CLINICAL STUDY

Pupillary distortion and staphyloma following trans-scleral contact diode laser cyclophotocoagulation: a clinicopathological study of three patients

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Abstract

Purpose **To search for the cause of scleral thinning and pupillary distortion following trans-scleral contact diode laser cyclophotocoagulation (TCDLC).**

Methods **We reviewed the records of 3 patients in whom there were complications of scleral thinning and pupillary distortion following TCDLC. One of the eyes was later enucleated, and we present the histopathological findings. Using the histopathological features in this patient, we discuss the possible pathogenesis of the scleral thinning and pupillary distortion.**

Results **Case 1 is a 46-year-old white woman who following TCDLC in an area of clinically normal sclera developed a staphyloma. Case 2 is a 52-year-old white woman who following TCDLC in an area of scarred sclera developed mild thinning. Case 3 is an 85-year-old white man who following TCDLC developed pupillary distortion, and gonioscopy revealed damage to the peripheral iris. Histological examination of case 1 revealed the staphyloma covered by a thin layer of conjunctival epithelium, collagen and vitreous condensation. We also observed cicatricial ciliary body contraction causing distortion of the pupil and lens.**

Conclusions **Therapeutic TCDLC can produce scarring of the iris root, anterior chamber angle, draining structures and ciliary body, and may result in pupillary distortion. Pre-existing scleral scars may predispose to scleral damage following TCDLC. We discuss a simple strategy to avoid this complication of TCDLC.**

Key words Cyclophotocoagulation, Diode, Histology, Laser, Pupillary, Scleral

Trans-scleral contact diode laser cyclophotocoagulation (TCDLC) appears to be a relatively safe and effective method of controlling intraocular pressure in cases of advanced or refractory glaucoma.[1–7] The diode laser delivers radiation at a wavelength of 810 nm. This wavelength of light usually passes through the sclera with minimal absorption, but is absorbed by the melanin pigment of the ciliary body. Histologically there is damage to the ciliary muscle and epithelium.[8–12] The pressure-lowering effect is probably due to a combination of increased uveoscleral flow and damage to the pigmented ciliary epithelium.

Pupillary distortion and scleral damage are rare complications of laser cyclophotocoagulation.[3,13–15] One case of focal scleral thinning has been reported following Nd:YAG cyclophotocoagulation,[14] but none following TCDLC. There are further reports of 2 cases of scleral perforation developing in areas of thin sclera following TCDLC,[3,13] and 1 case of pupillary distortion following TCDLC.[8]

We report 3 patients who developed complications after TCDLC: 2 patients without pre-existing scleral thinning developed varying degrees of scleral thinning (one with a staphyloma) and 1 patient suffered pupillary distortion. We also present the histological features following TCDLC in the case complicated by a staphyloma, and we discuss the pathogenesis of the scleral damage and pupillary distortion.

Materials and methods

We reviewed the records of 3 patients in whom there were complications of scleral thinning and pupillary distortion following TCDLC. All operations were performed by the same surgeon using a standard protocol with a diode system (Oculight SL_x) to produce the laser energy and deliver it through a contact G-probe attachment of the Iris medical diode laser (Mountain View, CA). The anterior edge of the G-probe footplate was placed at the

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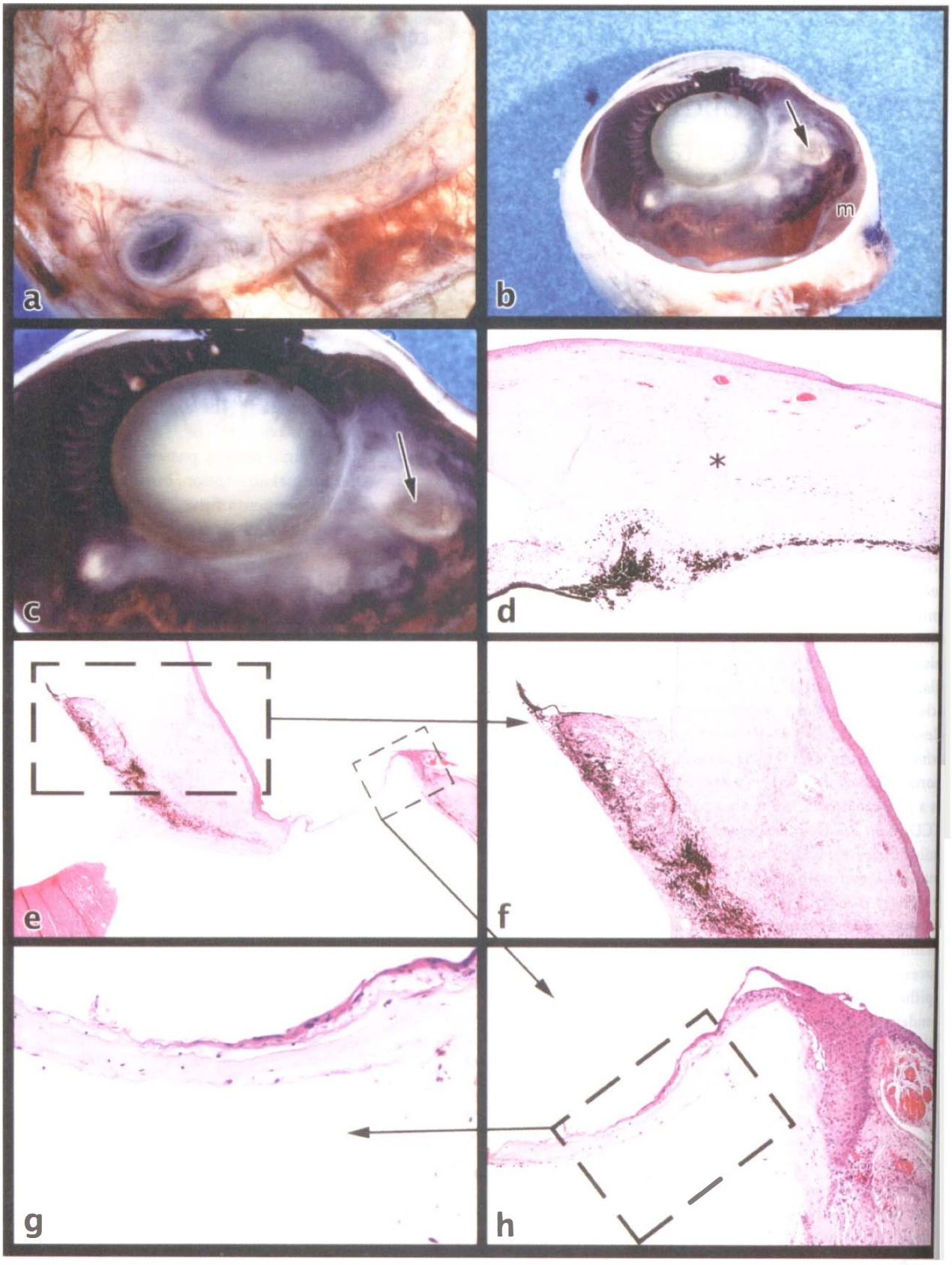

Fig. 1. *Case 1, right eye. Macroscopic pictures: (a) The pupil is white (cataract) and distorted inferiorly, with ectropion uveae. The well-circumscribed staphyloma is at 7 o'clock. (b) and (c) An oblique superior temporal calotte has been removed, exposing a 180° 5 mm wide band of ciliary body destruction, with a white cyclitic membrane extending onto the lens (and a posterior subcapsular cataract). The staphyloma (arrows) is in the pars plana, and there are two white scars in the pars plana/pars plicata. There is residual brown malignant melanoma (m) posterior to the equator, with a black nodule of melanophages at its anterior edge. Note two white Fuchs' 'adenomas' in ciliary processes above the lens. Histological sections: (d) The lasered ciliary body, limbus and anterior chamber angle (left), centred on the scleral spur (asterisk). Note the destruction and fibrosis of the ciliary epithelium (residual pigment), the ciliary muscle and trabecular meshwork; and anterior synechiae with neovascular membrane on the anterior iris surface. Sections (e)–(h) are from the area of the staphyloma. (e) An overview of the staphyloma just behind (below) the obliterated cilitary body. Note the obliteration of episcleral, scleral and uveal tissue in the staphyloma (e, g, h). The staphyloma has a thin layer of epithelium, collagen and consensed vitreous (g), and there is some epithelial downgrowth into the uvea (h, right). The iris is retracted towards the ciliary body scar, and there is traction on, and distortion of, the lens (e). Note the anterior synechiae and pupillary membrane (e). H&E sections. Original magnifications: (d) ×10; (e) ×5; (f) ×10; (g) ×50; (h) ×25.*

corneoscleral limbus and each laser application was spaced one half-width of the G-probe footplate apart.

Histopathological examination was possible in one patient's eye which was enucleated because of continuing discomfort.

Case reports

Case 1

A 46-year-old white woman presented in 1997 with a right posterior pole choroidal malignant melanoma. This was treated with stereotactic radiosurgery with a good response; however, she subsequently developed filamentary keratitis and rubeotic glaucoma. Despite maximum medical treatment the intraocular pressure remained high and she developed bullous keratopathy. TCDLC was performed: 10 applications to the inferior 180° of the sclera (each application being of 2 W power and 2 s duration). Five audible 'pops' were heard and no scleral thinning was observed at the time of TCDLC application. Two weeks later she had developed a staphyloma inferotemporally in the region of TCDLC application. The bullous keratopathy persisted and she developed ocular hypotony. The eye remained uncomfortable and she elected to have an enucleation 4 months later.

Macroscopically there was distortion and inferior displacement of the pupil, with ectropion uveae (Fig. 1a), and a well-circumscribed 2 mm diameter staphyloma, situated 2 mm from the corneoscleral limbus at 7 o'clock. Within the eye there was a regressed choroidal malignant melanoma in the temporal quadrant posterior to the equator, and a posterior subcapsular cataract. In the inferior half of the globe the area affected by the cyclodiode was seen as a 5 mm wide band of ciliary body scarring, with an overlying white cyclitic membrane extending onto the posterior surface of the lens. The staphyloma, and two further white scars (1 mm and 1.5 mm), were situated in the pars plana.

Histologically, the laser-treated area included focally pigmented scarring incorporating the iris root, anterior chamber angle, drainage structures and ciliary body (Fig. 1d, f). Traction from this scar had caused pupil and lens distortion (Fig. 1e). At the site of the staphyloma, there was sharp-edged defect of the episclera, sclera and uvea (Fig. 1e). The strand of tissue crossing the defect consisted only of a thin layer of conjunctival epithelial cells over strands of collagen, lined internally by condensed vitreous (Fig. 1g). Some conjunctival epithelial cells had grown downwards a short way over the uvea adjacent to the defect (Fig. 1h). In addition to the residual choroidal malignant melanoma posterior to the equator, radiation-induced changes were present, including rubeotic glaucoma with ectropion uveae, posterior subcapsular cataract and cystoid macular oedema.

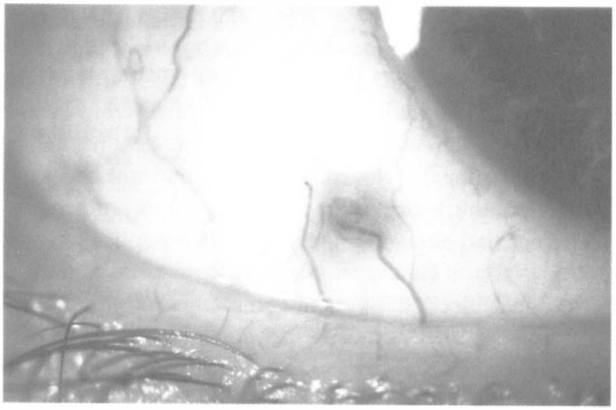

Fig. 2. *Case 2. Left eye, 2 weeks after TCDLC treatment. There is a 2 mm diameter focal area of scleral thinning in the treated region.*

Case 2

A 52-year-old white woman presented in 1996 with advanced proliferative diabetic retinopathy in the left eye. She had a vitrectomy, membrane peel and endolaser, which was complicated by an intraoperative retinal tear and detachment. In 1997 she had a trabeculectomy with 5-fluorouracil for uncontrolled glaucoma. She developed left rubeotic glaucoma in 1999, which could not be controlled on maximum medical treatment. TCDLC was performed, with 10 applications to the inferior 180° of the sclera, each application being of 2 W power and 2 s duration. As a result TCDLC was applied to the sclerostomy site. Two weeks later a 2 mm diameter focal area of scleral thinning was observed in the inferotemporal quadrant, corresponding to the previous sclerostomy site (Fig. 2).

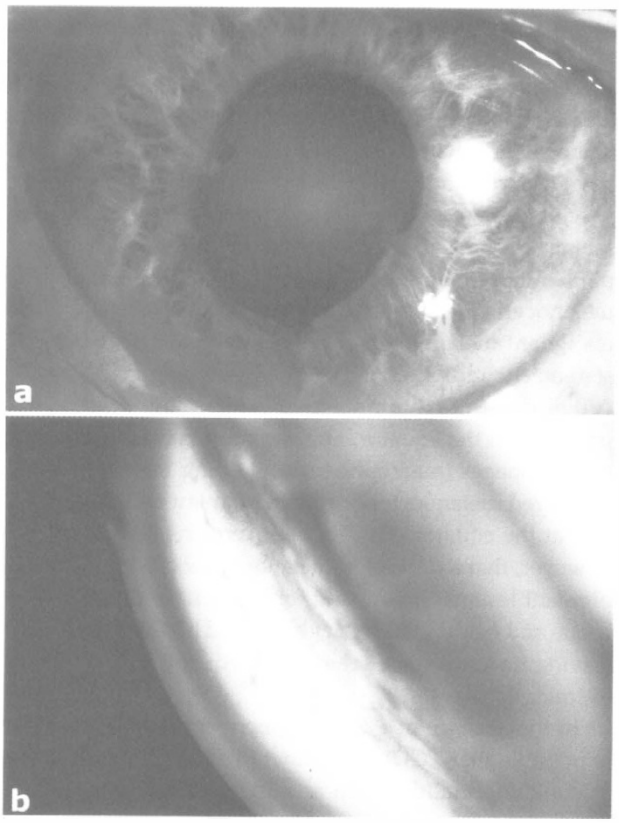

Fig. 3. *Case 2. Right eye, 6 weeks after TCDLC treatment. (a) There is pupillary distortion towards the treated area. (b) Gonioscopy shows damage to the peripheral iris and angle structures.*

Case 3

An 85-year-old white man presented in 1996 with a right inferonasal choroidal malignant melanoma, which was treated with stereotactic radiosurgery. In 1999 he developed rubeotic glaucoma which could not be controlled on medical therapy. TCDLC was performed; treatment had to be stopped after only 3 applications (of 2 W power and 2 s duration) because, in spite of the retrobulbar block, the patient complained of pain and kept moving the eye in the direction of the probe. The surgical limbus was difficult to identify because of the presence of arcus senilis. Two audible 'pops' were heard. Six weeks later he had developed pupillary distortion, and gonioscopy revealed mild peripheral iris damage in the treated region (Fig. 3).

Discussion

The exact mechanism by which TCDLC causes lowering of intraocular pressure is not known but the target tissue is usually taken to be the pars plicata. Photocoagulation of tissue occurs when pigments such as melanin or haemoglobin absorb light energy produced by the diode laser. Whilst the pigmented ciliary epithelium is the intended target in TCDLC therapy, any melanin or haemoglobin in the path of the laser could absorb energy, and cause tissue damage. In cases 1 and 2, tissue damage occurred as a result of TCDLC therapy. It is possible that this damage results from the absorption of laser energy by the pigment present in the lamina fusca, which can be of variable thickness and pigmentation, or areas of abnormal pigmentation within the damaged tissues or adjacent structures. None of the patients treated by TCDLC had any known local pigmented lesion in the area of resultant focal scleral damage, i.e. lesions such as haemosiderin within the sclera or episclera (which could have occurred following the previous surgery in case 2), vascular abnormalities of the conjunctiva, episclera, sclera of ciliary body, pigmented foreign body, scleral pigmentation, pigmented ciliary body adenoma, melanocytic melanoma or naevus. In case 1, a choroidal malignant melanoma had been previously treated by stereotactic radiotherapy, with a good response. Although residual tumour was present, this was posterior to the equator, and the only known nodule of melanophages (macrophages containing melanin from destroyed melanocytes or retinal pigment epithelial cells) was at the equator (Fig. 1b, c). It remains possible, however, that a deposit of melanoma (or one of the above pigment lesions) was in and/or adjacent to the damaged tissue and could have been obliterated by the diode laser, leaving no trace to be seen at subsequent histological examination.

In the absence of a local pigment-containing lesion, it is possible that TCDLC treatment in the presence of scleral abnormalities such as thinning could result in perforation or staphyloma. In case 2 the area of focal scleral damage was at the sclerostomy site performed during a previous vitrectomy. In this case the scar may have been structurally weak and/or thin. Scar tissue is known to absorb more heat than normal tissue. A combination of these factors may have resulted in the scleral damage seen in case 2.

Another possible cause of increased tissue damage is increased energy delivery by the diode laser. However, we believe this is most unlikely, since the same power/time setting was used to multiple sites but there was only tissue damage at focal sites. This suggests that some local factor is responsible for the damage.

There is, however, a further means by which the TCDLC could cause local damage. If blood and/or debris collects on the tip of the diode, an unchanged energy setting can cause a marked increase in tip temperature.[16] This local heat can then result in tissue damage. However, we found no evidence of charred tissue, in or around the staphyloma, in case 1 to support this hypothesis.

There has been one reported case of anterior displacement of the laser spot resulting in iris injury and pupillary distortion.[15] All of the above mechanisms assume that the diode laser has been correctly positioned on the sclera before discharge. However, because the pars plicata is not directly visible, the technique is essentially performed 'blindly'. Any variation in anatomy of the ciliary body,[17] or any problems with either the location or orientation of the probe, results in the laser spot being focused on tissue other than the pars plicata. Probe tip placement of 0.5 mm from the surgical limbus[18] and change in orientation of the probe tip[19] can result in peripheral iris damage. The G-probe is designed to fit the contour of the sclera, place burns 1.2 mm from the surgical limbus and orientate the diode beam parallel to the visual axis. In our case 3, identification of the surgical limbus was made difficult by the presence of arcus senilis and by the patient moving the eye. We believe that this resulted in the anterior edge of the probe being placed anterior to the surgical limbus, and the laser being directed towards the iris; this may explain the pupillary distortion seen post-operatively in case 3. In case 1, we have shown that TCDLC can produce cicatricial contracture of the ciliary body and pupillary distortion (Fig. 1e). Since there was only minimal iris damage, the pupillary distortion seen in case 3 may be due to a combination of damage to the peripheral iris and ciliary body cicatricial contracture.

In case 1 we have shown that TCDLC caused disruption of the epithelium of both the pars plicata and (to a lesser degree) the pars plana, with associated destruction of the muscle of the ciliary body, and these histological features are similar to those in previous reports.[8–12] As far as we are aware this is the first report of *in vivo* TCDLC-induced scarring of iris root, anterior chamber angle, drainage structures and ciliary body (Fig. 1d, e) with traction from this scar causing pupil and lens distortion (Fig. 1e). This is also the first report of the histopathological features of a staphyloma following TCDLC.

It appears that there may be several mechanisms, acting alone or in combination, whereby abnormal tissue damage can result from TCDLC treatment. Such tissue damage, with resulting iris and pupillary distortion and/or staphyloma, may be avoided if we pay attention to following:

1. Avoid or treat cautiously areas of abnormally thin or scarred sclera and pigmented or vascular lesions of the conjunctiva, episclera, sclera and ciliary body.
2. Inspect and wipe the tip of the G-probe between applications.
3. Transilluminate the eye to identify the position of the ciliary body. In cases where transillumination is not possible, ultrasound biomicroscopy or axial length measurements can be used as a guide to the location of the ciliary body.

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