Retinal phototoxicity after macular hole surgery induced by xenon light: a case series

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Retinal phototoxicity caused by light from endoilluminator used in vitrectomy has been described in the scientific literature. Xenon light can be one of the most harmful sources of radiation to the retina if adequate filters are not used as that radiation is near ultraviolet spectrum.

We present herein a case series of patients who developed retinal phototoxicity induced by xenon light, after macular hole surgery.

Macular phototoxic damage with foveal involvement remains a factual danger during vitrectomy using Xenon light as endoilluminator with inadequate filters.

Key words: retinal phototoxicity, xenon light, vitrectomy.

One of the endoilluminators used during vitrectomy is Xenon light. It has been recognized as causative source of retinal phototoxicity, with a few cases described in the literature.

Case Reports

Case 1
A 71 year-old Caucasian male underwent 20-gauge three-port pars plana vitrectomy (PPV) with internal limiting membrane (ILM) peeling and fluid-gas exchange (C3F8) due to a macular hole (MH) of 450 microns of diameter (Figure 1a). A complete ocular examination was performed prior and after surgery, including best corrected visual acuity.

Abstract
Retinal phototoxicity caused by light from endoilluminator used in vitrectomy has been described in the scientific literature. Xenon light can be one of the most harmful sources of radiation to the retina if adequate filters are not used as that radiation is near ultraviolet spectrum.

We present herein a case series of patients who developed retinal phototoxicity induced by xenon light, after macular hole surgery.

Macular phototoxic damage with foveal involvement remains a factual danger during vitrectomy using Xenon light as endoilluminator with inadequate filters.

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(BCVA), slit lamp biomicroscopy, fundus photography, optical coherence tomography (Carl Zeiss, Germany), using spectral domain software (Cirrus®). Fluorescent angiography (FA) was performed only after surgery.

The previous BCVA was 0.1. During surgery, we used a Xenon light released by Alcon called AHBI, which was put on top of the Accurus, as the Accurus only has a halogen light source. In order to facilitate ILM peeling, Brilliant Blue G (BBG) was used. The peeling of ILM lasted 20 minutes, and the total length of the surgery was about 45 minutes. Follow-up examinations were conducted for different periods: each four months for one year, each six months during the second year and annually during the next years.

One week after surgery the macular hole was closed and remained stable during follow-up. One month later, BCVA was lower than before surgery and we observed changes in Retinal Pigment Epithelium (RPE) at the posterior pole (mottled hypo and hyper pigmented retinal alterations) (Figure 2a).

Four months postoperatively, retinal atrophic areas combined with pigment dispersion in the macular area were noted. The FA showed blockage of the fluorescence in hyper-pigmented areas and staining of the lesions in RPE atrophic regions (Figure 3a). The characteristics of these lesions and surgical conditions implicated the endoilluminator as the source of photic injury.

At 48 months postoperatively, BCVA was hand motion at one meter, and funduscopic examination showed no changes (Figure 4a). OCT revealed closure of the macular hole with retinal thinning and loss of the inner/outer photoreceptor layer and RPE (Figure 5a).

**Case 2**

64 years old Caucasian women underwent 23-gauge three-port pars plana vitrectomy (PPV) with internal limiting membrane (ILM) peeling and fluid-gas exchange (C3F8) due to macular hole (MH) with an opening diameter of approximately 500 microns (Figure 1b). Similar complete ocular examination was

![Figures 2a, 2b, 2c. Fundus photography one month after vitrectomy in cases 1 (2a), 2 (2b) and 3 (2c), showing round hyper and hypopigmented lesions in macular region with compromise of foveal area. The extension of the damage is similar in the three patients except of patient 3, where we can appreciate extension outside the vascular arcades.](image)

![Figures 3a, 3b, 3c. Fluorescent angiography performed after light exposure in cases 1 (3a), 2 (3b) and 3 (3c). We observed a combination of hyper and hypoflourescence related to atrophic and hyper pigmented areas, respectively. FA revealed a slightly bigger extension of the lesions in comparison with funduscopic examination.](image)
performed prior and after surgery as in case 1.

Prior vitreoretinal surgery BCVA was 0.1. During surgery, we used the same light source as in Case 1. The peeling of ILM (assisted by BBG) lasted about 10 minutes, and the length of surgery was about 40 minutes. The distance of the light pipe was the same as in the first case, and similar to other vitreoretinal surgeries.

Ten days after surgery, the macular hole was closed and remained unchanged during follow-up period. Two months later, BCVA was lower than before surgery and we observed severe alterations in RPE at the posterior pole (Figure 2b), similar to Case 1. The changes increased substantially over the following months and were confirmed by FA (Figure 3b).

The follow-up period for this patient was 44 months. BCVA at last examination was Hand motion at one meter, and the funduscopy appearance was similar to that observed at one month postoperatively, with an increase in extension of atrophic regions involving macular area (Figure 4b). OCT revealed a disruption of the complex ERP-photoreceptors (Figure 5b).

Case 3

A 68-year old Caucasian woman underwent 23-gauge pars plana vitrectomy due to a macular hole (Figure 1c). BCVA prior surgery was 0.05. The surgical procedure was the same as reported before, with the exception of the use of Indocyanine Green (0.05%) as dye for ILM peeling. The length of the ILM peeling was 7 minutes and the total duration of the surgery 35 minutes.

One week after surgery, the macular hole was closed and remained closed during all follow-up period. However, one month after vitrectomy, funduscopy revealed the presence of severe ERP alterations at the posterior pole, showing a combination of atrophic areas with areas of pigment dispersion (Figure 2c). FA results are displayed in Figure 3c. In the last visit (follow-up period of 50 months), BCVA was hand motion at one meter; posterior biomicroscopy revealed a bigger extension of the damaged areas (Figure 4c) and OCT showed an intense disruption of photoreceptor layer (Figure 5c).

Discussion

Macular phototoxicity caused by fiber optic endoillumination during pars plana vitrectomy is well known. Some authors postulate that commercially available light sources for endoillumination during vitrectomy are not safe with respect to photochemical retinal damage as the emitted light is potentially...
harmful to the retina. In particular, blue and ultraviolet light are harmful, because the lens is a good absorber of these radiations and, consequently, the irradiance threshold for retinal damage is relatively high.6

Most of the radiation produced by Xenon light is near ultraviolet spectrum and this is why it can result more phototoxic than other sources of light.7

Funduscopic findings in Xenon light phototoxicity include paramacular lesions with severe alterations of RPE. However, the damage induced by this light, usually respect the fovea center, with slight repercussion on visual acuity.7

This protective effect by the fovea is probably related with the xanthophyll pigment, which is present in inner and outer plexiform layer and it is able to absorb blue and ultraviolet light, a way to protect the RPE and neurosensorial retina.3

Contrary to those reports, our case series showed intense involvement of foveal tissue with pigmentary mottling at the level of the retinal pigment epithelium.

As corneal shielding had eliminated operative microscope illumination and two different types of dyes had been used during vitrectomy in the three cases, endoillumination seemed to be the main source of injury. It is interesting to emphasize that in case 3, the use of indocyanine green as dye, may have also contributed to the development of the disease.

Probably the lack of use of preventive measures as adequate filters, the length of the surgical procedure and the high intensity of the light power led to the disease severity.

One point we need to emphasize, as previously reported, is the special danger of the light source used in our series due to the fact that it has a 420 nanometer filter rather than a 435 nanometer filter that most of the other new light sources use. However, the working distance of the light pipe was the same we had used for more than five hundred vitrectomies with different light sources and different dyes without any adverse event like this.

Conclusion

The case series describe macular phototoxicity with foveal involvement after utilization of a type of Xenon light source manufactured by Alcon (Alcon Laboratories, Inc., Fort Worth) for the Accurus vitrectomy system called AHBI (Accurus High Brightness Illuminator).

Factors probably associated with this disease are mainly: light power, exposure time (lengthy surgeries), endoilluminator-retina distance, and fundamentally, the type of filter incorporated in this light source, which seems to be insufficient to protect retinal tissue.

Vitreoretinal surgeons must learn to act over these factors to avoid the development of retinal alterations. It could be reasonable to use appropriate filters, as the safety of a light source is a function of its spectral curve and, fundamentally, of filtering. Xenon light may be not inherently dangerous, but it can be made dangerous if inadequate filters are used, as happened in our cases.8-10

REFERENCES