To the best of our knowledge, this is the first report using confocal microperimetry registered with SD-OCT to evaluate retinal sensitivity after spontaneous regression of soft drusen. We found that retinal sensitivities were similar in areas both with and without soft drusen in a region following soft drusen regression with preserved outer retina morphology. In the Beaver Dam eye study, regression of large soft drusen occurred in 30% of cases over a 10-year follow-up and was accompanied by the appearance of more severe lesions including RPE depigmentation, geographic atrophy, and macular neovascularization in nearly 30% of these eyes.[6] In our experience, regression of soft drusen without subsequent geographic atrophy is more likely to occur in eyes with concomitant cuticular drusen than in those lacking this finding. We hypothesize that the presence of a thick basal laminar deposit in eyes with cuticular drusen provides structural support to the overlying and adjacent RPE and photoreceptors during the process of drusen resorption.

A limitation of our approach is that baseline retinal sensitivity was not established at the initial visit for a longitudinal assessment of retinal function in the area of drusen regression. Instead, adjacent areas without soft drusen were used for comparison. While we cannot be certain that those locations had comparable retinal sensitivities at baseline, previous normative MAIA microperimetry data sets have reported similar retinal sensitivities in those same regions.[7]

Our results agree with a previous psychophysical study in patients with soft drusen regression using fine matrix mapping.[8] The authors demonstrated no significant difference in local sensitivities between areas with drusen regression and unaffected neighbouring areas on colour fundus photography. They also observed that after drusen regression, retinal mean sensitivity was inferior to that of age-matched control individuals without AMD. Our work agrees with this observation. Compared with fine matrix mapping, microperimetry enables direct measurements of focal retinal sensitivity that is precisely mapped to an eye-tracked near-infrared fundus image. Registration of SD-OCT and MAIA microperimetry eye tracked to near infrared shows preserved outer retinal morphology correlating with preserved visual function.

While soft drusen are well recognized as a major ocular risk factor for progression to advanced AMD, their presence may not always signal irreversible loss of retinal function, thus supporting the potential benefit of targeting the disease at this stage.

**Diogo Cabral,[*] Meera S. Ramakrishnan,[*,†] K. Bailey Freund[*,†]**

*Vitreous Retina Macula Consultants, New York, NY; [†]Department of Ophthalmology, New York University Grossman School of Medicine, New York, NY.

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Correspondence to K. Bailey Freund, MD; kbfreund@gmail.com

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## Footnotes and Disclosure

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## Ischemic cranial nerve 6 palsy and lipemia retinalis in a patient with hypertriglyceridemia


Severe hypertriglyceridemia can occur as a primary (genetic) disorder or secondary to diabetes mellitus, hypothyroidism, alcohol use, or medications. Ophthalmic signs of hypertriglyceridemia include xanthomas, xanthelasmas, and corneal arcus.[1] However, atypical presentations also can occur. Prior case reports describe presenting symptoms of intermittent vision loss due to associated hypertensive retinopathy or blurry vision from secondary central serous chorioretinopathy or branch retinal vein

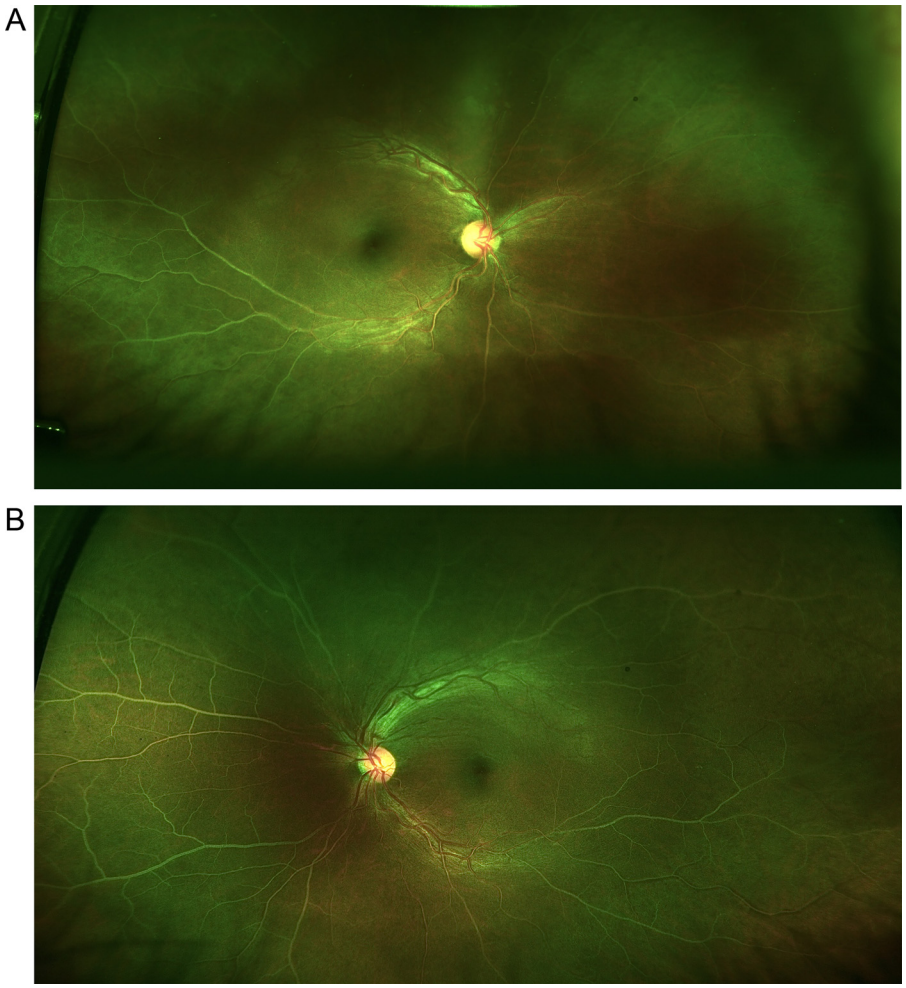


**Fig. 1—Ultra-wide-field fundus photography of the right (A) and left (B) eyes at presentation.**

occlusion.[2–4] In this report we describe a unique presentation of an isolated cranial nerve 6 palsy and lipemia retinalis in a young patient with severe hypertriglyceridemia. The case aims to inform clinicians to consider an urgent systemic evaluation for life-threatening hypertriglyceridemia when faced with this presentation.

A 43-year-old male with no known medical history presented to our clinic with 4 days of binocular horizontal double vision. He denied any other vision changes, recent trauma, headache, neck pain, tinnitus, fatigue, fevers, chills, weight changes, joint pain, or discomfort with chewing. His uncorrected visual acuity was 20/40 OU, and intraocular pressures were 10 mm Hg OU. He had no relative afferent pupillary defect, and his confrontation visual fields were full OU. Ocular motility testing revealed a −2 abduction deficit OD and full ductions in the other 8 cardinal directions. He had full extraocular movements OS. On strabismus examination, the patient had no manifest deviation in primary gaze, and the deviation was incomitant. He exhibited approximately 30 PD of esotropia in left gaze, left and upgaze, and left and downgaze. Slit-lamp examination was unremarkable OU. A dilated fundus examination revealed white and milky retinal vessels in both eyes (Fig. 1). He had no disc edema. He was afebrile and had no other cranial nerve or focal neurologic deficits on physical examination. A diagnosis of a right-sided cranial nerve 6 palsy and lipemia retinalis OU was made.

This patient was referred to the emergency department, where magnetic resonance imaging and angiography of the brain and orbits were emergently obtained and showed no acute infarct, no vascular stenotic disease, no masses, and an incidental aneurysm of the right anterior cerebral artery. Blood work revealed a total cholesterol level of 22.68 mmol/L (normal, <5.2 mmol/L), a triglyceride level of 61.73 mmol/L (normal, <1.69 mmol/L), and an HbA1C of 12.5% (normal, <6.5%). The patient was ultimately started on gemfibrozil and metformin and was counselled regarding dietary and lifestyle modifications. At his follow-up visit 1 week later, his serum cholesterol and triglyceride levels had normalized, and a repeat dilated fundus examination demonstrated resolution of his lipemia retinalis findings (Fig. 2). His abduction deficit OD was stable at short-term follow-up with ophthalmology, but he was noted to have resolution of diplopia and full extraocular motility several months later with his primary care physician.

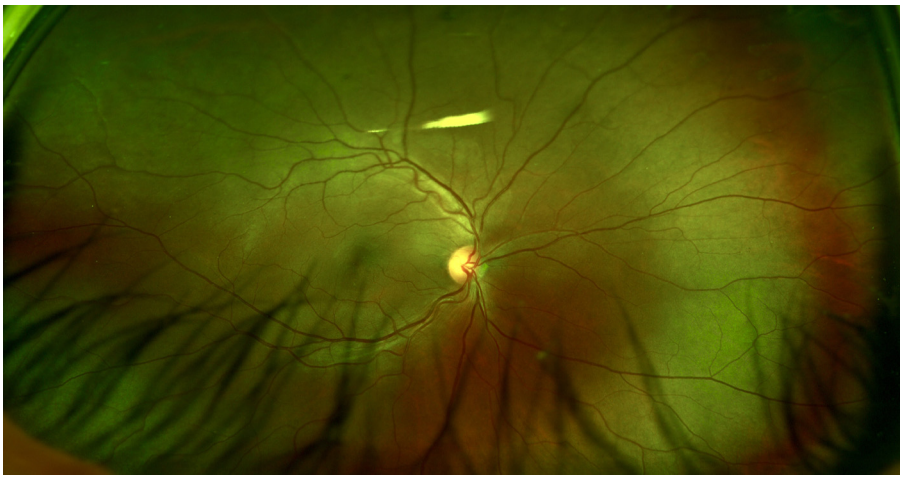

**Fig. 2—Ultra-wide-field fundus photography of the right eye after treatment.**

Lipemia retinalis is a rare manifestation of severe hypertriglyceridemia that develops due to light scatter by triglyceride-laden chylomicrons in the plasma when serum triglyceride levels exceed 16.94 mmol/L.[2] A staging system for lipemia retinalis was proposed by Vinger and Sachs: stage I (mild) is characterized by creamy vessels isolated to the periphery; stage II (moderate) is characterized by creamy vessels extending toward the optic disc; and stage III (marked) is characterized by all retinal vessels becoming cream coloured and the posterior pole becoming salmon coloured.[5] In most cases, lipemia retinalis is asymptomatic and is found incidentally on dilated fundus examination.

Lipemia retinalis itself is not visually significant. However, it is a critical ocular manifestation of a readily treatable life-threatening metabolic disorder associated with significant morbidity and mortality due to coronary artery disease, myocardial infarction, and stroke..[4] Patients should be referred for systemic management on an urgent basis, including a lipid panel. Treatment is aimed at reducing serum triglyceride levels to <5.65 mmol/L with pharmacotherapy.[4] Dietary and lifestyle modifications are also important for optimal control. The posterior-segment changes seen in lipemia retinalis typically improve once serum triglyceride levels have normalized.

In conclusion, this case describes an unusual presentation of hypertriglyceridemia and highlights the importance of a full dilated fundus examination when evaluating a patient with a cranial nerve palsy. A lipid panel is not part of the routine work-up of an abducens nerve palsy, so recognizing lipemia retinalis on fundus examination was critical in guiding management. Had the fundus findings not been recognized, a reasonable next step—after negative magnetic resonance imaging and considering his younger age—may have been a lumbar puncture, which would have subjected the patient to unnecessary procedures.

**Alice C. Jiang,[*,†] Rolake O. Alabi,[*,†] Zeeshan Haq,[*,†] Jay M. Stewart[*,†]**

*University of California, San Francisco, San Francisco, Calif.; [†]Zuckerberg San Francisco General Hospital and Trauma Center, San Francisco, Calif.

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Correspondence to Jay M. Stewart, MD; Jay.Stewart@ucsf.edu

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## Footnotes and Disclosure

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

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