ABSTRACT

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REFERENCES

1. The XENON LIGHT we use to perform vitrectomy MAY CAUSE PHOTOTOXICITY (1).

INDEPENDENTLY OF THE USE OF DIFFERENT DYES.

2. Moreover, FACTORS probably ASSOCIATED with the appearance of these lesions are, mainly: LIGHT POWER, EXPOSURE TIME, and ENDOILLUMINATOR-RETINA DISTANCE, and we must learn to act over these factors to avoid the development of retinal alterations (2,3).

3. It could be reasonable TO USE APPROPRIATE FILTERS, OR EVEN AVOID XENON LIGHT, in order TO PREVENT RETINAL DAMAGE, specially IN COMPLICATED CLINICAL CASES or LONG TIME SURGERIES.

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ABSTRACT

Advantages: To report three cases of retinal phototoxicity caused by Xenon light during idiopathic macular hole surgery.

Methods: Three eyes (3 patients), two females and one male with idiopathic macular hole, underwent three-port pars plana vitrectomy (Accurus, Alcon, Fort Worth, TX, EUA) with internal limiting membrane peeling and fluid-gas exchange (C3F8) due to the presence of an idiopathic macular hole of more than 500 microns of diameter. In all cases we used a Xenon light during surgery with a power of intensity ranging from 50 to 75%. Full clinical examination, with determined Best Corrected Visual Acuity (BCVA), fundus examination, retinography and Optical Coherence Tomography (OCT), were performed prior and after surgery. Fluorescein angiography (FA) was also performed in all cases after surgery.

Effectiveness/safety: One week after surgery the macular hole was closed in all cases and it 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 hyperpigmented retinal alterations) which were much more intense in the following months. Four months postoperatively, retinal atrophic areas combined with pigment dispersion in the macular area were appreciated. The FA showed blockage of the fluorescence in hyperpigmented areas and staining of the lesions in RPE atrophic regions. The characteristics of these lesions and surgical conditions implicated the endolaminator as the source of photic injury. OCT performed postoperatively revealed closure of the macular hole with retinal thinning and loss of the inner/outer photoreceptor layer and RPE in the three patients. The lesions remained unchanged at six months, one, two, three and five years later in all cases with a final BCVA lower than 10/100 in all eyes at their last visit.

In conclusion: macular phototoxic damage with foveal involvement remains a factual danger during vitrectomy using Xenon light as endolaminator, even with maximum precautions.

METHODS

We retrospectively reviewed cases of three patients with retinal damage after vitrectomy with Xenon light. A complete ocular examination was performed prior and after surgery, including BCVA, slit lamp biomicroscopy, fundus photography, Optic Coherence Tomography (Carl Zeiss, Germany), using spectral domain (Cirrus) or time domain (Stratus) software. Fluorescein Angiography (FA) was only performed after surgery.

Follow-up examinations were conducted for different periods: every four months for one year, every six months during the second year, and annually during the following years.

RESULTS

We reviewed 3 eyes (2 left eyes-OS, 1 right eye-OO) of 3 patients (two 68 and 64 years-old females and one 71 years-old male). All of them had undergone three-port pars plana vitrectomy (PPV) with internal limiting membrane (ILM) peeling and fluid-gas exchange (C3F8), due to Macular Hole (MH) of more than 500 microns of diameter. The previous VA was 0.1 (decimal notation) in all patients. In all cases we used a new Xenon light during surgery with an intensity power ranging between 50 and 75%, and the surgical time (exposure time) was 40 minutes approximately. In order to facilitate ILM peeling, Indocyanine Green (ICG) (0.05%) was used in one case and Brilliant Blue G (BBG) in two cases.

ONE WEEK AFTER SURGERY: macular hole was closed in all cases and it remained stable during follow-up.

ONE MONTH LATER: BCVA was lower than before surgery and we observed changes in retinal pigment epithelium at the posterior pole (mottled hypo and hyperpigmented retinal alterations) which were much more intense in the following months.

FOUR MONTHS LATER: retinal atrophic areas combined with pigment dispersion in the macular area. The FA confirmed the diagnosis of retinal phototoxicity. Similar changes were observed at six months, one, two, three and four years later in all cases WITH A FINAL BCVA LOWER THAN 1/10 IN ALL EYES AT THEIR LAST VISIT.

CONCLUSIONS

1. The XENON LIGHT we use to perform vitrectomy MAY CAUSE PHOTOTOXICITY (1).

2. Moreover, FACTORS probably ASSOCIATED with the appearance of these lesions are, mainly: LIGHT POWER, EXPOSURE TIME, and ENDOILLUMINATOR-RETINA DISTANCE, and we must learn to act over these factors to avoid the development of retinal alterations (2,3).

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REFERENCES

