ocular corticosteroid injection may be from CSCR that is related to a periorbital route should be included in the suggested etiologic association between the features of CSCR and corticosteroid therapy and development or exacerbation of CSCR.

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Figure 2. A, Cross-sectional optical coherence tomography demonstrates a collection of fluid under the fovea, confirming the diagnosis of central serous chorioretinopathy after a subtenon injection of corticosteroid. B, Optical coherence tomography shows resolution of the fluid and return of the foveal depression.

A variety of contributing factors may account for the paucity of reports linking CSCR and the common procedure of periorbital corticosteroid injection. In eyes with uveitis, clinical features such as synchiae, pupil constriction, or media opacity severe enough to warrant periorbital corticosteroid administration may preclude visualization of distinct macular details. Subtle fluid from CSCR that is related to a periorbital corticosteroid injection may be interpreted as CME or may be overshadowed by features of coexisting CME. Visual symptoms from secondary CSCR may be attributed to the primary diagnosis of uveitis, and thus the fundus examination that is required to detect this entity may be omitted. Optical coherence tomographic imaging may be especially helpful in this scenario to differentiate between the features of CSCR and CME. It is unlikely that the topical corticosteroid drops played a role in the development of CSCR because of the relatively brief time of administration and the lack of posterior-segment penetration, especially in this patient with phakic eyes. In contrast, periorbital corticosteroids are injected directly behind the globe to induce a posterior pole effect.

Because almost every other route of corticosteroid administration, including local intra-articular injection, has been linked with CSCR thus far, it appears logical that the periorbital depot corticosteroid injection would be associated with development of this disorder.²,³ The periorbital route should be included in the suggested etiologic association between corticosteroid therapy and development or exacerbation of CSCR.

Acitretin-Associated Maculopathy

Isotretinoin and etretinate are synthetic retinoids commonly used to treat skin disorders. Ocular adverse effects have been reported after short- and long-term use of these drugs.¹,² with keratoconjunctivitis sicca¹ being the most common one. Nictalopia and decreased dark adaptation have been described in patients treated with isotretinoin² and etretinate.³ Furthermore, abnormalities in the electroretinogram (ERG), including reduced scotopic amplitudes³ and color vision," have also been reported.

The case of a 32-year-old man who noted decreased visual acuity (VA) after long-term use of acitretin (Neotigason), a metabolite of etretinate, is reported. Slitlamp biomicroscopy, fluorescein angiography (FFA), and electrophysiology findings are described.

Report of a Case. A 32-year-old white man was seen in the emergency department on July 26, 2002, reporting a 3-day history of blurred
vision in both eyes. His medical history was remarkable for previous hepatitis B infection and severe psoriasis for which he had been treated with acitretin, 30 mg/d, for the past year. His ocular and family histories were unremarkable.

On ophthalmic examination, VA was measured at 6/9 OU. Anterior segment and intraocular pressures were normal. On fundus examination, retinal pigment epithelium (RPE) abnormalities at the macula and cystoid macular edema were present (Figure 1). Diffuse early and late hyperfluorescence at the level of the RPE at the macula was observed on FFA (Figure 1). Late leakage of fluorescein in foveal cystic spaces was also noted (Figure 1). Retinal changes were suspected to be possibly related to the use of acitretin, and thus, this treatment was then discontinued. Therapy with slow-release oral acetazolamide (Diamox-SR), 250 mg twice a day, was also started.

Three days later, his symptoms of blurred vision had improved, although his VA remained 6/9 OU. The cystoid macular edema had completely resolved (Figure 2). Only hyperfluorescence from RPE was present (Figure 2). Treatment with oral acetazolamide was discontinued.

Electrophysiology, including full-field ERG and pattern ERG (PERG), was performed on December 12, 2002, and disclosed no abnormalities. At his last follow-up visit, March 31, 2003, his VA was 6/6 OU and no macular edema was present. The RPE abnormalities, although attenuated compared with baseline, were still observed on FFA.

**Comment.** The patient described herein had diffuse RPE changes at the macula and macular edema. To our knowledge, these retinal abnormalities have not been previously described in patients using oral retinoids. The cystoid macular edema observed at the initial examination resolved in only 3 days after discontinuation of acitretin therapy and initiation of oral acetazolamide treatment. Because etretinate has a prolonged half-life, it seems more likely that the resolution of the macular edema observed was the result of the treatment with acetazolamide rather than being related to the discontinuation of the retinoid therapy. It is also likely that the

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**Figure 1.** Color fundus photographs (A and B) and fluorescein angiograms (C and D) obtained at the initial examination. Cystoid macular edema was observed on slitlamp biomicroscopy in both eyes and demonstrated in late frames of the fluorescein angiogram. In addition, diffuse hyperfluorescence at the level of the retinal pigment epithelium and involving most of the macula was also noted.
macular edema was the result of RPE dysfunction, since the leakage observed on FFA appeared to come from RPE and responded well to oral acetazolamide therapy. Furthermore, diffuse RPE changes were present on FFA and no abnormalities on the retinal vessels were seen.

Evidence suggests that retinoids are directly involved in the formation and accumulation of lipofuscin in the RPE that, in its turn, could compromise RPE function. However, the mechanism by which acitretin could have caused the RPE changes observed in this patient remains uncertain. Although a dysfunction in the fluid-pumping mechanism of the RPE appeared to be present in this patient, the alteration of the RPE was not severe enough to compromise the supporting role of the RPE on photoreceptor cell function and, thus, to impair the PERG. This is, however, not surprising. Salzman et al found that 47% of patients with aphakic macular edema had a normal PERG. Furthermore, diffuse RPE abnormalities on fundus autofluorescence images have been detected in some patients with a normal PERG (N. L. and G. E. Holder, PhD, unpublished data, 1998).

Because symptoms, VA, and macular abnormalities appeared months after initiating acitretin therapy and have continued to improve since its discontinuation, it is possible that this drug may have played a role in their occurrence. However, this could be confirmed only if the above anatomical and functional changes reappear following reinitiation of the therapy. Because it is not yet clear whether these changes are totally reversible, treatment with acitretin has not been reestablished.

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