ABSTRACT
Uveitis, glaucoma, and cystoid macular edema frequently develop in patients with juvenile idiopathic arthritis. The authors describe a case of cystoid macular edema associated with juvenile idiopathic arthritis that had not responded to intravitreal triamcinolone acetonide and bevacizumab but improved significantly following intravitreal injection with a dexamethasone 0.7 mg implant. [J Pediatr Ophthalmol Strabismus 2014;51:e25-e28.]

INTRODUCTION
Anterior uveitis, open-angle glaucoma, and cystoid macular edema (CME) frequently develop in patients with juvenile idiopathic arthritis (JIA) during the clinical course of disease. Corticosteroids are potent anti-inflammatory agents that can counteract many of the pathological processes thought to play a role in the development of CME, while there is no need to consider the common side effect of cataract formation and glaucoma. Some authors have suggested that intravitreal dexamethasone implants should be considered first-line therapy in CME, although intravitreal triamcinolone, ranibizumab, and bevacizumab have also been shown to be effective in some, but not all, reports. Recently, a sustained-release dexamethasone implant (Ozurdex; Allergan, Irvine, CA) has become available for the treatment of patients suffering from CME due to retinal vein occlusion and noninfectious uveitis.

We describe a case of CME associated with JIA that had not responded to intravitreal injection with triamcinolone acetonide and bevacizumab but improved significantly following intravitreal injection with a dexamethasone 0.7 mg implant.

CASE REPORT
A 10-year-old girl was referred to our clinic on January 21, 2013, with a 6-month history of z and uveitis associated with JIA. She was treated with systemic methotrexate (15 mg/m²/week) and cyclosporin A for JIA, and topical timolol, brinsolamide, brimonidine, and oral acetazolamide for glaucoma. The best-corrected visual acuity (BCVA) was 20/40 in the right eye and 4/40 in the left eye. The slit-lamp examination showed posterior subcapsular cataract bilaterally. There were +2 cells in the anterior chamber of both eyes. Intraocular pressure (IOP) was 35 mm Hg in the right eye and 34 mm Hg in the left eye. Fundus examination showed advanced glaucomatous damage in both eyes.

Topical dexamethasone 0.1% eye drops were added to her medication for uveitis. However, the IOP increased to 45 mm Hg in the right eye and 44 mm Hg in the left eye within 2 weeks. Ahmed Glaucoma Valve (New World Medical, Rancho Cucamonga, CA) implantation was performed bilater-
ally to decrease the IOP (Figure 1). One month after Ahmed Glaucoma Valve implantation, the IOP was 44 mm Hg in the right eye and 16 mm Hg in the left eye. To decrease the IOP of the right eye, a secondary Ahmed Glaucoma Valve implantation was performed. After 1 week, the IOP was 17 and 15 mm Hg in the right and left eye, respectively (Figure 2).

After 4 weeks, the BCVA decreased to 6/20 in the right eye and 1/20 in the left eye. Optical coherence tomography (OCT) demonstrated CME and subretinal fluid with a central macular thickness of 620 and 615 µm in the right and left eye, respectively (the scans had signal strength of 3/10 in the right eye and 4/10 in the left eye due to cataract formation) (Figure 3). For CME treatment, topical dexamethasone 0.1% eye drops were started and 4 mg of triamcinolone acetonide in 0.1 mL intravitreal injection was performed bilaterally. No improvement was observed in macular edema and BCVA through 4 weeks of follow-up and 0.05 mL (1.25 mg) of bevacizumab intravitreal injection was performed. After 1 month, the CME persisted based on OCT. Following informed consent by the patient’s family, a dexamethasone intravitreal implant (Ozurdex) was then surgically implanted bilaterally. One month after the injection, the CME had subsided completely with a central macular thickness of 341 µm in the right eye and 327 µm in the left eye, although subretinal fluid and BCVA were not improved. At 6 months postoperatively, CME and subretinal fluid had subsided completely with a mean central macular thickness of 295 and 280 µm, respectively (Figure 4). However, the BCVA was still the same for both eyes due to cataract formation and glaucomatous optic nerve damage. During the 6-month follow-up period, no uveitis was detected. No increase in IOP measured with the Tono-Pen (Reichert, Depew, NY) was noted in both eyes.
DISCUSSION

Intravitreal steroids are indicated in uveitis to control chronic inflammation and persistent macular edema, and also to prevent the development of choroidal neovascularization. In a large randomized study of posterior uveitis, a fluocinolone acetonide implant (Retisert; Bausch & Lomb, Rochester, NY) was reported to reduce recurrence rate and improve vision. However, a high incidence of cataract and glaucoma filtration surgery (93% and 40%, respectively) was required in these eyes.

The dexamethasone drug delivery system marketed as Ozurdex is approved by the U.S. Food and Drug Administration and European Medicine Agency for macular edema caused by central and branch retinal vein occlusion and for noninfectious uveitis of the posterior segment of the eye. The HURON study indicated that Ozurdex has effectively reduced and controlled vitritis in intermediate or posterior uveitis 2 months after a single injection. Another retrospective case series suggested the Ozurdex implant improved vision and controlled inflammation in 14 eyes with pediatric uveitis, often sparing the need for oral corticosteroids. The current case had glaucoma associated with JIA; therefore, we applied an Ozurdex implant that was reported to cause a lower incidence of glaucoma when compared to the Retisert implant.

In previous studies, the Ozurdex implant was reported to be safe and effective in the treatment of non-infectious uveitis and CME. However, it is unknown whether the dexamethasone 0.7 mg implant could even effectively reduce CME associated with JIA refractory to previous intravitreal steroid and anti-vascular endothelial growth factor injections. In the current case, the dexamethasone 0.7 mg implant was able to successfully control inflammation and improve CME. One possible explanation for the inefficiency of previous intravitreal injections could be the short half-life in the vitreous cavity.

This problem might be overcome by the sustained-release characteristic of the implant.

Typically, the most common adverse events associated with intravitreal corticosteroids (which
may have affected use in the past) include increases in IOP and cataract formation. In the current case, no increase in the IOP and cataract density was observed during the 6-month follow-up period. However, the patient already had bilateral tube shunts in place at the time of injection; therefore, we cannot comment about the effect of the implant on IOP.

The dexamethasone implant (Ozurdex) might be an effective treatment option in CME associated with JIA. However, further prospective, controlled studies need to be performed to better determine efficacy, duration of effect, and side effects.

REFERENCES