the sensory retina, including the focus, was torn from the surrounding retina when a posterior vitreous detachment occurred. In addition to the high myopia and age of 53 years, vitreous inflammation may have facilitated the development of the posterior vitreous detachment. Ophthalmologists should be aware that, although rare, an MH can develop secondary to fungal endophthalmitis.

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Rosai-Dorfman Disease With Bilateral Serous Retinal Detachment

Rosai-Dorfman disease (RDD) is a rare idiopathic disorder affecting predominantly young adults.1 A bilateral massive painless enlargement of lymph nodes, predominantly in the cervical area, characterizes the disease. Additionally, several ophthalmic manifestations involving the eyelid, orbit, and lacrimal gland have been reported.1,2,3 The soft tissue surrounding the eye is infiltrated by a circumscribed rubbery, nontender mass, leading to variety of clinical signs and symptoms, eg, exophthalmos, blepharoptosis, conjunctivitis, keratitis, diplopia, dry eyes, and photophobia. The involvement of intraocular structures is less frequently described.1,2,3 We report a case of RDD with bilateral serous retinal detachments and its resolution after pharmacologic treatment.

Report of a Case. A 60-year-old man with bilateral enlarged inguinal lymph nodes underwent a biopsy and was diagnosed as having RDD (Figure 1B). Two years later, the patient noticed a decrease in his visual acuity. His visual acuity was 20/40 OD and 20/50 OS. On slit-lamp examination, anterior uveitis with cells in the anterior chamber was seen. Biomicroscopy demonstrated mild macular edema and some focal hyperpigmentations in the midperiphery of both fundi. Although the patient was treated with topical corticosteroids, his visual acuity decreased to 20/100 OU during the next 6 months. During this period, the patient developed massive bilateral serous retinal detachments with shifting fluids in the inferior quadrant; these detachments remained during the next 2 years. B-scan ultrasound demonstrated a retinal detachment with underlying sonolucent choroidal thickening (Figure 2A). Because of the long-term retinal detachment, we considered antimetabolic therapy, and treated the patient daily with 50 mg of prednisolone orally and 3 times per day with 2% cyclosporine eye drops for 10 weeks. During this time, the patient’s visual acuity increased to
20/50 OU. The choroidal thickening remained constant; however, the retina reattached completely, as seen on B-scan ultrasound (Figure 2B). Optical coherence tomography (OCT) showed a reattached retina with circumscribed hyperreflective areas in the retina and choroid (Figure 1A). Although the therapy was gradually stopped during the next 3 months, the anatomical and functional status remained stable.

Comment. Intraocular manifestations with uveitis are rare in patients with RDD. Foucar et al reported the microscopic findings from 13 enucleated eyes with RDD, and only 1 eye had extensive infiltration of predominant histiocytes in the entire uvea. Histologic examination of the eye demonstrated retinal detachment over approximately 1 mm of thickened choroid and a focal infiltration of histiocytes in the retina. We believe that our patient has the same intraocular condition.

The rare nature of the disease and the self-limiting prognosis reduces the knowledge about effective treatment. Treatment with only topical corticosteroids is not effective in reducing anterior uveitis. However, the retinal detachment in our patient responded quickly to treatment combining topical cyclosporine and systemic corticosteroids.

Our patient developed rare, vision-threatening intraocular infiltrates. These lesions led to back scattering of laser light during OCT and possibly to abnormally high signals on the false color image. The OCT image morphologically assessed the anatomy in vivo and confirmed intraretinal and choroidal infiltrates in a patient with RDD.

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Juan Rosai, MD, New York, NY, reviewed the subsequent cervical lymph node biopsy and confirmed the histopathologic diagnosis in this case.
Voriconazole Treatment of Fungal Scleritis and Epibulbar Abscess Resulting From Scleral Buckle Infection

Fungal infections, both ocular and systemic, can be difficult to treat. Challenges include the difficulty of diagnosis, possibly diminished host immune response, drug resistance of organisms, the difficulty of drug penetration, and the limited number of antifungal agents.

Voriconazole (Vfend; Pfizer Pharmaceuticals, New York, NY) is a new triazole antifungal agent; potent and wide-ranging activity has been demonstrated in vitro and in clinical studies.1-5 In May 2002, oral and intravenous formulations of voriconazole were approved by the US Food and Drug Administration for primary treatment of acute invasive aspergillosis and for salvage therapy for infections caused by *Scedosporium apiospermum* and *Fusarium* species. As with other azole compounds, the primary mode of action of voriconazole is the inhibition of cytochrome P-450–mediated 14-α-lanosterol demethylation, which is an essential step in fungal ergosterol biosynthesis.6

Based on a MEDLINE search, we believe that this is the first case report of refractory fungal scleritis with a nodular epibulbar abscess due to scleral buckle infection that was successfully treated with voriconazole, and the first report of intraocular concentration of voriconazole in a human following oral administration.

Report of a Case. A 65-year-old immunocompetent woman sought treatment for a hyperemic and tender left eye associated with an inferonasal epibulbar nodule. According to the patient, the nodule began “like a pimple” but gradually grew during the preceding 2 months. During the past year, the patient had undergone 3 procedures at another facility for recurrent retinal detachments, including pars plana vitrectomy and a scleral buckling procedure. She had been given subconjunctival methylprednisolone acetate at the conclusion of each operation, and postoperative topical prednisolone acetate was continued for 2 to 3 months each time. On examination, visual acuity was hand motions OS. She was aphakic, and the retina was attached, with areas of preretinal fibrosis and chorioretinal scarring. Areas of peripheral scleral thinning were noted. The nodule was only minimally mobile and slightly tender, but the inferonasal sclera was particularly tender and erythematous (Figure 1).

Surgical exploration revealed that the nodule was an abscess. The scleral buckle was initially not disturbed because of significant necrosis of the sclera and the risk of globe rupture. Cultures yielded *Aspergillus fumigatus*, and topical 0.15% amphotericin B and oral ketoconazole were initiated. After a lack of clinical response to a 4-week course of therapy, itraconazole was substituted for ketoconazole in hopes of improving intraocular penetration. A continued lack of clinical improvement led to uncomplicated scleral buckle removal 1 month later. During the next 4 months, despite multiple debridements and continued use of topical amphotericin B and oral itraconazole, the infection continued to spread counterclockwise around the eye (Figure 2). After learning of the investigational use of voriconazole, we obtained institutional review board approval of voriconazole use on a compassionate basis. Other antifungal agents were discontinued, and treatment with oral voriconazole, 200 mg twice a day, was begun. After 1 week of treatment, ocular tenderness and left-sided headache disappeared. Redness of the eye improved during the next 3 months (Figure 3).

During the second month of therapy, simultaneously acquired serum and intraocular fluid samples were assayed for trough concentra-

![Figure 1](https://example.com/figure1.jpg)

**Figure 1.** The patient's left eye at the initial examination shows an inferonasal epibulbar mass and hypertropia due to mass effect.