The OCT angiograms showed reduced CNV flow area and CNV vessel loss following treatment. Because retinal vessel density did not decrease, reduced areas of CNV flow likely represent therapeutic effect.

Optical coherence tomographic angiography can also be used to evaluate blood flow of the choriocapillaris. In healthy eyes, the choriocapillaris appears confluent on OCT angiograms. As a precursor to CNV, reduced choriocapillaris flow was noted. Choroidal ischemia has been proposed as a normal feature with OCT angiography. Further study is needed to determine whether reduced choriocapillaris flow is associated with chronic CSC and whether it may increase risk of CNV development.

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Outcomes of an Algorithmic Approach to Treating Mild Ocular Alkali Burns

Ocular alkali burns can cause corneal blindness. They typically occur in young males in accidents or assaults, and the most common agents include lye, ammonia, magnesium hydroxide, potassium hydroxide, and lime. Treatment indicated at the time of injury is irrigation to return the pH to normal, but the optimal care following pH normalization is less well defined and, to our knowledge, has not been studied in clinical trials. In 2013, we proposed a clinical algorithm for treatment of acute ocular alkali burns to standardize their treatment, specifically defining the use of topical corticosteroids, oral vitamin C, oral doxycycline, bandage contact lenses, and amniotic membrane (Figure). We now describe our initial results with this algorithm at the Massachusetts Eye and Ear Infirmary Emergency Department and show that the algorithm sped the time to restoration of best-corrected visual acuity following mild ocular alkali injuries.

Methods | On approval of the Human Studies Committee of the Massachusetts Eye and Ear Infirmary, we reviewed the electronic medical records of patients who presented to the Massachusetts Eye and Ear Infirmary Emergency Department within 2 years prior to and 1 year after institution of the algorithm (June 1, 2013). Informed consent was not required owing to the retrospective nature of the study. We included patients burned with a known alkali agent or, if the agent was unknown, those with an ocular surface pH greater than or equal to 7.4 at presentation or documented in the medical record from an outside emergency department. Cases were categorized a priori in 2 ways: (1) best-corrected visual acuity at final visit worse than 20/30, vs equal to or better than 20/30; and (2) time to best-corrected visual acuity of more than 2 weeks, vs equal to or less than 2 weeks. To avoid mixing paired data (2 eyes from the same patient) with unpaired data, the initially worse eye in bilaterally injured patients was chosen for analysis by the 2-tailed Fisher exact test.

Results | Our sample included 28 patients (35 eyes) and 15 patients (17 eyes) who matched the inclusion criteria during the 2 years prior to and 1 year after institution of the guideline, respectively. Patient demographic characteristics are summarized in the Table. All patients reported normal vision prior to injury. Not all burns prior to institution of the management protocol were graded by the examining physician, but all patients included after the protocol was instituted had grade 1 burns by Roper-Hall criteria.4 All patients before and after initiation of the guideline had their eyes irrigated immediately and were seen at our facility within several hours of injury. Treating alkali-burned eyes with the guideline showed an increased proportion of patients whose visual acuity...
recovered to 20/30 or better (relative risk = 0.54; 95% CI, 0.17-1.67; P = .29). Management with the new guideline was associated with a greater proportion of patients whose visual acuity recovered to 20/30 or better within 2 weeks or less (relative risk = 0.42; 95% CI, 0.20-0.87; P = .005). Assuming patients lost to follow-up did not have visual acuity recovery to 20/30 or better within 2 weeks of injury gave a relative risk of 0.45 (95% CI, 0.24-0.84; P = .001).

Discussion | Optimal treatment of ocular alkali burns in the emergency setting is critical to reduce visual morbidity. Various interventions have been shown in animal models and in limited human case series to play a potential role in treatment. However, given the lack of level 1 evidence for any interven-

Table. Patient Characteristics and Nature of Injury

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>2 y Prior to Guideline</th>
<th>1 y After Guideline</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients, No.</td>
<td>25</td>
<td>25</td>
</tr>
<tr>
<td>Eyes, No.</td>
<td>35</td>
<td>17</td>
</tr>
<tr>
<td>Male, No. (%)</td>
<td>17 (61)</td>
<td>11 (73)</td>
</tr>
<tr>
<td>Age, mean (range), y</td>
<td>44.9 (21-82)</td>
<td>39.1 (14-70)</td>
</tr>
<tr>
<td>Circumstance, No. (%)</td>
<td>Work related</td>
<td>12 (42.9)</td>
</tr>
<tr>
<td></td>
<td>Not work related</td>
<td>15 (53.6)</td>
</tr>
<tr>
<td></td>
<td>Assault</td>
<td>1 (3.6)</td>
</tr>
<tr>
<td></td>
<td>Lost to follow-up, No. of patients (No. of eyes)</td>
<td>8 (10)</td>
</tr>
</tbody>
</table>
tion, evidence-based guidelines for treatment of ocular alkali burns are lacking. Despite the weaknesses and limitations of our small retrospective study, the results suggest that application of an evidence-based clinical algorithm in the acute phase of injury can improve outcomes in mild alkali burns, potentially reducing injury-related costs and recovery time. A prospective randomized clinical trial is needed to properly evaluate the benefits of any treatment protocol.

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Author Contributions: Dr Chodosh had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: Al-Moujahed, Chodosh. Acquisition, analysis, or interpretation of data: Al-Moujahed, Chodosh. Drafting of the manuscript: Al-Moujahed, Chodosh. Critical revision of the manuscript for important intellectual content: Al-Moujahed, Chodosh. Statistical analysis: Al-Moujahed, Chodosh. Administrative, technical, or material support: Chodosh. Study supervision: Chodosh.

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Observation

Bilateral, Multiple, Episodic Retinal Vein Occlusions Associated With Common Variable Immunodeficiency

Common variable immunodeficiency (CVID) is a heterogeneous family of primary immunodeficiencies characterized by reduced serum levels of immunoglobulins with reduced immunity.\(^1\) Reported ocular involvement with CVID includes retinal vasculitis,\(^2\) placoid choroidopathy,\(^3\) and retinal pigmentary alterations.\(^4\) In one report, 3 patients with CVID had retinal vasculitis and macular edema.\(^5\) Autoimmune disorders are found in 20% of these patients,\(^5\) and an association with uveitis has been suggested.\(^6\) We describe a patient with CVID who developed bilateral, multiple, episodic retinal vein occlusions, with peripheral retinal neovascularization at the boundary of areas of retinal capillary nonperfusion treated with scatter photocoagulation, and macular edema treated with bevacizumab.

Report of a Case | A girl in her teens with a history of CVID characterized by low IgM levels, lymphadenopathy, recurrent ear and sinus infections in childhood, and no antibodies to mumps, rubella, rubeola, or pertussis despite immunization for these presented with decreased vision in the left eye. Ophthalmoscopy revealed intraretinal hemorrhages, macular edema, and peripheral nonperfusion in the left eye, consistent with a central retinal vein occlusion. She was treated with intravitreal bevacizumab (1.25 mg/0.05 mL), followed by panretinal photocoagulation. Results of an extensive hypercoagulability workup including factor V Leiden, prothrombin 20210, protein C, protein S, antithrombin, anticardiolipin, and beta-2 glycoprotein I antibodies and dilute Russell viper venom time test were negative.

She was referred for further evaluation. Visual acuity was 20/20 OU, with no cells in the anterior or posterior segment and no iris neovascularization. Ophthalmoscopy showed retinal venous tortuosity and intraretinal hemorrhages in both eyes, but no residual macular edema in the left eye. Fluorescein angiography showed peripheral retinal capillary nonperfusion but none in the posterior pole in the left eye (Figure 1). Ultrasonography revealed no optic nerve head drusen. In the absence of neovascularization or macular edema, observation was recommended.

Two months after initial presentation, the patient developed recurrent macular edema in the left eye and a new branch retinal vein occlusion superotemporal to the optic nerve in the right eye, with macular edema and additional areas of capillary nonperfusion temporal to the edema. She received bilateral, monthly injections of bevacizumab for 3 months for the macular edema (Figure 2A). In the next 8 months, treatments were extended to 6-week intervals, while visual acuity remained 20/20 OU (Figure 2B). During the next 4 months, no treatment was judged to be indicated.

At this time, 17 months after initial presentation, the patient had exacerbation of the superotemporal branch retinal vein occlusion in the right eye, with a new area of retinal nonperfusion temporal to the border of a previous area of nonperfusion (Figure 2C). Retinal neovascularization was noted temporal to the area of nonperfusion, so that scatter photocoagulation was placed to these nonperfused areas 24 months after initial presentation. From 26 to 41 months after initial presentation, visual acuity remained 20/32 OD, and 3 additional bevacizumab injections were given for macular edema in that eye (Figure 2D).

At 42 months after initial presentation, the patient developed a new branch retinal vein occlusion inferotemporal to the optic nerve (Figure 2E) with macular edema in the right eye. Bevacizumab injections were resumed at