other changes in nucleotide sequence were detected.

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There was little difference in the phenotypes between two eyes of each individual in a pedigree also supports this hypothesis.

**CASE REPORT**

A 64 year old man was referred with a 6–8 month history of non-resolving conjunctivitis, epiphora, and resultant lower eyelid ectropion. The conjunctivitis was resistant to treatment with Ocuflow OS four times daily. As a result, the referring provider added Tobradex (tobramycin) and Naphcon A (naphazoline), and referred him for evaluation. His ocular history was notable for bilateral pterygium excision 2 years earlier and open angle glaucoma. The patient's current ocular medications included Timoptic 0.5% in both eyes twice daily, Iopidine left eye twice daily, and Tobradex in left eye three times daily. Of historical note, the patient began the Iopidine approximately 2 months before developing these symptoms in the left eye. The patient had no known drug allergies. Corrected visual acuities were 20/20 right eye and 20/100 left eye. External examination revealed left upper eyelid ptosis, left lower punctal and eyelid ectropion, and diffuse left eye papillary conjunctivitis (Fig 1).

The Iopidine and the Tobradex were discontinued. The patient began prednisolone (Pred-Forte 1%) left eye every 2 hours in addition to his current regimen of Timoptic and Ocuprox. Within 3 weeks, his left eye improved dramatically. However, he developed a cicatricial entropion with symblepharon, keratinised lower eyelid margin, and persistent left upper eyelid ptosis requiring surgical correction (Fig 2). Tissue samples submitted for histological examination revealed lymphocytic infiltration, admixed with plasma cells, and foci of a haemorrhage within the conjunctiva and subepithelial stroma. Immunohistochemical preparations revealed 50% B cells and 50% T cells, leading to the diagnosis of reactive lymphoid infiltrate.

Iopidine allergy causing lower eyelid ectropion progressing to cicatrical entropion.

**Figure 1** Initial patient presentation demonstrating left upper eyelid ptosis, left lower eyelid ectropion, and diffuse conjunctivitis.

Iopidine allergy reaction presenting with eyelid ectropion which further progressed to cicatrical entropion.
COMMENT
Our finding of reactive lymphocytic infiltrates in the conjunctival specimen is consistent with a type IV hypersensitivity reaction and the diagnosis of Iopidine allergy. To our knowledge, this is the first report of an ectropion progressing to a cicatricial entropion resulting from an Iopidine allergy response. Iopidine has been reported to cause upper eyelid retraction and entropion. Similarly, dipivefrin, another adrenergic agent and a topical antiglaucoma medication, has been reported to cause lower eyelid ectropion; however, this ectropion resolved 3 weeks after drug discontinuation. In our case, the inflammation induced by the topical medication caused the initial ectropion which progressed upon resolution and scar formation to cicatricial entropion. Iopidine induced conjunctivitis can produce both ectropion and entropion.

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