and 40 and much brighter than fat, which has a HU of −100.[9] The higher attenuation of the wood in our case is likely from the wooden fragment accumulating fluid in the 4 days following the initial injury. Additionally, the use of lung (window width = 1465; level −498) and bone (window width = 3077; level, 570) window settings, which have been reportedly useful, did not improve visualization of the wooden foreign body. The wooden foreign body was best visualized with a window width of 155 and level of 42, which is a narrower window than is previously reported in the literature.[10]

Orbital abscess, as seen in our case, is frequently associated with wooden foreign bodies. The infection rates with wooden foreign bodies within the orbit can be as high as 64%, with worse complications in those with intracranial extension.[1] Antibiotics are of limited efficacy when a wooden foreign body is present; this is thought to be due to the development of a biofilm that impairs the effectiveness of antibiotics.[1] Additionally, the ability of wood to fragment into multiple pieces can make retrieval of the wood very difficult; one case describes a patient undergoing four medial orbital explorations without removal of the foreign body until there was spontaneous extrusion of small pieces of wood through the sinus.[11] Our patient has not developed any evidence of recurrent infection 4.5 months following his presentation.

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Late exudative retinopathy after laser treatment for retinopathy of prematurity in a child with dyskeratosis congenita

Giovanni H. Greaves, MD, Brian Savoie, MD, David Rhee, MD, and Sylvia Kodsi, MD

Exudative retinopathy may be a manifestation of a variety of isolated ocular or systemic diseases in children. We report the case of a teenager with dyskeratosis congenita who developed a unilateral late exudative retinopathy after having previous laser treatment for threshold retinopathy of prematurity as an infant.

Case Report

A 14-year-old girl, former premature infant, presented at the Northwell Health pediatric ophthalmology clinic for her routine yearly examination. She denied any changes in vision. She was born at 29 weeks' gestational age weighing 930 g and was delivered by Caesarian section for fetal distress and placental bleeding. She subsequently developed threshold retinopathy of prematurity (ROP) in both eyes at 40 weeks and was treated with laser ablation. After laser treatment, she developed temporal dragging of the macula, high myopia, and mild refractive amblyopia in both eyes. At 8 years of age, bone marrow failure led to the diagnosis of autosomal dominant dyskeratosis congenita. Both she and her mother were found to have a causative *TERT* gene mutation.

One year prior to presentation, the patient's visual acuity had been 20/30 in the left eye. On her current examination, best-corrected visual acuity was 20/30 in the right eye and 20/100 in the left eye. Her pupils were round and reactive, with no evidence of afferent pupillary defect. She was orthotropic at distance and near, with full versions. Slit-lamp examination was unremarkable for any anterior segment or lenticular pathology. On cycloplegic refraction she had unchanged high myopic astigmatism in both eyes, −16.0 +4.00 ×93 in the right eye and −16.50 +4.75 ×90 in the left eye. Dilated fundus examination revealed

Author affiliation: Department of Ophthalmology, Northwell Health, Zucker-Hofstra School of Medicine, Great Neck, New York
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Correspondence: Giovanni H. Greaves, MD, Northwell Health, Department of Ophthalmology, 600 Northern Boulevard, Suite 220, Great Neck, NY (email: gionon5@gmail.com).
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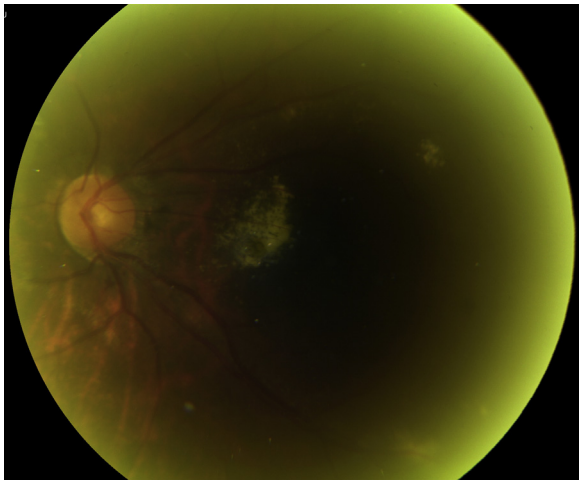

FIG 1. Fundus photography of the left eye showing temporal, superior, and nasal exudates.

temporal dragging of the macula in the right eye and, in the left eye, exudates nasal, temporal, and superior to the fovea (Figure 1). No clinically appreciable retinal tears, holes, or detachment were observed in the left eye. Fluorescein angiography in the left eye showed capillary nonperfusion superiorly in the periphery (Figure 2A), with midperipheral telangiectatic vessels and ischemia temporally (Figure 2B) in addition to the previous laser scars; late-phase angiography revealed leakage within and around the macula and areas of nonperfusion in the temporal macula (Figure 2C). Fluorescein angiography revealed only telangiectatic vessels and previous ablative therapy in the periphery of the right eye, best captured at her 1-month follow-up visit (eFigure 1).

The left eye was treated with additional photocoagulation to the ischemic and telangiectatic regions of the superior and temporal retina. Her best-corrected visual acuity initially improved to 20/50 in the left eye 1 month after laser treatment, with resolution of her exudates. At this visit, fluorescein angiography demonstrated further areas of nonperfusion superiorly and temporally. Subsequently she had another session of photocoagulation to treat these areas of nonperfusion. Within a month after this second laser treatment her visual acuity decreased to counting fingers at 6 inches, and on reexamination it was noted that she had a dense vitreous hemorrhage in the left eye without retinal detachment on B-scan ultrasonography.

After initial improvement of her vision, the vitreous hemorrhage worsened, and visual acuity in the left eye was reduced to hand motions. It was noted that because of the dyskeratosis congenita her platelet count had decreased to a nadir of 29,000. On consultation with hematology/oncology, the patient was started on danazol, with a corresponding rise in her platelet count to over 50,000; the patient's vitreous hemorrhage resolved over the course of 6 months to 1 year. Her best-corrected visual acuity was 20/40 in the left eye on her most recent eye examination, 2 years after the discovery of the exudative retinopathy.

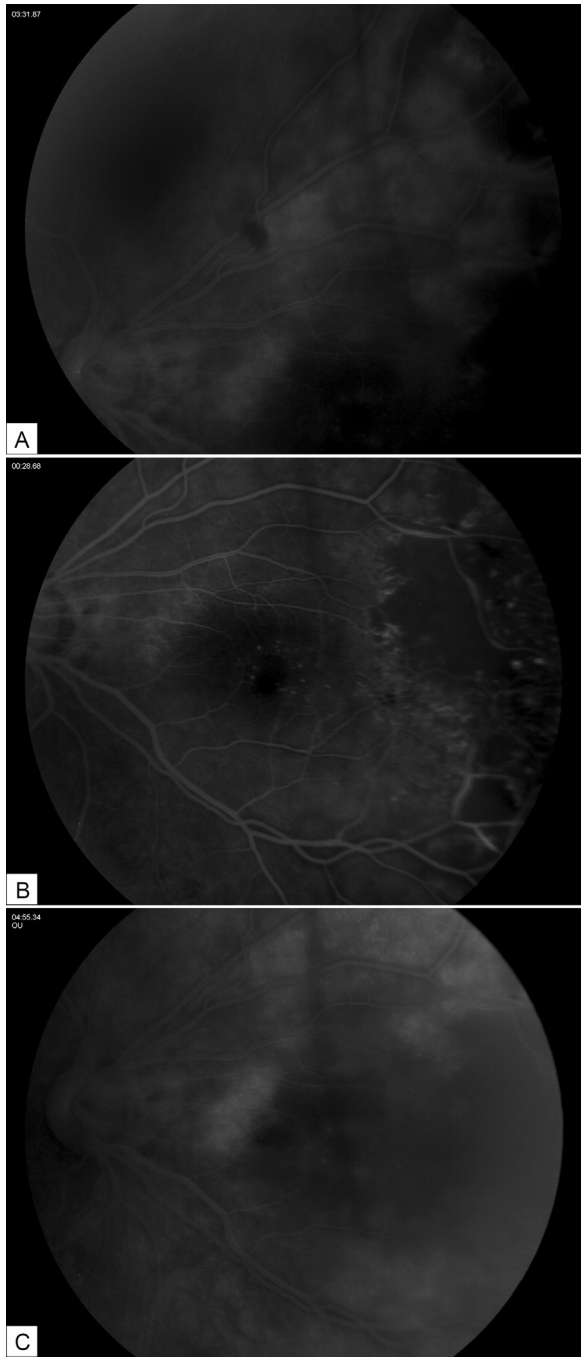

FIG 2. Late-phase fluorescein angiography (FA) of the left eye showing capillary nonperfusion in the periphery superiorly in the left eye (A), telengiectactic vessels and ischemia temporally (B), and leakage around the macula (C).

Discussion

Exudative retinopathy can be seen in children secondary to familial exudative vitreoretinopathy, ROP, Coats disease, inflammatory etiologies such as posterior scleritis or uveitis, and mass lesions such as a retinal capillary hemangioma or a choroidal melanoma or hemangioma. In addition, there are systemic diseases that cause defective telomere

maintenance with resulting short telomere that are also associated with exudative retinopathies, including dyskeratosis congenita, Revesz syndrome, Hoyeraal-Hreidarsson syndrome, and cerebroretinal microangiopathy with calcifications and cysts.[1,2] Dyskeratosis congenita is the only one of these with telomere abnormalities in which the patient has normal development and neurologic function; the others syndromes are often associated with neurologic abnormalities and developmental delays.[3]

Dsykeratosis congenita can be associated with many different mutations that can cause autosomal dominant, autosomal recessive, or X-linked inheritance.[3,4] Clinically the patient presents with the diagnostic triad of dysplastic fingernails and/or toenails, lacy reticular pigmentation of the skin, and oral leukoplakia.[5] There are multiple other manifestations of the disease, including dental, gastrointestinal, skeletal, neurological, genitourinary, pulmonary, and bone marrow aplasia.[5] Patients with dyskeratosis congenita are at increased risk for progressive bone marrow failure, myelodysplastic syndrome, acute myelogenous leukemia, solid tumors, and pulmonary fibrosis.[3] Ocular manifestations of dyskeratosis congenital include obstruction of the lacrimal drainage system, entropion, keratoconjunctivitis, and retina abnormalities.[5] Many retinal abnormalities have been described, including retinal vasculopathy with and without vitreous hemorrhage,[4,6] exudative vitreoretinopathy,[7] proliferative retinopathy,[8] retinal detachment,[5] Coats-like disease,[9] bilateral retinal hemorrhages,[10] retinal pigment epithelial changes, and retinal neovascularization.[5] Our patient had an exudative retinopathy complicated by vitreous hemorrhage, which could have been secondary to neovascularization or other retinal vasculopathies that were exacerbated by her low platelet count.

This case is unusual because the child already underwent extensive ablation of her peripheral retina to treat her ROP and still developed the exudative retinopathy described in dyskeratosis congenita. Diseases of the telomere such as dyskeratosis congenita should be considered when a child presents with peripheral ischemic or telangiectatic retinal disease.

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Fibrin glue–assisted excision of a large recurrent microphthalmic cyst

Ruth K. Jones, MBBCh, BSc, Zanna I. Currie, MBBS, FRCOphth, and Sachin M. Salvi, FRCS (Glasg), FRCOphth

Microphthalmic cysts are rare. Although small cysts can be left in situ to promote orbital expansion, large cysts require drainage or surgical excision. Complete surgical excision is notoriously difficult, and incomplete excision may result in cyst reformation. We describe a novel method of using fibrin glue to aid successful complete removal of a large recurrent microphthalmic cyst in a 6-year-old child who previously had multiple drainage and surgical attempts.

Case Report

A microphthalmic right eye was noted at birth in an otherwise healthy boy. Ocular ultrasound confirmed the diagnosis and that the eye had no visual potential. To

Author affiliation: Department of Ophthalmology, Royal Hallamshire Hospital, Sheffield, United Kingdom
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Correspondence: Dr. Ruth K. Jones, MBBCh, BSc, Department of Ophthalmology, Royal Hallamshire Hospital, Glossop Road, Sheffield, United Kingdom S10 2JF (email: ruth.jones@doctors.org.uk).
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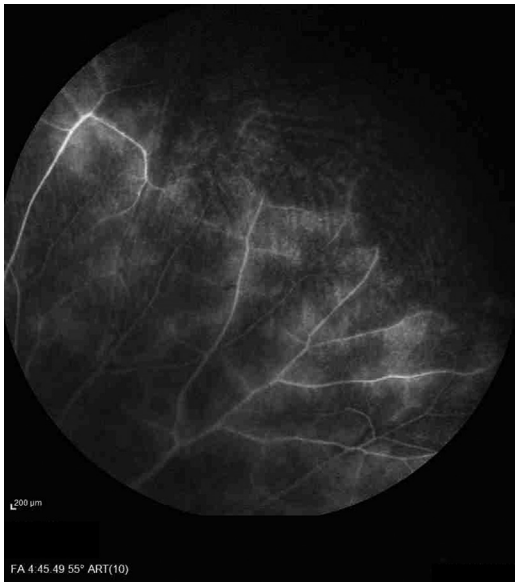

eFIG 1. Late-phase FA showing telangiectatic vessels in the periphery of the right eye.