these symptoms and signs after a child has spent time alone may signal participation in self-inflicted strangulation.[10]

In a child with features concerning for strangulation injury, it is important to question the child directly and in a non-confrontational manner, asking about self-infliction or infliction by others. The patient should be interviewed in private, as is typically done when asking pediatric patients about other sensitive subjects such as use of drugs and alcohol or sexual activity. With parents present in the room, patients might be less forthcoming to avoid parental discipline. Similarly the inquiring physician must normalize the nature of the conversation with the child to make the child feel comfortable disclosing the behavior to the physician and not feel as though he or she is being judged for the behavior. In spite of these measures by the physician, the patient may deny or not disclose strangulation—if clinical suspicion remains, strangulation injury should not be prematurely excluded.

Conjunctival/periorbital petechiae from a single strangulation episode will spontaneously resolve and should be observed. However, other secondary injuries from strangulation may require emergent evaluation in an emergency room or urgent care context; management of these injuries is beyond the scope of this report. The cause of strangulation must also be addressed. If participation in recreational self-inflicted strangulation is identified, this behavior requires management by the child's pediatrician and likely referral to a pediatric psychologist. Ongoing participation in the behavior carries high risk for the patient because recreational self-inflicted strangulation may result in accidental death and hypoxic brain damage.[6,7] Moreover, this high-risk recreational activity may be a marker for coexisting substance use or psychiatric illness.[11] If the strangulation is found to be a suicide attempt or concerning features such as suicidal ideation and intent are identified, emergent psychiatric evaluation is necessary. If strangulation is found to have occurred from child abuse, legal involvement including child protective services is necessary as dictated by local and federal protocols of the region.

This case highlights the importance of obtaining a careful social and psychiatric history in pediatric patients presenting with acute, bilateral conjunctival petechiae. Conjunctival and facial petechiae should alert the clinician to the possibility of an assault with severe strangulation but may also serve as a marker for self-inflicted asphyxiation with either self-harm or euphoric intent.

Aaron R. Kaufman, MD,* Daniel J. Oh, MD,*,† Ariel Gliksberg, MD,‡ Mary Lou Schmidt, MD,‡ Mehmet C. Mocan, MD*

*Illinois Eye and Ear Infirmary, Department of Ophthalmology and Visual Sciences, University of Illinois at Chicago, Chicago, IL; †Edward S. Harkness Eye Institute, Columbia University Irving Medical Center, New York-Presbyterian Hospital, New York, NY; ‡Department of Pediatrics, University of Illinois at Chicago, Chicago, IL.

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Correspondence to:
Mehmet C. Mocan, MD.; cmocan@uic.edu.

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Footnotes and Disclosure

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

Multimodal imaging of sclerochoroidal calcification associated with choroidal neovascular membrane

Sclerochoroidal calcification is a benign condition typically diagnosed in asymptomatic, older, white adults, which can simulate other more serious conditions such as choroidal melanoma, metastasis, or lymphoma.[1] It is characterized as yellow or yellow-white single or multifocal

lesions typically found in the superotemporal quadrant near the vascular arcades, which can be unilateral or bilateral.[1] Although sclerochoroidal calcification is most often idiopathic, systemic testing at the time of diagnosis is required to exclude associated disorders of calcium-phosphorus metabolism.[1]

Sclerochoroidal calcification has rarely been associated with choroidal neovascularization (NV) with variable clinical courses documented in the literature thus far.[1–4] Herein, we report a case of sclerochoroidal calcification and associated choroidal neovascular membrane (CNVM) characterized by multimodal imaging, including optical coherence tomography angiography (OCTA).

An 87-year-old white man with a 15-year history of stable, bilateral idiopathic sclerochoroidal calcification and antiphospholipid antibody syndrome on chronic anticoagulation was referred to the retina service for evaluation of a subretinal hemorrhage adjacent to sclerochoroidal calcification in the right eye. Head computed tomography (CT) scan at the time of initial diagnosis in 2004 demonstrated bilateral scleral hyperdensities consistent with calcification (Supplementary Fig. 1A [available online]); however, B-scan ultrasonography was not performed at initial diagnosis. He also underwent systemic evaluation to exclude disorders of calcium-phosphorus metabolism, which was negative. At the time of referral, the patient was asymptomatic. Best-corrected visual acuity was 20/25 in both eyes, and intraocular pressures were normal. Fundus examination of both eyes demonstrated several, ill-defined yellow choroidal lesions with areas of discrete calcification and overlying Retinal pigment epithelium (RPE) atrophy along the superotemporal arcade. The right eye demonstrated a subretinal hemorrhage along the superior border of the sclerochoroidal calcification (Fig. 1A). The left eye was notable for a stable choroidal nevus along the superonasal arcade with overlying drusen (Fig. 1B). Fundus autofluorescence of both eyes revealed hyperautofluorescence of the sclerochoroidal calcification, hypoautofluorescence in adjacent areas of RPE atrophy, and blockage owing to the hemorrhage in the right eye (Supplementary Fig. 1B, C [available online]). Fluorescein angiography of the right eye revealed hyperfluorescent late staining of the bilateral calcific lesions associated with blockage from the associated subretinal hemorrhage and a focal area of leakage suggestive of secondary choroidal NV (Fig. 1C, D). Enhanced depth imaging optical coherence tomography (EDI-OCT) ` of the right eye showed multiple pigment epithelial detachments (PEDs) associated with subretinal fluid and subretinal hyper-reflective material suggestive of an active CNVM (Fig. 2A). Other areas of sclerochoroidal calcification bilaterally demonstrated that the lesions arose from the sclera with overlying choroidal thinning (Fig. 2B, C). Choriocapillaris segmentation of en-face flow OCT angiography of the right eye demonstrated a shadow artefact from the PED and hemorrhage and a possible lacy vascular network of choroidal NV that co-localized with the area of leakage found on fluorescein angiography (Fig. 2D). Given the asymptomatic nature and peripheral location of the CNVM, close observation with follow-up every 3 months was advised.

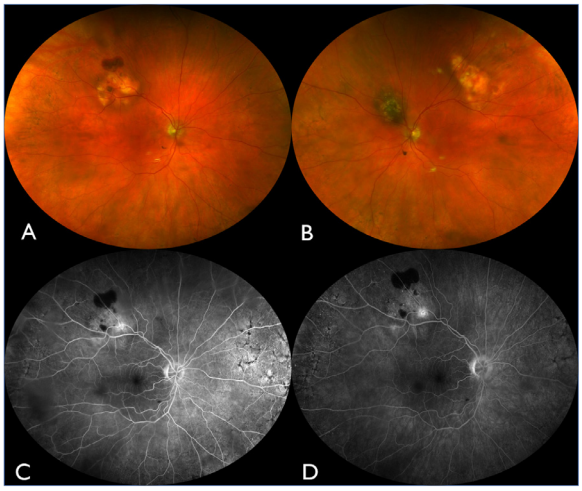

Fig. 1—An 87-year-old white man was referred for evaluation of a subretinal hemorrhage. Ultra-widefield pseudocolor fundus photograph of the (A) right and (B) left eye revealed several, bilateral, ill-defined yellow choroidal lesions with areas of discrete calcification and overlying RPE atrophy along the superotemporal vascular arcades, (A) a subretinal hemorrhage along the superior border of the sclerochoroidal calcification in the right eye, and (B) a stable choroidal nevus with overlying drusen along the superonasal arcade in the left eye. (C) Late venous phase fluorescein angiogram of the right eye demonstrated blockage superior to the sclerochoroidal calcification along with hyperfluorescence of other areas of sclerochoroidal calcification consistent with intrinsic staining. (D) Recirculation phase fluorescein angiogram revealed subtle leakage, difficult to distinguish from the characteristic late staining of sclerochoroidal calcification.

In the English literature, only 4 cases of CNVM associated with sclerochoroidal calcification have been documented with variability in clinical course.[1–4] In the most recent case published by Bessette and Singh, the patient required a number of treatments, including bevacizumab injections, photodynamic therapy, and argon laser photocoagulation, to achieve resolution of active leakage.[2] Two other cases were also successfully treated with argon laser photocoagulation.[3,4] A consecutive case series investigating the clinical manifestations and systemic associations of 27 patients with sclerochoroidal calcification by Honavar et al. reported one case of choroidal NV that remained stable without treatment through 1 year of observation.[1] Although sclerochoroidal calcification is most often idiopathic, systemic testing at the time of diagnosis is required to exclude associated disorders of calcium-phosphorus metabolism, such as Bartter syndrome, Gitelman syndrome, and primary hyperparathyroidism.[1] In the series by Honavar et al., 31% of patients were diagnosed with Gitelman syndrome, which carries a risk for serious systemic complications, including a higher risk of

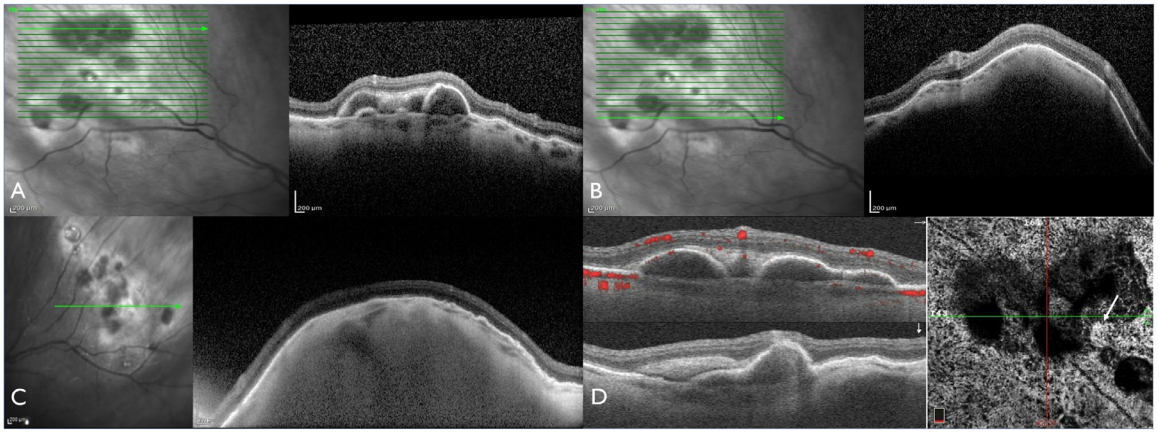

Fig. 2—Optical coherence tomography (OCT) and en-face flow optical coherence tomography angiography (OCTA) of an 87-year-old man with a history of sclerochoroidal calcification referred for evaluation of a subretinal hemorrhage. (A) OCT of the right eye showed multiple pigment epithelial detachments (PEDs) with subretinal fluid and subretinal hyper-reflective material suggestive of an active choroidal neovascular membrane (CNVM). (B, C) Other areas of sclerochoroidal calcification in both eyes demonstrated that the lesions arose from the sclera with overlying choroidal thinning. (D) Choriocapillaris segmentation of en-face flow OCTA of the right eye demonstrated a shadow artefact from the PED and hemorrhage and a possible lacy vascular network of CNVM (white arrow).

cardiovascular instability with anaesthesia.[1] In the case reported herein, the patient's laboratory testing demonstrated no underlying metabolic disorder, and considering his peripheral involvement and excellent visual acuity, he was closely observed to monitor for lesion enlargement or macula-threatening hemorrhage.

Sclerochoroidal calcification can be well characterized by multimodal imaging. On fundus autofluorescence imaging, the hyperautofluorescent nature of the lesions can presumably be explained by overlying choroidal thinning, resulting in the unmasking of underlying scleral hyperautofluorescence as well as the intrinsic hyperautofluorescence of calcific tissue within sclerochoroidal calcification.[1] This choroidal thinning can also explain hyperfluorescence with persistent late staining on fluorescein angiography, which can make subtle leakage from choroidal NV difficult to identify, as exemplified in this case.[1] Therefore, a multimodal imaging approach, including OCTA, may improve detection and monitoring of associated CNVM via direct visualization of the neovascular network arising from the choriocapillaris.

A study by Inoue et al. demonstrated a sensitivity of 85.7% for detecting type 1 choroidal NV with OCTA and structural OCT versus a sensitivity of 66.7% for fluorescein angiography alone, suggesting the importance of a multimodal imaging strategy to improve detection.[5] Not only can OCTA improve detection of associated CNVM, but OCTA might also help elucidate the cause of RPE atrophy commonly associated with sclerochoroidal calcification. Previous studies evaluating choroidal thickness in sclerochoroidal calcification have demonstrated a reduction in overlying choroidal thickness with subsequent outer retinal layer disturbances.[1] Future series of sclerochoroidal calcification with OCTA could provide additional pathophysiologic information regarding the evolution of retinal and RPE atrophy associated with these lesions.

In summary, we presented a case of sclerochoroidal calcification and associated CNVM documented with multimodal imaging, demonstrating that OCTA can be a valuable test to detect CNVM that is masked by late staining of sclerochoroidal calcification on fluorescein angiography. OCTA should be further explored as a tool to better understand the pathophysiology of sclerochoroidal calcification and associated changes in the retina, RPE, and choriocapillaris.

Supplementary Materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.jcjo.2020.11.007.

Blake H. Fortes, Lauren A. Dalvin, Sophie J. Bakri
Mayo Clinic Department of Ophthalmology, Rochester, MN.

Correspondence to:
Sophie J. Bakri, MD: bakri.sophie@mayo.edu.

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Footnotes and Disclosure

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Waxing and waning poppers maculopathy

Poppers are volatile aromatic liquids typically available in vials. They make a popping sound upon opening. Poppers are a member of the alkyl nitrites, chemicals with nitric oxide donor characteristics. Poppers have been a popular recreational drug for years owing to their transient euphoric, myorelaxant, and aphrodisiac effects presenting just seconds after inhalation.[1,2] Although they are illegal to sell as such, they are easily obtainable, and are often sold as air fresheners online or in nightclubs.[2] In recent years, the use of poppers has been associated with maculopathy that might cause fluctuating vision, scotoma, photophobia, metamorphopsia, and phosphenes.[2,3] The symptoms are usually bilateral and reflect morphologic changes in the macula. A yellow foveal spot can be seen on fundus examination, which corresponds to a subtle disruption of the foveal ellipsoid zone on spectral-domain optical coherence tomography (SD-OCT) images.[2–4] Visual improvement and resolution of the morphologic changes were described in some cases after cessation of popper use. We are reporting a case of a reversible popper maculopathy with a 2.5-year follow-up.

Case Report

In March 2018, a 50-year-old man presented with a 6-month history of central, glaring, slowly enlarging bilateral scotoma, which appeared 3 months after starting to use poppers regularly several times per week. His medical history included arterial hypertension, asthma, thalassemia, and narcolepsy; past ocular history was negative. At presentation, Snellen best-corrected visual acuity was 20/20 both eyes, on fundoscopy bilateral pale yellowish dots were seen in the fovea, the rest of the eye examination showed no abnormalities. The microperimetry (MP1, Nidek Technologies, Padua, Italy), colour vision, and fluorescein angiography were unremarkable. Infrared reflectance imaging showed dark foveal spots in both eyes (Fig. 1). Fundus autofluorescence imaging revealed very subtle foveal changes in both eyes (Fig. 1). SD-OCT (HRA + OCT Spectralis, Heidelberg Engineering, Heidelberg, Germany) demonstrated bilateral focal hyperreflectivity and disruption of the foveal ellipsoid zone (Fig. 1). Multifocal electroretinogram (ERG) performed with RETIscan system (Roland Consult, Brandenburg an der Havel, Germany) showed a reduced central retinal function (Fig. 2). Scotopic and photopic ERG showed a bilateral prolonged photopic response, alongside with reduced amplitudes in the left eye. At that time, we suggested abstinence of popper use and the use of lutein supplements.

In November 2018, after 8 months without using poppers, the patient reported resolution of the symptoms. The eye examination and SD-OCT showed no abnormalities (Fig. 1).

In May 2019, the patient reported recurrence of glowing central bilateral scotoma after resuming popper usage several times per week. The SD-OCT demonstrated foveal changes similar to when the patient was first examined (Fig. 1). Furthermore, the ERG showed a reduced central retinal function, similar to the first presentation. In September 2019, the patient reported complete resolution of the symptoms again. Barely noticeable foveal changes were observed on SD-OCT. From December 2019 to August 2020, the patient had a fluctuating intensity of visual disturbances and similarly fluctuating SD-OCT changes.

In August 2020, the patient reported complete resolution of all symptoms. Eye examination and SD-OCT showed no abnormalities (Fig. 1). Multifocal ERG in the right eye was normal and borderline normal in the left eye (Fig. 2). The patient revealed that he continued to use poppers several times per week but was careful to use only those that did not induce visual symptoms. He had noticed visual impairment after using butyl nitrite, isobutyl nitrite, and isopropyl nitrite. The use of amyl nitrite or pentyl nitrite caused no symptoms.

Discussion

In recent years, poppers maculopathy has become increasingly recognized as a complication of poppers abuse.[3] The pathogenesis remains unclear; however, it is believed to be the result of the toxic effects of nitric oxide.[2,4,5] Our patient developed symptoms soon after using poppers for the first time. Diagnostics showed typical changes that entirely resolved after abstinence and reoccurred after using certain types of poppers. This is comparable to some other case reports.[4,5] To the best of our knowledge, we are the first to report a case where, surprisingly, the symptoms and function improved entirely after switching from butyl nitrite, isobutyl nitrite, or isopropyl nitrite to amyl nitrite or pentyl nitrite-based poppers, although the patient continued to use them several times per week. This is supported by a case series of Rewbury et al. where symptoms were linked to certain popper brands, and it was suggested that chemically different poppers differ in their toxic effect.[5] We speculate that the