Conflict of Interest Disclosures: Dr Fujimoto receives royalties from intellectual property owned by Massachusetts Institute of Technology and licensed to Carl Zeiss Meditech Inc, and dexamethasone suspension, 0.1%, both 4 times daily in her right eye. After 1 month, BCVA remained at 6/7.5 OD but worsened to 6/9 OS, corresponding to optical coherence tomographic findings of resolving ME in her right eye with progression in her left untreated eye (Figure 1, week 4). To exclude other causes of ME, fluorescein angiography was performed, demon-
strating late-phase leakage of dye in the left central macula (Figure 2). At this stage, topical ketorolac and dexamethasone were prescribed for both eyes twice daily with temporary improvement of BCVA to 6/7.5 OU after 3 weeks. Increased instillation frequency of topical anti-inflammatory eyedrops (initially 4 times daily, then every 2 hours) led to resolution of ME (Figure 1, week 23) with improvement of BCVA to 6/6 OU. Unfortunately, attempts at weaning topical anti-inflammatory drugs failed while the patient continued to use fingolimod. After consultation with her neurologist, a decision was made to cease fingolimod use, resulting in resolution of her ME 5 weeks later (Figure 1, week 35).

Comment. Fingolimod-associated ME is observed in up to 0.5% of patients with MS receiving fingolimod. The proposed pathophysiological mechanism behind FAME is loss of sphingosine 1–phosphate receptor 1 signaling in endothelial cells, subsequent downregulation of adhesion complexes, and enhanced vascular permeability.

In previous reports, FAME resolved after cessation of fingolimod use. Because our patient’s neurological symptoms were well controlled and she wished to continue treatment with fingolimod, we elected to manage her FAME with topical medications. Our patient was partially responsive to topical nonsteroidal anti-inflammatory drugs and glucocorticoids since attempts at dose reduction led to exacerbation of FAME. The mechanism behind this responsiveness is unclear, but we speculate that an inflammatory component may have contributed in this case. Topical nonsteroidal anti-inflammatory drugs have been successfully used to manage cystoid ME. More recently, Afshar et al successfully managed FAME with topical nonsteroidal anti-inflammatory drugs. Further clinical trials may assist in optimizing management of FAME without cessation of fingolimod use.

Although not present in our case, optic neuritis is a diagnostic consideration in patients with MS who have visual disturbance. This may be differentiated from ME by the presence of a relative afferent pupillary defect, dyschromatopsia, and ocular pain on eye movements. An other possibility is that the ME may be unrelated to fingolimod use. Microcystic ME, predominantly affecting the inner nuclear layer, is reported in 4.7% of patients with MS and is more common in eyes with a history of optic neuritis. We cannot exclude preexisting microcystic ME because our patient was receiving fingolimod prior to her first clinic attendance and she did not have pretreatment optical coherence tomographic investigations, even though ophthalmological evaluation is recommended by the manufacturer prior to commencement of fingolimod treatment. Additionally, the frequency of
of FAME in patients with preexisting microcystic ME is unknown as previous clinical trials used time-domain optical coherence tomography, which was not sufficiently sensitive to detect microcystic ME.8

In conclusion, our observations suggest that inflammation may contribute to the pathogenic pathway of FAME. Topical anti-inflammatory drugs may be useful in managing patients with FAME when cessation of fingolimod use is not desired. Our case also illustrates the importance of regular ophthalmological review of patients receiving fingolimod.

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COMMENTS AND OPINIONS

Validity of the Results of a Contact Lens Sensor?

We read with great interest the study by Mansouri et al titled “Continuous 24-Hour Monitoring of Intraocular Pressure Patterns With a Contact Lens Sensor: Safety, Tolerability, and Reproducibility in Patients With Glaucoma.”1

The authors describe their results about safety and tolerability of the contact lens sensor (CLS) Triggerfish (Sensimed AG) in 40 eyes of 40 patients either with glaucoma or suspected of having glaucoma. Safety and tolerability were good. We, too, could find the same satisfying results in our patients (so far 30 eyes, one of us [C.F.] 3 times).2

In addition, the authors studied the reproducibility of the measurements by repeating their examinations 1 week apart. The Pearson correlation showed an overall correlation of r = 0.59, determined as fair to good. We studied the reproducibility in only 5 subjects (young, healthy eyes; not for 24 hours, but for 2 hours) and found the same results.3

Besides safety, tolerability, and reproducibility, one very important quality is yet missing: the validity of the results gained by the CLS in young subjects as well as in elderly patients with and without glaucoma. Leonardi et al, the “inventors” of this CLS, (only) performed measurements in juvenile porcine eyes by inducing very short-acting spikes of intraocular pressure (IOP) (injection and ejection to ± 10 mm Hg within 50 seconds4 or +3 3 mm Hg within 5 seconds5) and reported excellent results.

We performed a similar experiment in 1 enucleated human eye but increased the IOP stepwise by 5 mm Hg using a pump for much longer periods (30 minutes). We could not obtain the expected stepwise profile by means of the CLS.6

No further experimental studies have been published on the validity of the results of the CLS so far. We therefore built a setup of 5 young subjects with healthy eyes in whom we measured the IOP in one eye with ap-