European Journal of Ophthalmology / Vol. 14 no. 1, 2004 / pp. 74-82

Case report

Indocyanine green dye-enhanced micropulsed diode laser: a novel approach to subthreshold RPE treatment in a case of central serous chorioretinopathy

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PURPOSE. *To present a case of central serous chorioretinopathy (CSC) treated with indocyanine green (ICG) dye-enhanced subthreshold micropulsed diode laser photocoagulation.*
METHODS CASE REPORT. *A 35-year-old man presenting with recurrent CSC with persistent serous detachment of the sensory retina in his left eye who declined treatment with a 532 nm laser. Subthreshold treatment, with no visible endpoint, was performed with an 810 nm diode laser 15 minutes after the injection of 25 mg ICG in 2 cc of 5% glucose solution. The laser energy was delivered over the active leakage sites with a sequence of repeated 500 ms "envelopes" each containing a train of 250 micropulses with 500 mW peak power at 10% duty cycle (200 µs ON and 1,800 µs OFF) and each separated by 500 ms intra-envelopes relaxation time. Due to the absence of visible laser-induced lesions, post treatment ICG digital angiographic images were taken without further dye injection to verify that the hypofluorescent spots resulting from the subthreshold laser applications coincided with the points of leakage.*
RESULTS. *After 7 days, the patient presented with a less hyperopic refraction, improved visual acuity, and reduction of serous neuroepithelial detachment. No signs of laser treatment were visible at fluorescein angiography. After 8 weeks, the serous neuroepithelial detachment was almost completely resolved.*
CONCLUSIONS. *ICG dye-enhanced subthreshold micropulsed diode laser photocoagulation appears to be a safe and effective treatment and represents a possible approach for the management of chronic CSC with persistent central serous neuroepithelial detachment. Immediate post treatment ICG angiography, without ICG reinjection, allows documenting the actual number and location of the delivered subthreshold laser applications. (Eur J Ophthalmol 2004; 14: 74-82)*

KEY WORDS. *Central serous chorioretinopathy, Dye-enhanced photocoagulation, Minimum intensity photocoagulation, Indocyanine green angiography, Optical coherence tomography*

Accepted: December 11, 2003

1120-6721/074-09$15.00/0

INTRODUCTION

Central serous chorioretinopathy (CSC) is characterized by idiopathic serous detachment of the sensory retina secondary to focal defects of the retinal pigment epithelium (RPE), e.g., multiple small serous detachments and/or localized areas of atrophy (1). It is most commonly seen in men 20 to 50 years of age and can be associated with type A personalities (2), organ transplantation (3, 4), systemic and intranasal use of steroids (5, 6), systemic lupus erythematosus (7, 8), Cushing's disease (9), and other systemic factors (10). CSC usually regresses spontaneously within several months but recurrence is observed in 33% to 50% of all cases (11, 12). Direct focal laser treatment of the RPE leakage point accelerates resolution of the detachment but the final vision is similar to that produced by spontaneous healing (13-15). For this reason, laser photocoagulation is normally considered only in particular cases such as those associated with persistent (>4 to 6 months) or progressive detachment (with or without inferior guttering), permanent CSC changes in the contralateral eye, multiple recurrences, or the need for rapid recovery of vision for professional reasons. Treatment complications include central scotoma, loss of contrast sensitivity, accidental foveal damage, retinal distortion, and choroidal neovascularization (CNV) (16-18), which also develops spontaneously in around 15% of CSC patients (19).

Recent studies (20-23) support the hypothesis that subthreshold, minimum intensity photocoagulation (MIP) with 810 nm micropulsed diode laser can minimize the iatrogenic damage and that the mild laser-induced lesions confined to the RPE (with no intraoperative and postoperative visible endpoint by ophthalmoscopy) can suffice to trigger the therapeutic benefits. The absence of an intraoperative retinal blanching renders subthreshold treatments difficult to deliver and to document. With the purpose of increasing the RPE treatment selectivity and of obtaining an immediate postoperative documentation of the invisible MIP applications, we have used a novel technique using indocyanine green (ICG) staining of RPE cells prior to MIP treatment. To our knowledge, this is the first time that a case of CSC with persistent serous neuroepithelial detachment has been treated with this ICG staining MIP technique.

METHODS

A 35-year-old man presented with idiopathic chronic CSC in his left eye with two serous detachments of the sensory retina (Fig. 1A).

Fluorescein angiography (FA) showed two main active leakage sites associated with serous detachments (Fig. 1B). A third less pronounced active leakage site was also observed adjacent to a retinal vein bifurcation above the optic disk (Fig. 1B). On optical coherence tomography (OCT) the central detachment was characterized by retinal thickening due to edema (Fig. 1, C and D).

At admission examination, best-corrected visual acuity (BCVA) was 0.0 logMAR (with +0.50 D cylinder 90°) in the right eye and 0.3 logMAR (with +1.25 D sphere = +0.50 D cylinder 90°) in the left with marked metamorphopsia.

Due to the persistence (>6 months) and the progressive increase of the central detachment, laser treatment of the related active leakage site locations was offered to shorten the course of disease. Informed about the possible complications (central scotoma and potential risk of CNV formation) of the conventional treatment with a 532 nm laser, the patient declined. As an alternative, he was offered an ICG dye-enhanced MIP treatment with a micropulsed 810 nm diode laser. He accepted, despite the specific warning that the absence of an intraoperative visible endpoint could carry the risk of an insufficient treatment. After signing the informed consent, the patient received an injection of a bolus of 25 mg ICG in 2 cc of 5% glucose solution (Infracyanine SERB). The laser treatment, with an 810 nm infrared diode laser (IRIS Medical OcuLight SLx, IRIDEX Corporation, Mountain View, CA) in its MicroPulse operating mode, was started after 15 minutes from the injection, when the background fluorescence due to ICG staining of the RPE–Bruch membrane complex became apparent at ICG angiography (24).

The parameters employed in this treatment were based on our clinical experience with ICG-enhanced micropulsed laser therapy, and, in terms of both irradiance and fluence, they were lower than values that produce a barely visible endpoint and higher than those associated with no appreciable therapeutic effect.

The peripheral active leakage site was treated first using a sequence of 50 repeated 500 ms "envelopes," each containing a train of 250 micropulses with 500

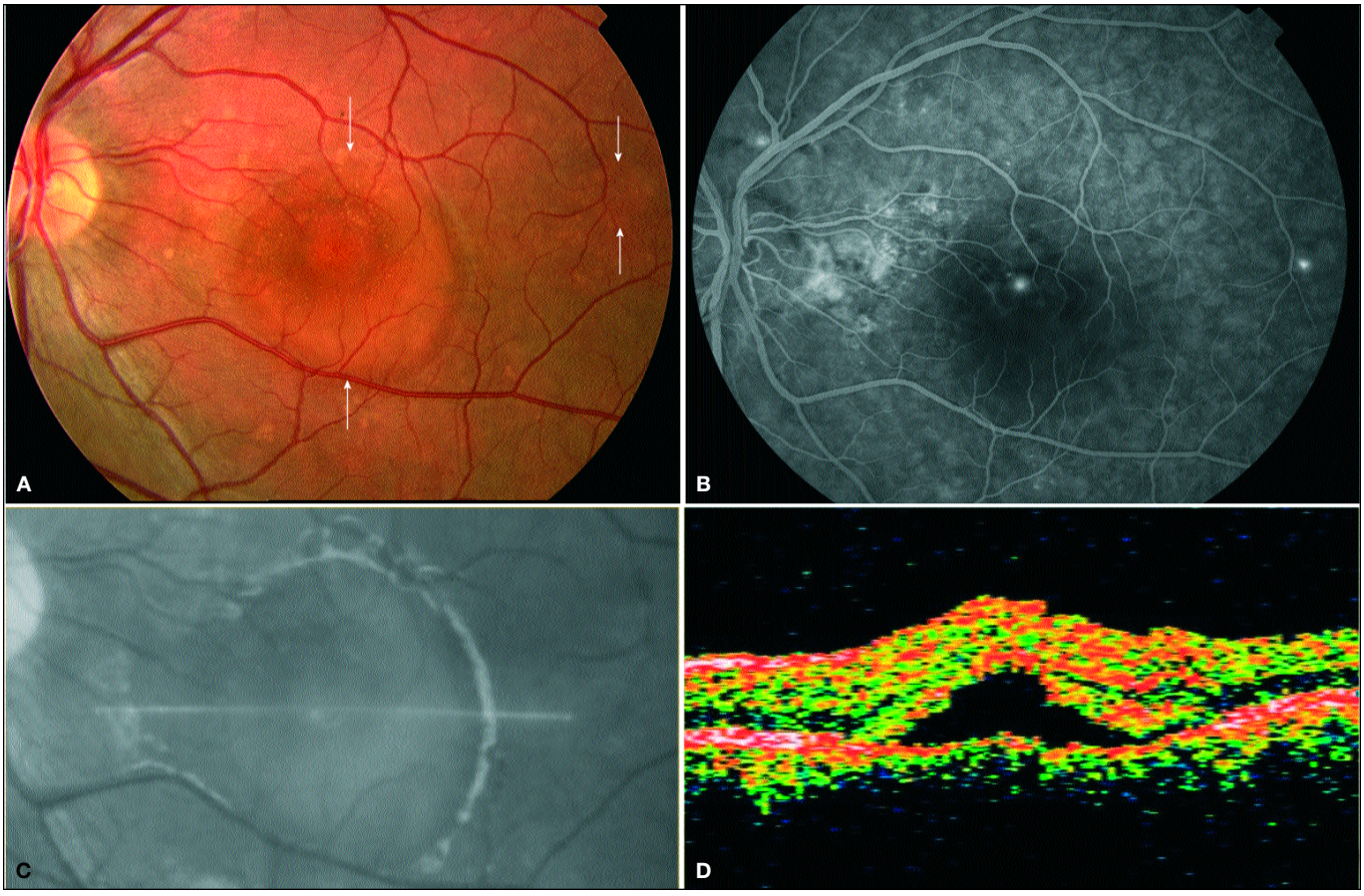

Fig. 1 - A) *Color retinography at presentation shows two serous retinal detachments (white arrows).* **B)** *Fluorescein angiography at the same time shows two related active leakage sites.* **C)** *Optical coherence tomography (OCT) panoramic view of the retinal serous detachment scanning.* **D)** *OCT scan showing thickened retina and serous retinal detachment.*

mW peak power at 10% duty cycle (200 µs ON and 1,800 µs OFF) and each separated by 500 ms intra-envelopes relaxation time, for a total exposure duration of 50 seconds.

This proved to be a true subthreshold treatment with no intraoperative or postoperative visible tissue reaction. For the central active leakage sites, as a further precaution against unwanted late side effects, the number of sequential envelopes was reduced to 25, for total exposure duration of 25 seconds.

The third active leakage site was not considered clinically significant and was not treated. As expected, no ophthalmoscopically visible fundus changes appeared during or after the treatment. The only sign of treatment was revealed by the postoperative ICG angiography, without reinjection, which showed hypofluorescent dark spot at each treated site (Fig. 2, A and B). The digital overlay of the ICG angiography hypofluorescent spots on the FA image (Fig. 2C) demonstrates that the subthreshold laser treatment has been effectively placed over the points of leakage. The digital overlay of the ICG angiography hypofluorescent spots on the fundus color photography (Fig. 2D) demonstrates that the subthreshold laser treatment has not produced visible fundus changes in both treated sites.

Figure 3, A and B, shows fundus color photography and FA findings immediately before the laser treatment. At the 7-day follow-up visit, the patient presented with reduced metamorphopsia, less hyperopic refraction (+0.50 D sphere = +0.50 D cylinder 90°), improved VA up to 0.0 logMAR, and reduced serous neuroepithelial detachment (Fig. 3C). FA showed neither signs of dye leakage nor signs of laser-

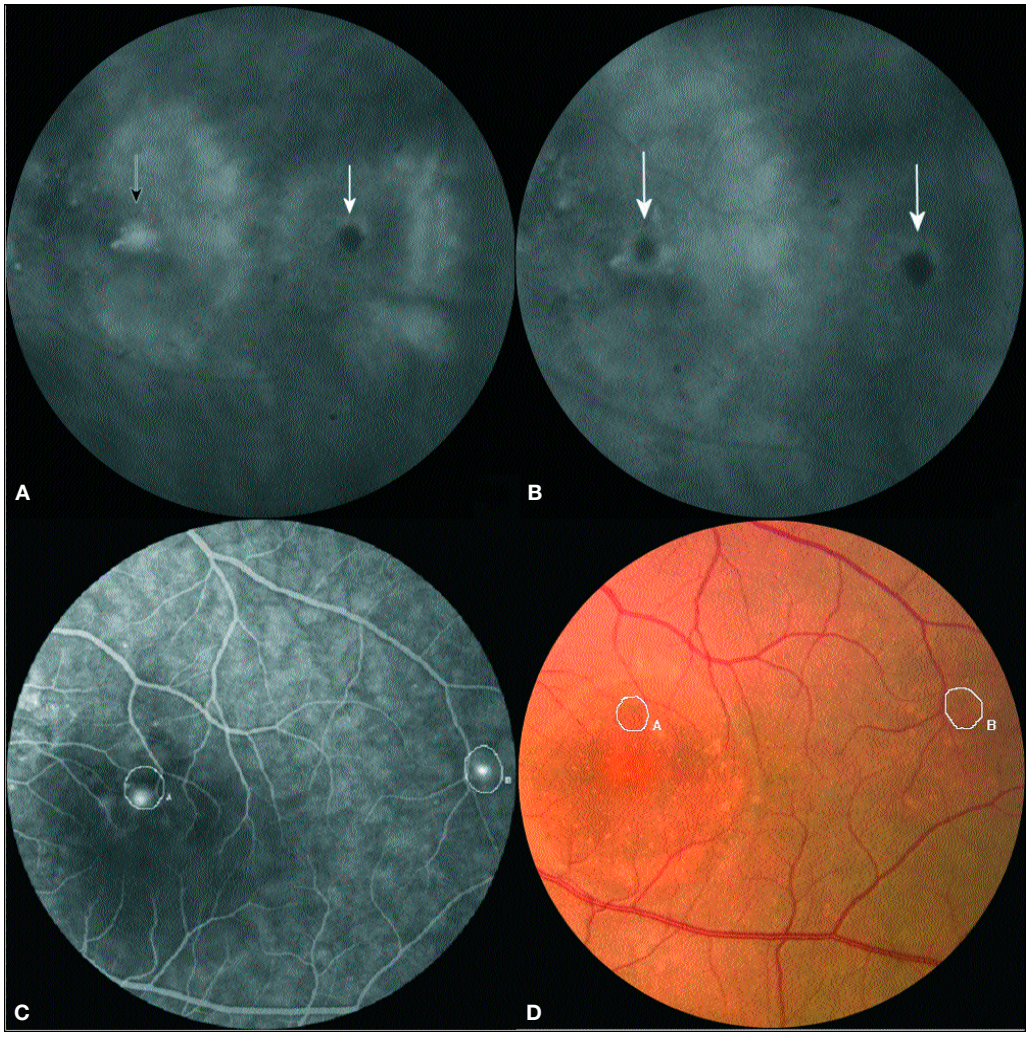

Fig. 2 - A) *Indocyanine green angiography (ICGA) hypofluorescent dark spot surrounded by a hyperfluorescent halo after the first micropulse laser treatment of the peripheral lesion (white arrow). The untreated macular pinpoint is still stained with the dye (black arrow). This first treatment was intended as a test burn.* **B)** *Late-phase ICGA immediately after the second micropulse laser treatment. In both locations, the subthreshold treatment is revealed only by ICGA dark hypofluorescent spots surrounded by hyperfluorescent halos (white arrows).* **C)** *Digital overlay of the ICGA dark hypofluorescent spots on the fluorescein angiography (FA) image: there is a correspondence between the dark spots and the active leakage sites.* **D)** *Digital overlay of the ICGA dark hypofluorescent spots on the color fundus photography. No visible retinal changes can be detected at the sites of treatment.*

induced scar but only diffuse aspecific RPE degenerative changes due to the prolonged retinal detachment (Fig. 3D).

After 8 weeks (Fig. 3, E and F), the neurosensory detachment was almost completely resolved and FA did not show any pinpoint leakage or RPE window focal defect related to laser-induced scar. Hyperopia was further reduced (+0.50 D cylinder 90°) and VA improved to -0.1 logMAR. Figure 4 shows the correlation between the fundus color image (A and B), the FA (C and D), and the OCT scans (E and F), before and 8 weeks after the ICG-enhanced MIP treatment. At presentation, OCT cross sectional images of the center of the fovea showed the thickening of the detached retina and low reflectivity of swollen intraretinal areas. Eight weeks after the treatment, the OCT scans showed thinning of the retina and near total absorption of subretinal fluid. These variations of retina thickness are a common feature in the evolution of CSC (25).

DISCUSSION

Numerous authors have reported that conventional laser treatment with visible endpoint and associated neurosensory retina thermal damage can facilitate resolution of the exudative manifestations in CSC (13-15). This approach remains controversial, however, because equally favorable visual outcomes can

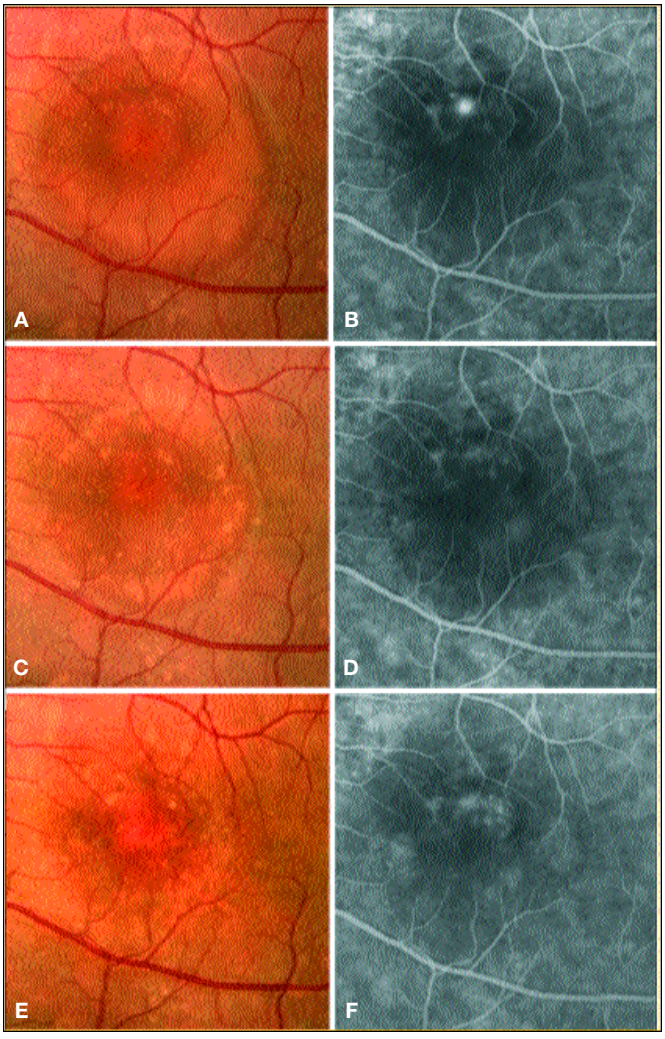

Fig. 3 - A) *Before the minimum intensity photocoagulation (MIP) treatment.* **B)** *Fluorescein angiography (FA) before the MIP treatment.* **C)** *One week after the treatment: reduction of the sensory-epithelium detachment.* **D)** *One week after the treatment, there is no evidence of pinpoint dye diffusion or laser scar on FA.* **E)** *Eight weeks after the treatment, there is sensory-epithelial detachment flattening. No laser scar is evident.* **F)** *Eight weeks after the treatment, there is neither pinpoint dye diffusion nor sign of laser-induced retinal pigment epithelial change on FA.*

generally be achieved without any treatment at all (26, 27). Nevertheless it has been suggested that the persistence of subretinal fluid may be associated with a reduced final VA (28), and this has compelled the study of experimental active leakage site treatment modalities to reduce complications and side effects. In a recent report, CSC showed a favorable clinical response to an RPE-selective subthreshold laser treatment, which was administered using an experimental 532 nm Nd:YAG laser delivering repetitive short pulses (0.8-1.7 microseconds) with quasi-adiabatic tissue interaction and thermo-mechanical effects (29, 30).

RPE specificity and neurosensory retina sparing have been two major considerations in devising our subthreshold treatment protocol. To this end, the treatment of ICG retaining RPE cells with an 810 nm diode laser in its MicroPulse delivery mode has been chosen for the following theoretical advantages:

• In melanin, the major retinal light absorbing chromophore, 810 nm has lower absorption and longer extinction length than any visible wavelength of most used lasers. This translates in deeper tissue interaction, enabling a more discrete and controllable photothermal elevation at the RPE to minimize the thermal damage to overlying neurosensory retina (31).

• The use of repetitive microsecond pulses at low duty cycle and low repetition rate (10% and 500 Hz) minimizes the intraoperative axial thermal spread from the RPE toward the adjacent structures (32).

• The use of 810 nm diode laser on ICG-stained RPE cells enhances treatment's RPE selectivity and neurosensory retina sparing capability. ICG bound to plasma proteins has an absorption peak at 805 to 810 nm that perfectly matches the diode laser emission (33). ICG angiography shows that in CSC the dye accumulates at higher concentration at the active leakage sites (Fig. 2A). Enhanced localized laser uptakes allows for the use of reduced laser energy and this can lead to a more effective retina sparing.

• Long exposures with multiple trains of repetitive microsecond laser pulses allow for 1) gradual stimulation of natural thermoprotective mechanisms leading to increased thermotolerance of the neurosensory retina and 2) more consistent and predictable subthreshold treatment with no visible endpoint (34).

• The photo bleaching of ICG fluorophore retained in the RPE cells caused by the treatment provides the documentation of all subthreshold laser applications, which show as hypofluorescent spots on postoperative ICG angiography without ICG reinjection (Staurenghi G, Unpublished data).

In this case, prompt clinical resolution occurred after the dye-enhanced subthreshold MIP treatment, which was so light that no signs of treatment were observ-

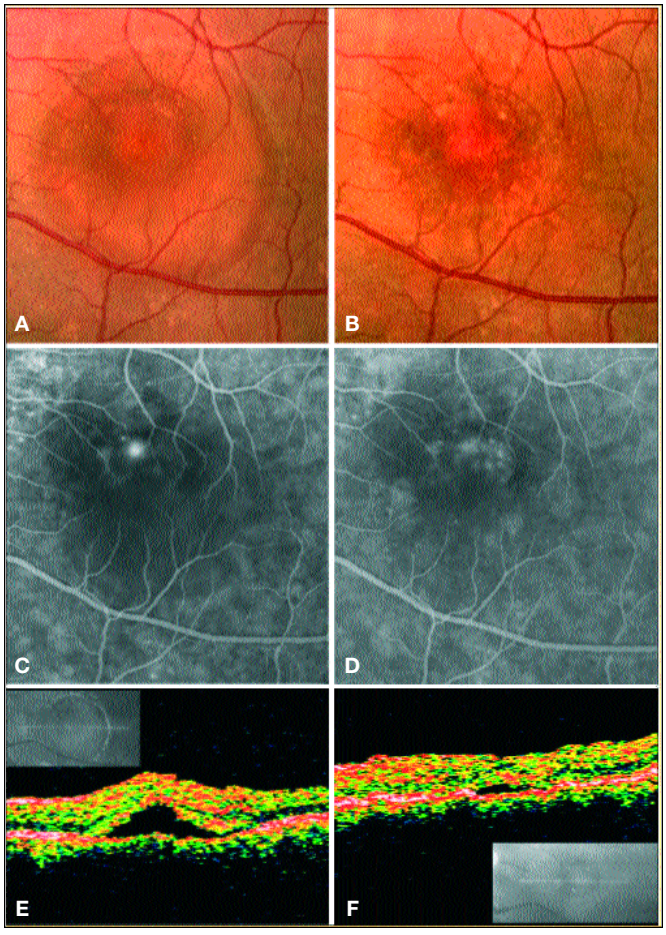

Fig. 4 - A) *Serous detachment of the sensory retina before the minimum intensity photocoagulation (MIP) laser treatment.* **B)** *Eight weeks after the MIP laser treatment.* **C)** *Fluorescein angiography (FA) before the MIP laser treatment: two main leakage points are detectable.* **D)** *No evidence of pinpoint dye diffusion 8 weeks after the MIP laser treatment on FA.* **E)** *Optical coherence tomography (OCT) appearance of the serous sensory-epithelial detachment before the treatment.* **F)** *OCT of the central retina 8 weeks after the MIP laser treatment: the flattening of the serous detachment is nearly completed.*

able on color fundus photography or on FA. No retina blanching and no RPE alteration (laser scar) occurred. Two hypofluorescent spots on postoperative ICG angiography without dye reinjection provided the only documentation of laser-induced changes at the RPE cells or, more correctly, at the ICG dye retained in the RPE cells. The absence of intraoperative and postoperative inner retinal blanching seems to support the ability of this treatment to spare the neurosensory retina by axially confining the laser photothermal effects at the RPE, as reported in experimental works (35, 36).

The treatment of ICG-stained chorioretinal structures with an 810 nm laser, for the purpose of enhancing both laser uptake and treatment selectivity, is not a novelty and has been proposed for the treatment of occult CNV secondary to age-related macular degeneration (ARMD) (37, 38), CNV feeder vessels (39), choroidal metastasis (40), and choroidal hemangioma (41).

ICG has been recently proposed as a photodynamic agent, which can be light activated by the 810 nm diode laser, for treatment of neovascular complexes secondary to ARMD (42, 43) and active leakage sites in persistent CSC (44).

The common rationale is that, once bound to plasma proteins, ICG has an absorption peak at 805 to 810 nm that perfectly matches the diode laser emission. The exposure of ICG-stained tissues to low intensity 810 nm laser light would result in a more selective treatment with synergistic photothermal (heat conversion by both endogenous chromophores and by the ICG dye) (45) and photochemical (photooxidation type II) cytotoxic damage mechanisms (46, 47).

In this case, the clinical improvement is not supported by intraoperative or postoperative ophthalmoscopically or angiographically observable laser-induced effects, which could confirm a laser treatment, as conventionally intended. A natural resolution could be responsible for the clinical improvement, but the leakage still present in the untreated third active leakage site suggests a treatment effect (Fig. 5, A-C).

If the ICG-enhanced subthreshold laser treatment, rather than a natural resolution, is responsible for the clinical improvement, then the hypothesis for the mechanism of action should be attempted. This task may prove particularly challenging considering that the pathogenesis of CSC and the mechanism of action of laser photocoagulation are both poorly understood.

Different pathogenetic models have been proposed to explain the neurosensory detachment that occurs in CSC. Some authors have hypothesized the presence of a primary defect localized in the cells of the RPE (47), while others suspect that a focal RPE lesion occurs as a result of diffuse circulatory changes at the level of the choroid (48, 49). The first model (47), which is based on the hypothesis of transport

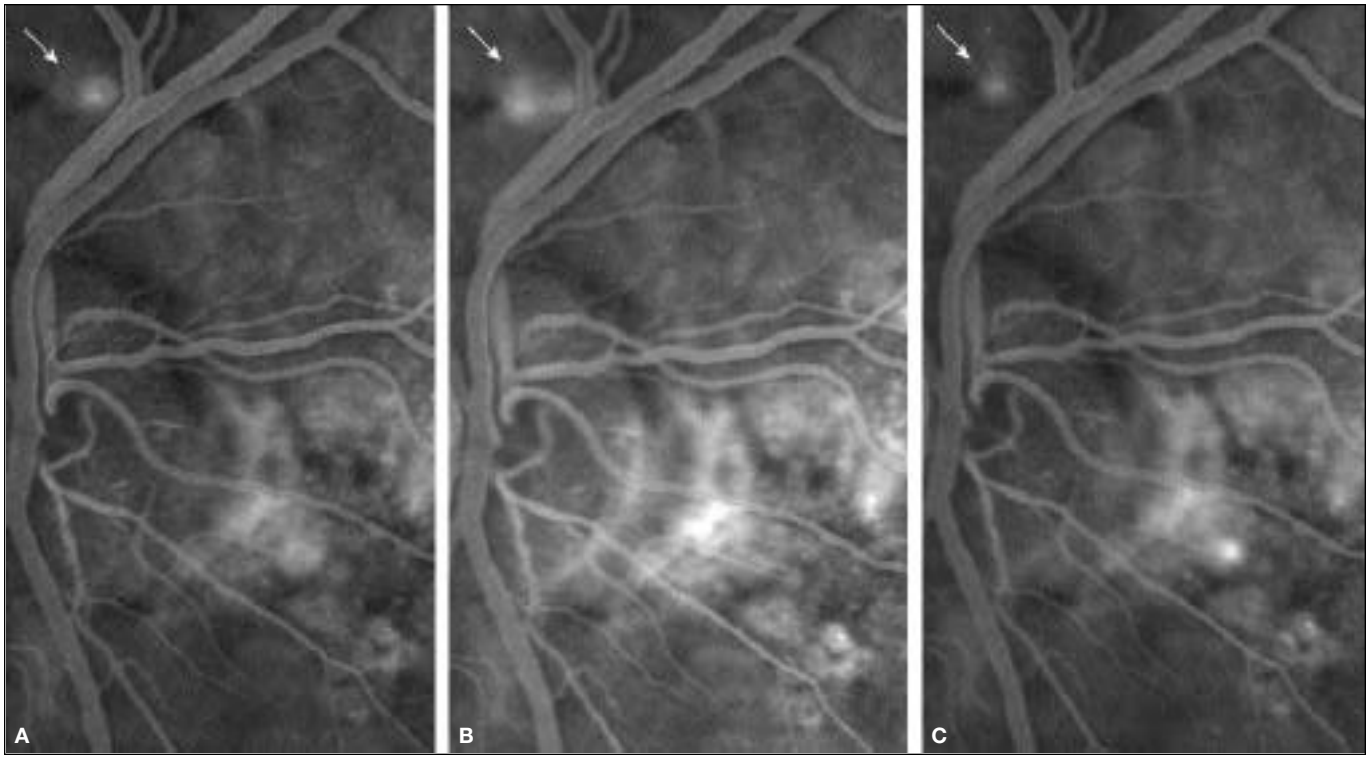

Fig. 5 - A) *Fluorescein angiography detail of the third active leakage site before the treatment. The untreated active leakage site is still evident 1 week* **B)** *and 8 weeks* **C)** *after the minimum intensity photocoagulation laser treatment.*

polarity inversion in a localized group of RPE cells damaged by immune, neuroendocrine, or infective processes, suggests that subthreshold laser injury to such cells might be capable of destroying those cellular elements responsible for the pathology (50).

The second model (48, 49) suggests that localized choroidal vascular changes might alter the natural permeability of the choroidal capillaries, leading to increased leakage and pooling of fluid and plasma proteins beneath the RPE cell layer. The pressure exerted by this fluid would cause stretching of the overlying RPE cells that could compromise the tight junctions, thus allowing the fluid to leak into the subretinal space through a focal defect of the outer blood retinal barrier (BRB), which constitutes the active leakage sites. In this model, the hypothesis on the mechanism of action of subthreshold MIP laser treatment of the active leakage sites could be that the healing response, following the laser-induced RPE cellular injury, can restore the integrity of the outer BRB, closing the source of subretinal fluid (51).

CONCLUSIONS

ICG-enhanced subthreshold micropulsed diode laser photocoagulation for persistent neurosensory detachment in CSC appears to be a rational and attractive protocol, whose benefits, based on this study, are not conclusive. Despite the limitations of this case report, the encouraging result and the lack of complications suggest that ICG-enhanced subthreshold micropulsed diode laser photocoagulation could be considered for the treatment of other RPE-related retinal disorders, which normally respond to conventional photothermal treatment and could benefit from a less damaging subthreshold protocol.

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