Varicella-Zoster Virus Retinitis Presenting as an Acute Vitreous Hemorrhage

Acquired varicella-zoster virus (VZV) retinitis produces 1 of 2 patterns of retinal involvement, each with its own characteristic clinical features. In the acute retinal necrosis syndrome, affected patients are typically healthy and are initially seen with vitritis, retinal vasculitis, and confluent necrotizing retinitis preferentially affecting the peripheral retina. In the progressive outer retinal necrosis syndrome, patients are immunodeficient and have multifocal areas of deep retinal opacification located in the peripheral retina with or without macular involvement. They usually have a clear vitreous, no hemorrhage, and an absence of vascular inflammation.

We recently treated a patient with the acquired immunodeficiency syndrome (AIDS) who had unilateral sudden visual loss and dense vitreous hemorrhage obscuring most retinal findings. Vitreous biopsy and laboratory analysis using polymerase chain reaction established the diagnosis of VZV retinitis.

Report of a Case. A 28-year-old man with AIDS had a 1-day history of severe visual loss in his left eye. He complained that the vision had been blurred in the eye for several weeks, but that the previous morning he awoke with “no vision.” The patient had been aware of his status as positive for the human immunodeficiency virus for 2 years. The AIDS diagnosis was based on a CD4 lymphocyte count of 4 cells per microliter (normal, >500 cells per microliter). His ocular history was unremarkable except for an episode of conjunctivitis 3 years previously.

His visual acuity was 20/20 OD and hand motions OS. The right eye was entirely normal. The left eye had small keratic precipitates and rare aqueous cells. A detailed fundus examination of the left eye was precluded because of a moderately dense vitreous hemorrhage obscuring most details. The macula was largely obscured. The optic nerve head was partially visible and peripapillary retinal edema and hemorrhage could be appreciated (Figure 1). The peripheral retina was visible with difficulty. Isolated areas could be seen with difficulty and showed intraretinal whitening and hemorrhage. No retinal breaks were present in these areas.

Echographic examination of the left eye showed an attached retina. The patient was treated with induction doses of ganciclovir for presumed cytomegalovirus retinitis. Serum was obtained and tested negative for syphilis, active toxoplasmosis, and sarcoidosis. Skin test results for tuberculosis were negative and a chest x-ray film was normal. His hemoglobin level was 100 g/L (normal, 133-171 g/L) and his platelet count was normal.

During the next few days there was no improvement in visual acuity or in the degree of vitreous hemorrhage. Because of the severe nature of the patient’s ocular condition and the lack of a specific diagnosis, he underwent a pars plana vitrectomy and a vitreous biopsy 4 days after the initial examination. After removing the vitreous hemorrhage, widespread retinal opacification, intraretinal hemorrhage, and retinal necrosis were noted (Figure 2). The temporal retina was very necrotic and at the completion of the vitrectomy a large retinal defect was noted. Because of this large hole and a developing intraoperative retinal detachment, an air-fluid exchange was performed and the eye was filled with silicone oil to prevent postoperative retinal detachment.

Polymerase chain reaction analysis of the vitreous biopsy specimen was positive for VZV and negative for herpes simplex virus, cytomegalovirus, and toxoplasmosis. Postoperatively, the patient con-
continued to receive intravenous ganciclovir. The retina remained attached, and retinal edema and hemorrhages resolved during the course of the antiviral therapy. The optic nerve became pale and the visual acuity fell to no light perception OS. The right eye has remained normal 1 year after the initial examination.

Comment. Spontaneous vitreous hemorrhage can occur in a variety of retinal disorders, but it is rare in ocular infections. Varicella-zoster virus retinitis in patients with AIDS typically manifests as progressive outer retinal necrosis with minimal hemorrhage. Our patient had some features of the progressive outer retinal necrosis syndrome, including profound immunosuppression, minimal anterior chamber reaction, rapid progression, and a poor visual outcome despite treatment. However, he had a markedly atypical presentation, with an acute, spontaneous vitreous hemorrhage. His human immunodeficiency virus status and the presence of retinal whitening in the periphery suggested an infectious cause and the need for laboratory diagnosis. This hemorrhagic variant of VZV retinitis adds to our knowledge of the various manifestations possible in this recently described condition.

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REFERENCES

Orbital Hemorrhage in the Nonoperated Eye as a Complication of General Endotracheal Anesthesia

We report an unusual complication of general endotracheal anesthesia. A healthy 74-year-old woman suffered a subperiosteal hemorrhage in the superior right orbit after general endotracheal anesthesia for a combined phacoemulsification, intraocular lens implantation, and trabeculectomy in the left eye. Vision was uncompromised and the hematoma resolved without surgical intervention. We conclude that recovery from general endotracheal anesthesia is capable of raising intravascular pressures and may have led to hemorrhage within a presumed normal orbit in our patient. Consideration must be given to this potential complication in patients with lesions that may predispose to intraorbital hemorrhage.

Report of a Case. A 74-year-old healthy woman underwent left phacoemulsification, posterior chamber intraocular lens implantation, and trabeculectomy. She had been experiencing frequent painful spasms from postherpetic neuralgia and was concerned about movement during the procedure. General endotracheal anesthesia was therefore provided at her request.

During the procedure, erythromycin ophthalmic ointment was placed in the right eye and the eye was then taped closed. The procedure was completed in 45 minutes and was uneventful. The highest documented intraoperative blood pressure was 160/80 mm Hg. The patient experienced a short episode of coughing during extubation.

The following morning, orbital swelling and ecchymosis of the upper eyelid was present in the right (nonoperated) eye. On questioning, the patient reported having noted painless right orbital swelling approximately 30 minutes after surgery. Visual acuity was 20/25 OD (unchanged from her preoperative baseline). There was no afferent pupillary defect and color vision was normal. Motility was partially restricted in all fields of gaze and there was 8 mm of axial proptosis. Intraocular pressure in the right eye was elevated to 36 mm Hg, above her baseline of 21 mm Hg.

A preoperative complete blood cell count was normal. Platelet count was 190×10^9/L. Coagulation studies were not performed. No antiplatelet medications were used in the perioperative period. Because there were no signs of compressive optic

Left, Coronal magnetic resonance image with gadolinium enhancement and incomplete fat suppression demonstrates subperiosteal blood (arrow) in the right orbit. Right, Parasagittal view demonstrates the hemorrhage displacing the superior rectus muscle inferiorly (arrow).
neuropathy, no therapeutic intervention was undertaken. Magnetic resonance imaging (Figure) was ordered to look for a lesion that might have predisposed to hemorrhage. The magnetic resonance image demonstrated a subperiosteal collection of blood in the superior right orbit; however, no other abnormalities were present. The proptosis resolved gradually during the next 4 weeks with no residual deficits. Fortunately, in our patient the hemorrhage occurred in the orbit of the nonoperated eye; otherwise, we may have erroneously believed our procedure was its cause.

Comment. Spontaneous orbital hemorrhage after general endotracheal anesthesia has been previously reported.1-3 Hemorrhage associated with a Valsalva maneuver has been reported during labor,4 weightlifting,5 and with the elevation of intravascular pressure that occurs during external cardiac massage and strangulation. Other conditions associated with spontaneous orbital hemorrhage include cavernous hemangioma, lymphangioma, varix, idiopathic inflammatory pseudotumor, hypertension, hemophilia, blood dyscrasias, leukemia, renal disease, vascular disease, and scurvy.

A Valsalva maneuver causes increased intravascular pressure that is transmitted to the orbital veins, which lack valves. Venous distension may then lead to hemorrhage. Krohel and Wright6 differentiated venous hemorrhage and arterial hemorrhage by the severity and acuity of symptoms. They believed that most venous hemorrhages resolved spontaneously without sequelae. However, arterial hemorrhage, such as might be caused by trauma or arteriosclerosis, frequently required surgical drainage because of optic nerve compression.

Hemorrhage beneath the periosteum has been reported4 but is less common than hemorrhage within the soft tissues of the orbit. When surgical drainage of blood is indicated, imaging studies can be helpful in directing the operative approach and should be considered.

The views expressed herein are those of the authors and do not necessarily reflect the official policy or position of the US Navy, Department of Defense, or US Government.

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Valsalva-Induced Subperi orbital Hemorrhage During Migraine

Common causes of orbital hemorrhage include trauma, surgery, vascular anomalies, tumors, and blood dyscrasias. Subperi orbital (subperiosteal) hemorrhage is less frequent. This entity has been reported rarely in healthy individuals after forceful Valsalva maneuver.1-3 We describe a 37-year-old man who suffered subperiosteal hemorrhage after an episode of migraine-associated emesis.

Report of a Case. A 37-year-old white man with a medical history remarkable only for migraine headache was seen 2 days after a typical migraine episode that included nausea and emesis. Immediately following emesis, the patient noted diplopia, decreased visual acuity of the left eye, pain with eye movement, and fullness about the left orbit.

Best-corrected visual acuity was 20/20 OU. Confrontation visual fields and pupillary responses were normal. Pain occurred with versions in all directions. Trace limitation of left supraduction and 6 prism diopters (PD) of right hypertropia were observed. Mild left upper eyelid edema and ecchymosis were present (Figure 1). Palpebral fissure heights and marginal reflex distances were 10 and 4 mm in the right eye and 8 and 3 mm in the left eye. Ballotment of the globes revealed increased resistance to retraction on the left and exophthalmometry measurements were 16 and 17 mm. Slitlamp biomicroscopy, tonometry, and funduscopy results were unremarkable.

Magnetic resonance imaging after gadolinium enhancement identified a 10×15-mm mass along the medial aspect of the left orbital roof contained within an enhanced, elevated rim of periorbit (Figure 2). Intramass signal characteristics were most consistent with hematoma. Blood indexes and clotting parameters were normal.

Management was limited to observation. The diplopia and pain resolved, and the patient was without complaint 3 weeks later. Alternate cover testing revealed 2 PDs of asymptomatic right hypertropia. Exophthalmometry results were unchanged.

Comment. Acute subperi orbital hemorrhage following Valsalva maneuver in otherwise healthy individuals has, to our knowledge, been reported 3 times. The first case oc-
curred after emesis in a 9-year-old child with influenza. A second case resulted while a 23-year-old man lifted weights. The third case afflicted a 20-year-old pregnant woman during labor.

All 4 patients had with orbital pain or fullness, normal or slightly decreased visual acuity, supraduction deficit, vertical diplopia, and axial proptosis. Each patient had normal intraocular pressures and pupillary responses. Interestingly, all were affected in the left orbit. In each case, radiography and/or echography demonstrated a superior subperi orbital hematoma. Three cases were observed and experienced complete resolution of symptoms by 1 month. Surgical exploration revealed an organized blood clot in the fourth patient.

Straining associated with the Valsalva maneuver increases intraabdominal and intrathoracic pressures. The resultant increase in jugular venous pressure is transmitted to the orbit by valveless veins. Rupture of a bridging subperi orbital vessel presumably results in subperi orbital hemorrhage.

In patients with a medical history and clinical examination suggestive of Valsalva-induced subperi orbital hemorrhage, we recommend computed tomography for confirmation. Magnetic resonance imaging, which was obtained in this case prior to referral, may also be used but is less cost-effective. Alternatively, the diagnosis can be secured by echography in the hands of an experienced clinician. We recommend observation as the initial management in patients without elevated intraocular pressure or optic neuropathy.

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This study was supported by an unrestricted grant from Research to Prevent Blindness Inc, New York, NY, and the Wisconsin Lions Foundation, Rothschild. Additional support was provided by the Veterans Administration Hospital, Madison. Dr Lucarelli is a 1996-1997 Heed Ophthalmic Fellow.

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Protracted Postsurgical Blindness With Visual Recovery Following Optic Nerve Sheath Fenestration

Optic nerve sheath fenestration is an accepted surgical treatment for pseudotumor cerebri when visual loss occurs despite medical therapy. When performed via a medial approach, optic nerve sheath fenestration may require considerable traction on the optic nerve. The nature, severity, and potential reversibility of neural injury caused by sustained rotational traction on an already-compromised optic nerve is unknown. We describe a patient who suffered complete loss of vision in the surgically treated eye following optic nerve sheath fenestration and who subsequently recovered 20/30 visual acuity.

Report of a Case. A 22-year-old woman noted headaches, pulsatile tinnitus, and bilaterally blurred vision beginning 2 weeks after the onset of a middle ear infection. She denied pain with eye movements or other neurological symptoms. Physical examination showed an obese woman with a blood pressure of 138/88. Corrected visual acuity was 20/80 OD and 20/70 OS. Both pupils reacted sluggishly to light, and there was no afferent pupillary defect. Eye movements were full. Slitlamp examination through dilated pupils showed no vitreous cells. Fundus examination showed bilateral optic disc swelling with retinal striae and hard exudates extending from the optic discs to the maculas (Figure 1). A lumbar puncture showed an opening pressure of greater than 390 mm of water with normal cerebrospinal fluid chemistry. Systemic and serological evaluations for secondary causes of pseudotumor cerebri were negative. Humphrey 30-2 visual field testing showed concentric constriction of both visual fields to 5° in the right eye and 10° in the left eye (Figure 1). Results of magnetic resonance imaging of the head were normal. The patient was treated with oral acetazolamide, 500 mg, twice daily, and an optic nerve sheath fenestration was performed in the right eye via a medial approach. Several attempts were made to rotate the globe before a successful fenestration was achieved without injury to the optic nerve or its surrounding vessels.

Six hours after the operation, the patient had no light perception in the
right eye. Indirect ophthalmoscopy showed persistent optic disc swelling and no retinal abnormalities. Fluorescein angiography showed normal retinal arterial perfusion. Postoperative magnetic resonance imaging showed no signs of orbital or nerve sheath hemorrhage. The patient was treated with intravenous methylprednisolone sodium succinate, 250 mg, 4 times daily, and oral acetazolamide, 500 mg, 4 times daily, but continued to have no light perception in the right eye for 36 hours. During this same period, visual acuity improved to 20/30 OS and the visual field expanded. Thirty-six hours after the operation, she began to identify hand motions with the right eye. After 3 days of treatment with intravenous methylprednisolone, she was discharged from the hospital and was treated with a 3-week tapering dose of oral prednisolone. Following cessation of corticosteroid therapy, her visual acuity was 20/80 OD. Three months after the operation, her visual acuity was 20/30 OD and 20/25 OS. Ophthalmoscopic examination disclosed resolution of papilledema in both eyes and diffuse optic atrophy in the right eye (Figure 2). Visual field testing showed a 5° inferior paracentral island of vision in the right eye and normalization of the visual field in the left eye (Figure 2).

**Comment.** To the best of our knowledge, this is the second published case of visual recovery from total blindness following optic nerve sheath fenestration. Flynn et al described a patient with pseudotumor cerebri who sustained complete loss of vision for a 5-hour period following an optic nerve sheath fenestration that was also performed via a medial approach. Following treatment with intravenous dexamethasone, the visual acuity recovered to 20/800. The authors advocated emergent treatment with intravenous steroids for postsurgical blindness following optic nerve sheath fenestration.
Although complete loss of vision is a recognized complication of optic nerve sheath fenestration, the pathophysiology of "tractional" optic neuropathy with late recovery of function has received little attention. The severe preoperative loss of visual field in our patient indicates that there were a limited number of functioning axons in the right optic nerve that could have been damaged by a number of mechanisms. In descending order of likelihood, these include the following: (1) a stretch injury to the optic nerve; (2) ischemia of the optic disc caused by torsion, traction, or repeated abrupt elevations in intraocular pressure; (3) vasospasm of the central retinal artery, posterior ciliary arteries, or both; and (4) postoperative orbital edema. Axonal demyelination from a stretch injury followed by remyelination of affected axons is consistent with our patient’s slow recovery of vision over a 3-month period. The influence of high-dose corticosteroid therapy on this patient’s visual recovery is uncertain since the visual acuity continued to improve long after cessation of corticosteroid therapy. Although complete loss of light perception is classically held to be a dire prognostic sign, this case demonstrates that protracted postsurgical blindness following optic nerve sheath fenestration does not preclude significant visual recovery.

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Figure 2. Top, Postoperative optic disc views following recovery of vision after optic disc fenestration in the right eye. Note temporal optic disc pallor in the right eye and resolution of papilledema in the left eye. Bottom, Postoperative Humphrey 30-2 visual fields showing 5° inferior paracentral island of vision in the right eye and normalization in the left eye.