Uveitis Exacerbation After Varicella-Zoster Vaccination in an Adult

Herpes zoster ophthalmicus (HZO) results from reactivation of latent varicella-zoster virus (VZV) in the ophthalmic division of the trigeminal ganglion. Anterior uveitis (AU) is a well-described complication of HZO. Since becoming available in 1995, the live VZV vaccine Zostavax (Merck and Co, Inc) has successfully decreased VZV reactivation. Consequently, the Centers for Disease Control and Prevention recommend that all appropriate adults aged 60 years and older receive Zostavax, and prior HZO is not a contraindication to vaccination. However, the term appropriate is not well defined in the context of prior ocular disease complications. Herein, we describe an adult patient with a history of HZO experiencing acutely exacerbated AU 3 weeks after receiving Zostavax.

Report of a Case. An 86-year-old white man initially had herpes zoster dermatopathy in the right V1 distribution, which was treated promptly with valacyclovir hydrochloride, 1g 3 times per day. Several days after onset of the dermatopathy, he had pain, redness, and photophobia in the right eye. On examination of the right eye, centrally grouped keratoprecipitates (KPs) (without corneal stromal or epithelial involvement), 2+ cells, and flare were found. The AU was treated with topical prednisolone acetate, 1%. The patient developed a chronic low-grade AU in the right eye; it was well controlled with topical prednisolone acetate, 1%. He gradually developed mild microcystic corneal edema in the right eye, with specular microscopic imaging failing to reveal either a recognizable endothelial mosaic or guttae. The endothelial count in the contralateral eye was normal despite prior uncomplicated cataract surgery in both eyes. The microcystic edema was attributed to endotheliitis, possibly at the onset of the VZV AU as there were no recurrences of KPs or more than minimal AU. Three years after the initial visit, he was weaned completely off prednisolone acetate, 1%, after several months without any evidence of active AU.

During the following 7 months, the patient was followed up without any treatment and there was no recurrence of AU or worsening corneal edema. Subsequently, the patient received the Zostavax vaccine on his own initiative at a pharmacy. Three weeks after vaccination, the patient had visual acuity of 20/200 OD, decreased from his baseline of 20/40 OD. Examination showed worsened corneal edema, centrally located KPs in a linear distribution, and 1+ cells and flare in the anterior chamber. The presence of corneal edema and KPs not localized in the Arlt triangle, normal intraocular pressure, and mild anterior chamber cells suggest recurrent endotheliitis. No corneal epithelial or inflammatory stromal involvement was observed. The patient was treated with valacyclovir hydrochloride, 1g 3 times per day for 7 days, and intensive topical prednisolone therapy, resulting in return to his baseline condition.

Comment. This is a case of an adult patient initially manifesting HZO AU and endotheliitis and then experiencing a uveitis exacerbation that was significant for worsening corneal edema attributed to AU and endotheliitis and temporally associated with Zostavax administration. Because the patient had not previously experienced an exacerbation with KPs and corneal decompensation as occurred after receiving the vaccine, this temporal association suggests that his exacerbation was due to the vaccine.

A single prior case report describes VZV ocular disease, specifically interstitial keratitis, in an adult patient 35 days after vaccination, and a few pediatric cases of uveitis after administration of a live attenuated VZV vaccine have also been reported. This case adds to the literature suggesting risk of ocular inflammation recurrence after VZV vaccination. While isolated case reports are insufficient to conclude that AU is a contraindication to VZV vaccination, we advise caution in vaccinating patients with a history of HZO.

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4. Lin P, Yoon MK, Chiu CS. Herpes zoster keratouveitis and inflammatory occu-
Anti–vascular endothelial growth factor (VEGF) therapy has revolutionized the treatment of neovascular age-related macular degeneration (AMD). Recent theoretical modeling suggests that monthly anti-VEGF therapy may dramatically decrease the incidence of blindness due to AMD in this country. We describe 2 cohorts of patients with incident neovascular AMD in the last decade, selected to have 1 cohort before and 1 after the advent of anti-VEGF therapy to explore whether there are data to support the theoretical model in a clinical practice setting.

Methods. In this retrospective cohort study, all cases of choroidal neovascularization secondary to AMD seen by 2 of us (N.M.B. and S.B.B.) in 2002 and 2008 were identified from record review and billing records. All eyes with incident neovascular AMD and at least 12 months of follow-up were included. Records were reviewed for baseline demographic variables, types of treatment received, and follow-up visual acuity (VA) at 24 (±6) months. For patients with follow-up less than 19 months, 2-year data were imputed with the last observation. The primary outcome measure was the prevalence of legal blindness (defined as VA of ≤20/200 OU) in each cohort at 2 years. The secondary outcome was the prevalence of mild or moderate visual impairment, defined as the development of VA less than 20/40 (mild) or less than 20/80 (moderate) in the better-seeing eye, at 2 years. Multivariable logistic regression analysis was performed, adjusting for age, gender, and baseline VA.

Results. Totals of 84 patients (91 eyes) in 2002 and 41 patients (43 eyes) in 2008 were identified with incident neovascular AMD and longer than 12 months of follow-up. Twenty-six of 91 eyes (29%) in the 2002 cohort and 3 of 43 eyes (7%) in the 2008 cohort had follow-up less than 19 months, for which 2-year data were imputed. Subsequent to 12 months following treatment initiation, most patients had stable vision. Two-year outcomes were analyzed with and without imputed data and no substantive differences were apparent. Baseline characteristics in the 2 cohorts appeared comparable with respect to mean age (77 years in 2002, 76 years in 2008), gender (39% women in both cohorts), and prevalence of neovascular AMD in the fellow eye (45% in both cohorts). Mean baseline VA in the study eye was slightly worse in 2002 (20/80; 95% CI, 20/50-20/125) than in 2008 (20/63; 95% CI, 20/32-20/80), for which results were adjusted using multivariable logistic regression in reported data. Forty of 41 patients (98%) in the 2008 cohort received anti-VEGF therapy. This therapy was not available for the 2002 group, who were managed using photodynamic therapy, laser photocoagulation, or observation. The Table shows the 2-year prevalence of legal blindness, mild and moderate visual impairment in both cohorts, the 2-year prevalence of visual impairment in the study eye, and the relative odds of outcomes adjusted for age, gender, and baseline VA in the study eye.

Comment. This study confirms that the prevalences of legal blindness and moderate visual impairment 2 years following the diagnosis of neovascular AMD have decreased substantially following the introduction of anti-VEGF therapy. These data correspond well to theoretical modeling data previously published. There are several limitations inherent to this retrospective cohort study, including the unknown effect of the exclusion of patients who did not have continued follow-up and use of a cohort from a tertiary academic-based center. Nonetheless, the results suggest that despite the increasing prevalence of neovascular AMD, the prevalence of blindness due to neovascular AMD is decreasing provided that the stabilization in VA between 12 and 24 months is maintained thereafter.

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