S Tiew and A Choudhary

Royal Liverpool University Hospital, Liverpool, UK
E-mail: stephtiew@gmail.com

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Sir,
Poppers maculopathy or retinopathy?

The term 'poppers' refers to exogenous volatile nitric oxide (NO) donors that have been widely abused for recreational purposes. Inhalation of poppers provides rapid-onset, short-acting euphoria and myorelaxation. Several reports described persistent visual loss after poppers consumption.[1,2]

In all presented patients, functional and morphologic damage was limited to the fovea.[1,2] We report two patients who not only developed a characteristic maculopathy following poppers consumption but also additionally showed a bilateral decline in full-field electroretinography (ERG). A 45-year old and a 33-year old male patient presented with a bilateral visual loss (0.63/0.4 and 0.5/0.4). Funduscopic examination revealed bilateral, foveal yellowish dots. Figure 1 shows a pathologic full-field ERG. Electrooculography gave normal Arden ratios. Spectral domain optical coherence tomography (SD-OCT) showed central hyperreflective alterations in the photoreceptor layer and an interruption of the ellipzoid zone. The former patient reported only sporadic consumption. The latter regularly consumed poppers over 1 year and stopped at the diagnosis notification. Interestingly, 6 month follow-up of the latter showed a restitution of foveal architecture in SD-OCT; however, pathologic changes in ERG persisted.

High concentrations of NO are suspected to represent the underlying cause of retinal damage linked to poppers intake.[2] NO regulates photoreceptor metabolism in rods and cones primarily through activation of guanylyl cyclase, a key enzyme of phototransduction.[3] Experimental studies showed that higher doses of NO induce apoptotic cell death in photoreceptors.[4]

Our electrophysiological data suggest that retinal toxicity of poppers is not restricted to central cone photoreceptors only, as suggested by previous reports, but that damage may well extend peripherally also affecting rod photoreceptors. It remains elusive whether the degree of retinal damage and the involvement of rods depend on the highest level of local NO concentration or on frequency, subtype, or amount of poppers intake.

Albeit morphologic similarities in SD-OCT, a previously suggested light-induced damage seems less plausible given our findings in full-field ERG.[5] Other than photopic maculopathy, differential diagnosis includes adult onset vitelliform dystrophy and occult macular dystrophy.

Our report suggests a change in nomenclature from poppers maculopathy to poppers retinopathy, emphasizes the harmful potential of poppers consumption and stresses the need for a higher level of awareness.

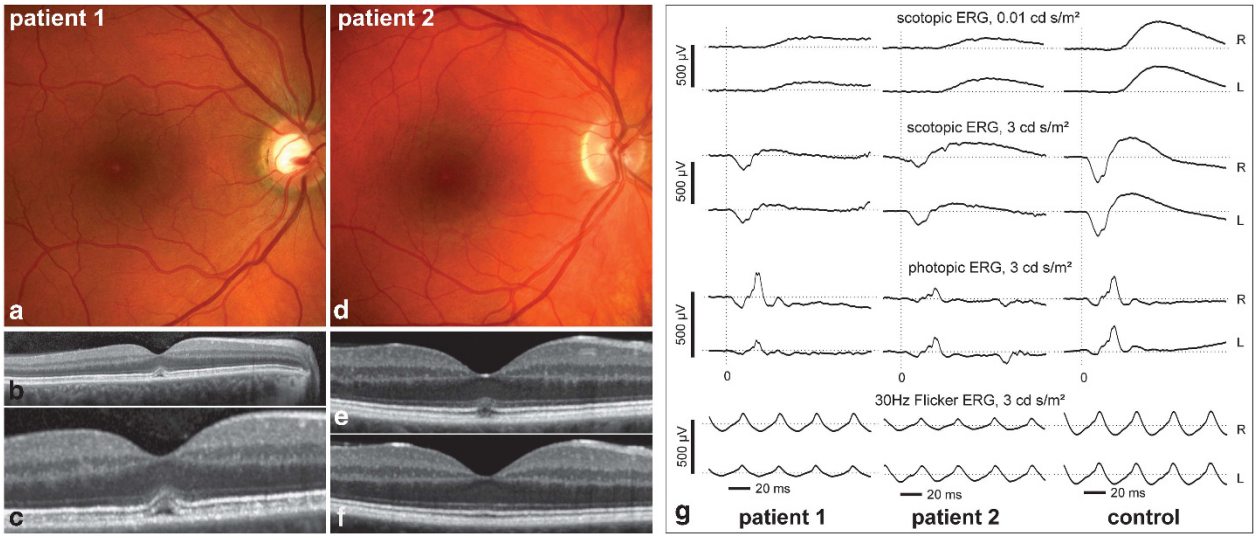

Figure 1 Morphologic and electrophysiologic data of the described patients. (a–c) Colour fundus photography (CFP) and spectral domain optical coherence tomography (SD-OCT) of patient 1 show the characteristic changes in the fovea. Panel (c) shows a magnified view of (b). (d–f) CFP and SD-OCT of patient 2 including follow-up SD-OCT scan after 6 months (f). (g) Comparison of full-field ERG measurements of both patients and an age-matched healthy control subject. A clear impairment of rod and cone function is visible by reduced scotopic a-wave and b-wave amplitudes as well as reduced photopic b-waves amplitudes. Moreover, photopic 30 Hz Flicker ERG is nearby the lower border of normative values.

Conflict of interest

The authors declare no conflict of interest.

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CR Clemens, F Alten, D Loos, CE Uhlig, P Heiduschka and N Eter

Department of Ophthalmology, University of Muenster Medical Centre, Muenster, Germany
E-mail: Christoph.Clemens@ukmuenster.de

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Sir,
Comment on 'The effect of erythropoietin on the severity of retinopathy of prematurity'

We read with interest the article 'The effect of erythropoietin on the severity of retinopathy of prematurity'.[1] Even if the results are interesting, we have a few concerns and comments. The use of sedatives to screen all the babies for ROP is surprising and can be risky for these vulnerable infants.[2] The babies with stage 3 ROP were treated when they reached threshold stage despite ETROP recommendation[3] to treat babies, at high risk, prethreshold, and beyond. This questions the efficiency of the treatment strategy followed in the study. The images of the infants needing treatment were reviewed by another examiner before treatment probably implies that the findings of the screening physicians required reconfirmation. In such a case, babies labelled with severe ROP after erythropoietin injection should also have been reconfirmed by a retina specialist. Although the indication to give erythropoietin was under the discretion of the paediatrician, some details like baseline haemoglobin concentration and platelet count at the time of erythropoietin injection would have been more informative as they are known to affect the severity of ROP independent of erythropoietin. Reports say that effect of erythropoietin on ROP depends whether it was given at early or late post-natal life.[4] So a similar division in the study could have given extra information.

The cumulative dose of erythropoietin received by neonate with mild ROP (3200 units) was more than those with severe disease (2750 units). To establish erythropoietin as a contributory cause, it is important for the cause to not only have statistical significance but also alter the effect on altering the cause with a proven dose–response relationship, which the study fails to show.

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RR Modi and TR Padhi

L V Prasad Eye Institute, Bhubaneswar, India
E-mail: tapas@lvpei.org

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Sir,
Comment on 'Photocoagulation guided by wide-field fundus autofluorescence in eyes with asteroid hyalosis': single and double pass of light in the ocular media

I enjoyed reading the case report from Ogino *et al*.[1] I agree that examining and treating patients with concurrent asteroid hyalosis and proliferative retinopathy is a significant challenge. I also agree that autofluorescence images and fluorescein angiography are much less affected by the presence of the vitreous opacities than fundoscopy and colour images.

The given reason that the wavelengths of light used to obtain these images are less affected by the asteroid bodies is however incorrect.

During biomicroscopy or colour photography light passes through the ocular media, reflects from the RPE/choroid, and exits through the ocular media into the imaging system. This is known as a double pass and is a multiplication not addition. The image quality is reduced by 'media-squared' not 'media-doubled'.

In FFA and AF the light exiting the eye has its origin entirely in the posterior layers of the globe. The incident light is absorbed and molecule-bound electrons are raised into higher energy levels. The electrons make a transition to an intermediate energy level and new light