roid in the implant led to recurrence of inflammation, and the presence of a foreign body may have incited the formation of vitreous bands to the implant. A perioperative vitreous hemorrhage was most likely the main contributor to band formation in patient 4. Because the explanted material was not sent for pathologic examination, we do not have information on the histological characteristics of the vitreous bands.

In conclusion, Retisert implant placement may lead to the formation of vitreous bands. When vitreous bands cause visually significant traction, patients may benefit from vitreoretinal surgery. In addition, the surgical procedure for implant removal or exchange in patients with clinically visible vitreous bands should be modified to include a complete vitrectomy in order to avoid retinal traction at the time of implant removal.

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Non–AIDS-Related Kaposi Sarcoma Involving the Tarsal Conjunctiva and Eyelid Margin

Kaposi sarcoma (KS) is a low-grade, multicentric vascular neoplasm that has been described in 4 clinical forms: classic, African, immunocompromised, and epidemic (AIDS-related) KS. Though ocular involvement by KS is rare, it is typically observed in the epidemic form and is most commonly seen as conjunctival or eyelid lesions in the setting of multicentric disease. We describe a patient with ocular KS without any of the risk factors previously described for the 4 clinical subtypes of KS.

Report of a Case. A 62-year-old Hispanic man was seen at the Long Beach Veterans Affairs Medical Center in California with a 12 × 10-mm hemorrhagic, telangiectatic, papillomatous lesion on his upper eyelid margin and tarsal conjunctiva, as well as an adjacent 3 × 4-mm broad-based lesion on the upper eyelid margin (Figure 1). The patient reported that the larger mass grew from a pinpoint, red lesion on his upper eyelid to its current size over the course of 1 month. He stated that the lesion occasionally bled and was irritating to the eye. His medical history was significant for hypertension and his ocular history included chronic open-angle glaucoma. Social history revealed occasional alcohol use and a remote history of smoking in his teenaged years. Based on the clinical appearance, a presumptive diagnosis of pyogenic granuloma was made, and the lesion was injected with intraleosional steroids (0.1 mL of triamcinolone acetonide [Kenalog] 40 mg/mL). On follow-up examination 3 weeks later, the pedunculated mass had grown to 15 × 13 mm, and an excisional biopsy was performed. The satellite lesion was not excised. The pathologic findings from the biopsy revealed KS with areas of inflammatory cellular infiltrate (Figure 2), and immunohistochemical stains revealed the presence of human herpesvirus 8 (HHV-8) (Figure 3).

The patient was subsequently treated with cryotherapy to the base of both lesions at the eyelid margin and tarsal conjunctiva. Following treatment, the satellite lesion re-
Kaposi sarcoma may also occur in the setting of exogenous immunosuppression, typically after solid-organ transplantation; KS lesions in these patients resemble those seen in the classic form of the disease, but the lesions may be distributed in other areas, including the visceral cavity.5-7 The epidemic of HIV-related KS may be the AIDS-defining diagnosis in up to 10% to 15% of patients. In Africa, this form of KS is commonly seen in heterosexual adults, while in developed countries such as the United States, afflicted patients are typically homosexual males.5 The epidemic form of KS is the most aggressive, frequently occurring in multicentric fashion with involvement of skin, mucosal membranes, lymph nodes, and the visceral cavity.

We report a rare case of ocular KS in a Hispanic patient without HIV or other forms of immunosuppression. Although it is possible that our patient has an underlying immune deficiency that has not been detected, his normal white blood cell count, negative serologic testing for HIV, lack of opportunistic infections, and negative review of systems suggest a competent immune status at the time we saw him. Ocular involvement by KS in a patient who is serologically negative for HIV is extremely rare. In 1994, Ron et al6 reported a case of conjunctival KS in an 83-year-old Jewish male patient of East European origin; this was presumed to be an atypical manifestation of classic KS as the patient later developed multifocal cutaneous lesions in the lower extremities. An association with non-AIDS KS and visceral lesions has been reported in the past as well. Kalinske and Leone10 described a patient with eyelid and conjunctival KS, who was found to have a gastrointestinal malignant neoplasm. However, the findings from a colonoscopy performed on our patient were normal. Our case is unique as our patient had localized ocular involvement without evidence of immunosuppression or the typical background or risk factors suggesting the classic or endemic forms of KS.

Through DNA analysis, HHV-8 has been identified as the causative, etiological agent for KS.11 Infection by HHV-8, which is also known as KS-associated herpesvirus, is thought to precede the development of the tumor in all clinical variants of KS. This virus has also been associated in 2 AIDS-related lymphoproliferative disorders: primary effusion lymphoma and the plasma-cell variant of multicentric Castleman disease12 as well as the vascular tumor recurrent angiolymphoid hyperplasia with eosinophilia.13

In summary, ocular KS should be suspected in patients demonstrating the clinical features of this vascular neoplasm without the previously described risk factors. Even in atypical cases, the diagnosis of KS can be confirmed by its distinctive histopathologic features and the demonstration of HHV-8 in the tissue specimen.
Neovascular Glaucoma From Advanced Coats Disease as the Initial Manifestation of Facioscapulohumeral Dystrophy in a 2-Year-Old Child

Facioscapulohumeral dystrophy (FSHD) is an autosomal dominant muscular dystrophy estimated to affect 1 in 20,000 white persons. The clinical features of this condition range from minimally detectable myopathy to severe disability. There is a characteristic pattern of weakness that affects predominantly the face (facio) and shoulder (scapulohumeral) muscles and later descends inferiorly to the abdomen and the legs.1,3 Symptoms become manifest in the teen years to early adulthood and progress slowly. Recent studies have shown a characteristic deletion in the long arm of chromosome 4 (4q35),2 but the exact mechanism of this disease remains unknown.

Classic Coats disease is a congenital, idiopathic retinal telangiectasia that can progress to severe retinal exudation and detachment.4 Retinal telangiectasia compatible with Coats disease can be an extraocular manifestation of FSHD, but most affected patients have asymptomatic retinal telangiectasia found at ocular screening after diagnosis of FSHD.5 The ocular findings rarely progress to advanced Coats disease.3 We describe a young child who had advanced eye findings of unilateral neovascular glaucoma from bilateral retinal telangiectasia 3 years before FSHD became apparent.

Report of a Case. A 23-month-old healthy girl had sudden onset of redness and pain in her right eye and was found to have leukokoria and neovascular glaucoma. At examination, the right eye could not fix or follow, and the left eye could fix and follow small objects. Intraocular pressure was 42 mm Hg OD and 18 mm Hg OS, and oral acetazolamide therapy was started. The right eye had diffuse neovascularization of the iris. Ophthalmoscopy showed total retinal detachment, aneurysmal dilatation of the retinal vessels, and subretinal exudation. The left eye showed shallow temporal retinal detachment with peripheral telangiectasia and mild retinal exudation (Figure 1). These findings were consistent with Coats disease in both eyes: stage 4 in the right eye and stage 3 in the left eye.4 Management included enucleation of the right eye and laser photoagulation and cryotherapy of the telangiectasia in the left eye (Figure 2). Within 6 months, the left eye was stable without exudation and the fovea remained intact.

At age 26 months, the child developed myoclonic seizures and atypical absence seizures. Subsequently, generalized hypotonia with protruding abdomen (Figure 3), flat facial appearance, open drooling mouth, and protuberant tongue became apparent. At age 30 months, hearing loss and speech delay were confirmed and hearing aids were placed. Genetic and metabolic evaluation revealed no specific syn-

Figure 1. A 2-year-old girl with neovascular glaucoma and leukokoria was found to have bilateral Coats disease. Enucleation of the glaucomatous right eye was performed. Three years later, facioscapulohumeral dystrophy was diagnosed. A. Facial appearance with leukokoria and painful neovascular glaucoma of the right eye. B. Right eye. Note total retinal detachment with subretinal exudation from Coats disease. C. Left eye. Fluorescein angiogram demonstrates peripheral telangiectasia.