

Conclusion

We have presented a low-cost and easily reproducible simulation model for the practise of corneal foreign body and rust ring removal that improves participant confidence and may readily be incorporated into departmental clinical skills training.

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Footnotes and Disclosure

The authors have no proprietary or commercial interest in any materials discussed in this article.

Bacillary layer detachment associated with photodynamic therapy-related acute exudative maculopathy in a case of central serous chorioretinopathy

A 45-year-old man with a history of recurrent/persistent central serous chorioretinopathy (CSCR) over the preceding 2 years



presented to the Duke Eye Center with progressive vision decline in his left eye. His baseline visual acuity (VA) was 20/60. Foveal pigment changes were observed in the left eye. The findings of an anterior segment examination of the left eye and a complete eye examination of the right eye were unremarkable. On spectral-domain optical coherence tomography (SD-OCT), there was subretinal fluid (SRF) and elongation of the photoreceptor outer segments. There was a focal granular hyper

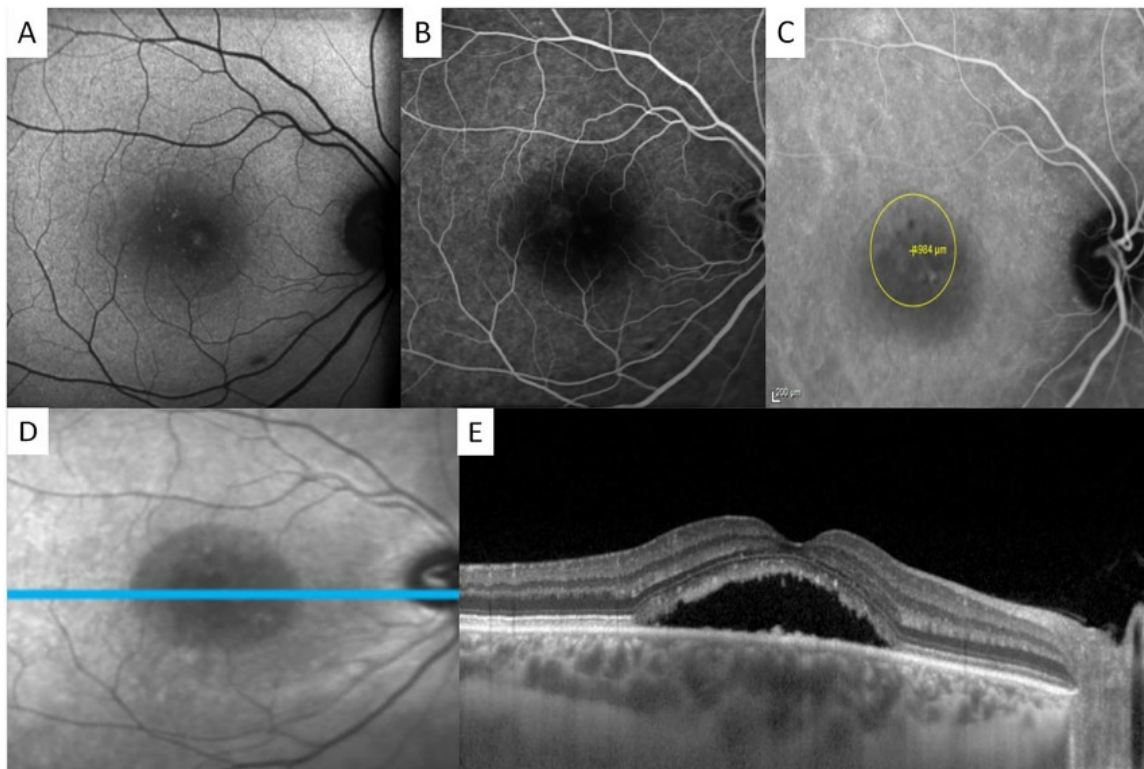


Fig. 1 – Multimodal images of the patient with chronic central serous chorioretinopathy at the baseline (diagnostic). (A) Fundus autofluorescence imaging shows central hypoautofluorescence secondary to the masking (blocking) effect of the fluid. (B) and (C) Fluorescein angiography shows a focal granular hyperfluorescent area, whereas indocyanine green angiography shows punctate hyperfluorescent spot. The yellow circle shows photodynamic therapy spot size and location. (E) A horizontal optical coherence tomography B-scan shows subretinal fluid, elongation of the photoreceptors layer with the most protruding outer segment of photoreceptor, and hyper-reflective subretinal deposits demonstrating a noticeable bulge in the retinal pigment epithelium with choroidal thickening.

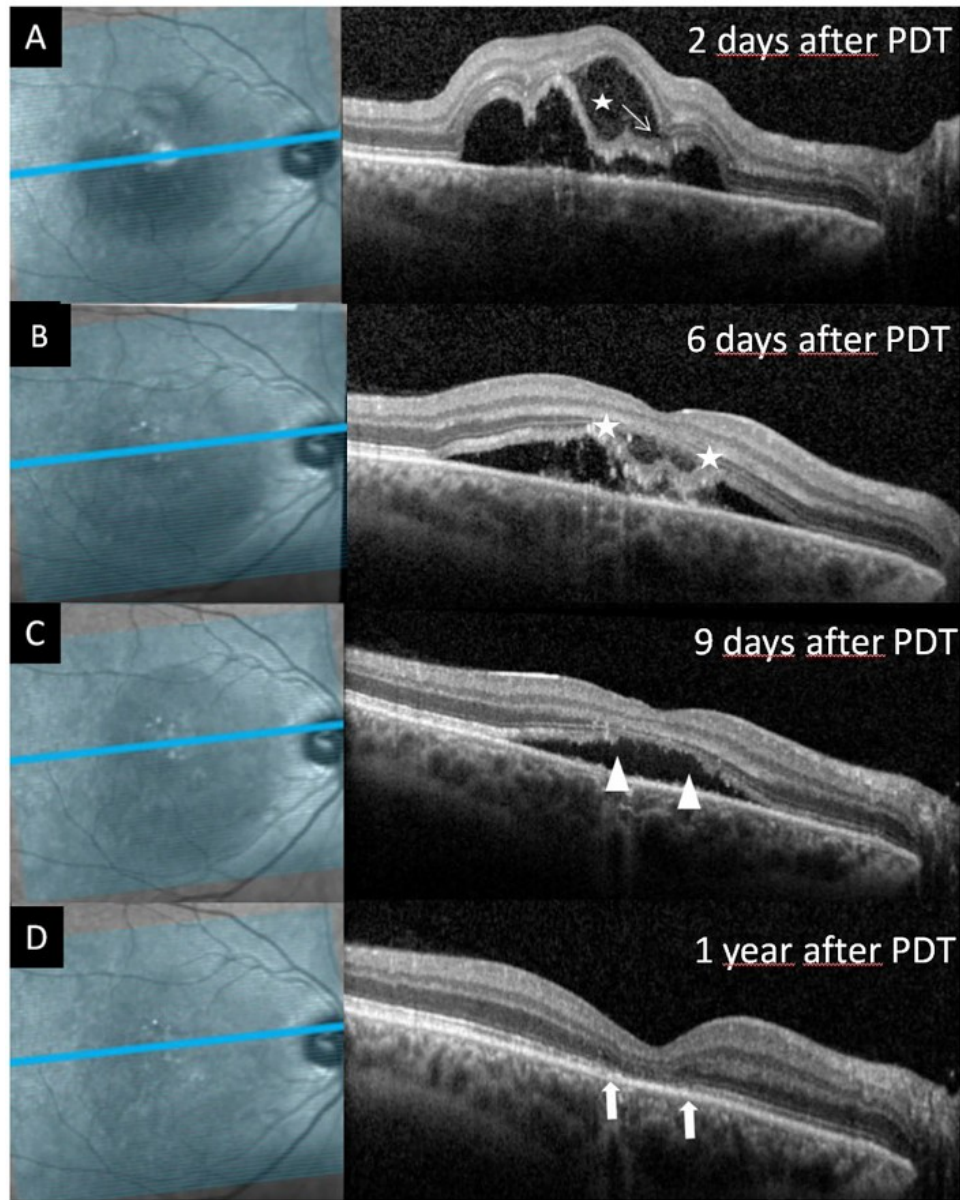


Fig. 2—Optical coherence tomography B-scan images after photodynamic therapy (PDT) in chronological order. (A) B-scan 2 days after PDT shows a significant increase in subretinal fluid (SRF). The outer retinal separation is demonstrated at the level of the hypore-reflective myoid zone (MZ) under the hyper-reflective external limiting membrane that separates the ellipsoid zone (EZ) from inner cell bodies (arrow), defined as the bacilli layer detachment (BALAD). The hyper-reflectivity of the fluid inside the inner retinal cystoid space is greater than that of the subretinal fluid (asterisk). **(B)** B-scan 6 days after PDT shows a significant decrease in SRF and the fluid inside the BALAD. The outer nuclear layer (ONL) thinning is shown at the location of BALAD (between asterisk). **(C)** B-scan 9 days after PDT shows resolution of BALAD with persistent SRF. It also shows that the elongation of the outer segments of the photoreceptors is interrupted in the anatomical location of the previous BALAD, creating cavitation (between arrowheads). **(D)** B-scan 1 year after PDT shows complete resolution of the SRF with ONL and EZ atrophy.

fluorescent area on fluorescein angiography, and on indocya-nine green angiography, there was a punctate hyperfluorescent spot consistent with a CSCR diagnosis (Fig. 1).

He was treated with full-fluence photodynamic therapy (PDT) (50 J/cm^2) at 600 Mw/cm^2 intensity over 83 seconds. Three days after the treatment, he presented with significantly decreased VA in the treated eye (20/200). There was a marked increase in foveal SRF on SD-OCT. In addition, there was separation at the level of the hypo-reflective

myoid zone external to the hyper-reflective external limiting membrane, corresponding to a bacillary layer detachment (BALAD), starting at a superior edge of the fovea and extending superiorly. The SRF and the BALAD formation significantly improved within 4 days. SD-OCT image showed the outer nuclear layer thinning at the BALAD-affected area (Fig. 2B).

The photoreceptor's outer segments were interrupted in the anatomical location of the previous BALAD, creating

cavitation in the affected area (Fig. 2C). After the SRF had resolved, there was a loss of the ellipsoid zone (EZ) and outer nuclear layer thinning at the site of the previous BALAD that extended into the foveal centre. One year after the PDT, there was partial EZ restoration and improvement of the VA to 20/80 but not to the pre-PDT baseline of 20/60.

Photodynamic therapy–induced acute exudative maculopathy (PAEM) is a short-term complication of PDT described as a transient massive subretinal exudation after PDT, with or without acute vision loss. Histopathologic studies show damage to the choriocapillaris endothelium, resulting in a critical inflammatory response, causing a sudden increase in the permeability of the choroidal vessels and retinal pigment epithelium pump dysfunction, resulting in exacerbated fluid accumulation.¹ PAEM usually presents within a week after PDT, correlating well with the previously described histopathologic changes during the first week after PDT. In addition, hyperacute choroidal exudation in the subretinal space causes a sudden increase in hydrostatic pressure, which exceeds the tensile strength in the outer retina, resulting in the separation at the myoid level called BALAD as an OCT imaging finding may be accompanied by PAEM.^{2,3} In our case, PAEM, accompanied by BALAD formation, developed on the third day post-PDT.

Fernández-Vigo et al.³ performed a prospective study on patients treated with PDT and found that the incidence of PAEM was 30.4%. BALAD developed in 46% of these eyes, and SRF completely resolved in 57.1% at 1 month.³ As shown in imaging studies, vascular remodelling is complete within 4 weeks after the PDT, which is also the period for the PAEM resolution reported in the studies and case reports in the literature.¹ We observed in our patient, similar to the other studies, BALAD formation resolved within 1 week before the SRF, which resolved a month after the PDT treatment.³ The studies have a consensus that BALAD resolves faster with an EZ disruption.⁴ Similar to those outcomes, we observed an EZ atrophy on SD-OCT after the fluid resolution, especially and only anatomically present at the BALAD location. We observed that photoreceptor elongation was interrupted in the BALAD area, creating cavitation on SD-OCT images during the period when BALAD was resolved entirely and SRF persisted (Fig. 2C). This was perhaps a precursor to the outer retinal atrophy observed at the exact location after the SRF resolution. Ramtohl et al.⁵ observed progressive restoration of the EZ with a persistent focal attenuation in all eyes with BALAD at a long-term follow-up. After a 1-year follow-up, our patient showed minimal restoration of foveal EZ (corresponding BALAD location) and partial vision improvement. Although the anatomical results of the reports related to BALAD are similar, there are differences between the functional outcomes, and the

effect of BALAD on the vision outcomes in the accompanying disease has not yet been clarified.

Fernández-Vigo et al.³ observed that there was no significant change in BCVA at 3 months when compared with the BALAD subgroup of those with PAEM but without BALAD. In our case, the patient's vision was decreased with an increase in exudation, and after the resolution of the SRF, VA increased but did not return to the baseline. This vision loss and EZ atrophy outcomes can be attributed to either PAEM, BALAD, or chronic CSCR. As in this case, the fact that BALAD accompanies a wide variety of complex pathologies makes it challenging to determine the effect of BALAD on functional outcomes.

In conclusion, PDT settings (duration and fluency) might contribute to acute fluid accumulation and accompanied BALAD formation. These acute changes may resolve rapidly with the onset of the vascular remodelling process, leaving a local EZ disruption in the area, which can cause a decrease in VA.

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