Meningococcemia
With Vitreous Opacities: Endophthalmitis or Vitreous Hemorrhage?

Visual acuity after treatment with meningococcal endophthalmitis is variable. A child with meningococcemia and disseminated intravascular coagulation (DIC) developed vitreous opacities. Anterior segment inflammation was never observed.

Report of a Case. A 26-month-old, previously healthy girl became lethargic with a petechial rash that progressed to purpura. Intravenous cefotaxime sodium was given for presumed meningococcemia. Blood cultures yielded Neisseria meningitidis. Periocular ecchymoses prompted an ophthalmic consultation.

The patient was nonresponsive and was receiving mechanical ventilatory support during the examination. No relative afferent pupillary defect was present. The eyelids and conjunctiva were edematous and ecchymotic (Figure 1, left). Intraocular pressures and portable slit-lamp examination showed no abnormalities. An undilated fundus examination (due to the patient’s unstable neurologic status) of the optic discs and posterior poles of both eyes showed no abnormalities.

Multiorgan system failure developed with respiratory distress syndrome, thrombocytopenia, DIC, and acute tubular necrosis requiring hemodialysis. Her unstable neurologic status and high-frequency ventilation requirements prevented reliable sequential posterior segment evaluations. The eyelid edema and ecchymoses resolved. Four weeks after the initial examination, anterior segments were still quiet and bilateral dense vitreous opacities were seen on dilated fundus examination. Retinochoroidal thickening and vitreous cell were noted on ultrasonography. At the time, vitreous opacities were believed to be hemorrhagic, secondary to DIC, because ocular inflammation was absent. A second ultrasound examination 11 days later demonstrated bilateral vitreous opacities and a possible traction retinal detachment in the left eye.

A pars plana vitrectomy, membrane peel, gas fluid exchange, endolaser, and scleral buckle procedure were performed in the left eye. Extermacular areas of traction retinal detachment and necrotic retina were observed. The macula showed no abnormalities. Vitrectomy specimen cultures were negative but Gram stains demonstrated gram-negative intracellular organisms with vitreous hemorrhage (Figure 2). Pockets of neutrophils were observed in association with macrophages. Penicillin sodium was given intravitreally in the right eye and subconjunctivally in both eyes for possible subclinical endophthalmitis. Intravitreal antibiotics were not administered in the left eye because signs of active infection were absent and intraocular gas was present. Three weeks later, vitrectomy and scleral buckle were performed in the right eye. Intraoperative findings were similar to those in the left eye.

Postoperative visual acuity with correction was 20/40 OD and 20/80 OS. Extensive chorioretinal scarring was seen (Figure 1, right) in the mid peripheral fundus. Subsequently, visual acuity decreased in the left eye due to a posterior subcapsular cataract. Cataract extraction and intraocular lens placement were performed. Postoperatively, the patient would not fix and follow in the left eye. Amblyopia was diagnosed and full-time occlusion therapy was started.

Comment. Pain, photophobia, redness, decreased vision, and vitreous inflammation are usually present with endogenous endophthalmitis. Anterior chamber inflammation, considered typical of this condition, was never seen in our patient.

We describe the occurrence of meningococcal intracellular organisms associated with vitreous hemorrhages in a patient with meningococcemia and DIC. The most likely explanation for the patient’s ocular lesions is subclinical meningococcal en-
dophthalmitis. The intravenous antibotics the patient received may have rendered the organisms nonviable, and the ocular findings may have been related to partially treated or subclinical endophthalmitis; however, thrombotic events from DIC could cause a similar clinical finding. If DIC were the explanation, the neutrophil-containing organisms may have entered the vitreous cavity in association with the hemorrhage. Necrotic areas of retina were likely the result of septic emboli or vascular occlusion from DIC.

To avoid amblyopia, bilateral vitrectomies were necessary to clear the visual axes. This case illustrates the importance of considering intraocular infection in the setting of meningococcal meningitis and presents alternative hypotheses to explain the clinical events.
bulbar conjunctiva inferior to the limbus (Figure 3, Figure 4, and Figure 5). Adjacent to the nodule, a narrow zone of pigmented cells, possibly melanophages, was present in the episclera (Figure 3, Figure 4, and Figure 6), that was extensively observed around the globe. Nests of cystic compound nevus (Figure 6) were extensively present in other areas of the bulbar conjunctiva. The palpebral conjunctiva and the intraocular components showed no abnormalities. Extensive intradermal nevus composed of small round cells (Figure 7 and Figure 8) were present on the skin of the eyelids. The nevus cells had features of congenital cutaneous nevus, with deep penetration in the skin and maturation at the base.

Comment. Large congenital cutaneous nevi are associated with a risk of developing melanoma since childhood.1 Marghoob et al2 prospectively followed up 92 patients (median age, 3 years) with large congenital cutaneous nevi for an average of 5.4 years and found that the cumulative 5-year, life-table risk for the development of malignant melanoma was 4.5%. The calculated stan-
It is known that congenital blue nevi of the eyelids and orbit can lead to eyelid and orbital melanoma. Tel-lado et al. studied 21 orbital melanomas. They found that 90% of patients had an associated blue nevus and 47.5% of patients had some form of congenital melanosis.

Acquired conjunctival nevi are recognized to be precursors of conjunctival melanoma, but the risk of developing melanoma is unclear. It is known that congenital blue nevi are associated with a risk of developing melanoma, especially in the eyelids and orbit. Tello et al. studied 21 orbital melanomas. They found that 90% of patients had an associated blue nevus and 47.5% of patients had some form of congenital melanosis.

Gerner et al. analyzed 343 surgically removed conjunctival nevi. Six cases that recurred during observation were originally classified as compound nevi. Among them, 1 was recurred as malignant melanoma. They mentioned that in stationary and indolent cases in adults, excision as a routine procedure was unnecessary, since the risk of evolution of conjunctival nevi into melanoma was very low. In Japan, malignant melanoma of the conjunctiva is rare, with an estimated 6 to 7 cases per year.

In our patient, the history indicates that large nests of nevus cells involving bulbar conjunctiva, caruncle, and eyelid skin had been present since birth. Generally speaking, large nevi are associated with a risk of developing melanoma. The many nevus cells of this patient probably increased the risk of developing melanoma, a condition that should be rare in Japanese patients.

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**Eyelid Nodule: A Sentinel Lesion of Disseminated Cryptococcosis in a Patient With Acquired Immunodeficiency Syndrome**

Cutaneous cryptococcosis is a rare infection caused by Cryptococcus neoformans an encapsulated saprophytic yeast. It is 4 to 8 µm in diameter and is surrounded by a polysaccharide capsule that lives especially well in dust and soil contaminated by the excreta of pigeons. The initial site of infection is usually the lung, where cryptococcosis occurs without symptoms and persists in a latent stage for a long time. Hematogenous dissemination of the yeast may involve any organ of the body, principally the brain (70%-90% of the cases) with a meningoencephalitis or a subacute meningitis and, to a lesser extent, the skeleton, eyes, and skin. We describe a man affected by acquired immunodeficiency syndrome in whom disseminated infection with C neoformans started with a nodule on the border of his right eye.

**Figure 1.** Small papular lesion on the border of the right eyelid.

**Figure 2.** Numerous cryptococci organisms surrounded by gelatinous capsules (periodic acid–Schiff, original magnification ×1000).

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tinel lesion that preceded the spread-
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primary versus secondary disease: report of two
cases and review of literature. Am J Dermatopa-
3. Sarosi GA, Sierfarb PM, Tosh FE. Coccanus
 Coccosis: a sentinel of disseminated dis-
4. Calista D, Stagno A, Landi C. Cutaneous le-
chronic manifestations are umbeli-
culated papules, and ulcerated nod-
osed individuals with defective cell-
occurrence more frequent, involving from
5% to 10% of patients during their
life, 10% to 15% of whom had cu-
taneous involvement.3 Most promin-
ent on the face and neck, less fre-
quently located on the trunk and
extremities, the typical clinical cu-
taneous manifestations are umbeli-
culated papules with a tiny, central
hemorrhagic crust quite similar to
molluscum contagiosum.1 Less of-
ten, violaceous papules, vesicles in
a varicelliform pattern, crusted
plaques, subcutaneous nodules, or
cellulitis have been reported.4 The
most frequent opthalmologic mani-
festations of infection with C neofor-
mans involve the posterior segment of
the eye with papilledema, optic
erve atrophy, endophthalmitis, and
choroiditis.5 One existing
report describes limbal and choroi-
dal mass and another iris inflam-
atory mass in patients with
acquired immunodeficiency syn-
drome.6,7

The prognosis depends on the
degree of the patient’s immuno-
depression, the involvement of C neo-
fomans, and the prompt start of
therapy.4,6 The patient we observed
is interesting because he showed a
single nondiagnostic lesion on the
upper border of the eyelid as a sen-
tinel lesion that preceded the spread-
ing of the cryptococcal infection by
5 weeks. A rapid diagnosis permit-
ted the prompt start of appropriate
therapy. Notwithstanding this, our
patient experienced an initial spread
of cryptococcal lesions on his skin
although then serum and the colony-
stimulating factors titer of crypto-
coccal antigens were decreasing slowly. Now, after a 22-month fol-
low-up, the patient is well. Occa-
sional relapses of the opportunistic
infection have been controlled with
amphotericin B.

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Scedosporium apiospermum of the Orbit

Scedosporium apiospermum is an emerging fungal pathogen commonly
found in soil (even in hospital
potted plants1) and polluted water. Ocular infection can occur including
keratitis, conjunctival mycet-
tome, endophthalmitis, and pan-
ophthalmitis. Reports of orbital
involvement have been rare. We
describe a 68-year-old man with leu-
kenia who developed an orbital sub-
periosteal abscess from extension of
contiguous fungal sinusitis. We
also review the role of pharmacotherapeutics in the management of
this disease. Remarkably, this
patient defervesced on antifungal
therapy without aggressive surgi-
cal debridement.

Report of a Case. A 68-year-old
man with an 8-month history of acute lymphogenous leukemia
was seen in consultation for the
hematology service. He com-
plained of a red, protruding left eye and progressive diplopia over 3
days. He had been admitted to the
hospital with neutropenic fevers 5
days earlier.

He had left periorbital edema
and erythema (greatest inferomed-
ally). Palpation of the affected area
revealed no crepitus or bony abnor-
mality. Sensory divisions of the tri-
geminal nerve were intact. The left
eye was displaced forward 2 mm and
laterally 2 mm. Best-corrected vi-
sual acuity was 20/20 OD and 20/25
OS. Papillary responses were brisk
with no relative afferent pupilary de-
fect. Slitlamp examination showed
no other abnormalities. Intraocular
pressures were normal. Left eye ab-
duction was notably diminished and
there was a large angle esotropia.
Retinal evaluation showed no ab-
normals.

White blood cell count was 0.5
X 109/L, demonstrating profound
neutropenia. Computed tomog-
ographic scan (Figure 1) revealed a
pansinusitis with a soft tissue dens-
ity extending into the left infero-
medial aspect of the orbit. Needle
drainage of the left aspect of the ma-
xxillary sinus produced 30 mL of thick,
yellow fluid. Initial smears demon-
strated septated hyphae and were
negative for bacteria.

Amphotericin B (1 mg/kg per
day) was immediately started and
itraconazole (400 mg/d) was added
the next day. The fungus was iden-
tified as S apiospermum (Figure 2)
and the infectious disease consul-
ant recommended aggressive treat-
ment due to the relative in vitro ins-
sensitivity to antifungal therapy.
Surgical debridement of the si-

inuses and orbit was recommended;
however, the patient declined this
procedure. By day 2 of medical
therapy, the patient’s fever cleared
and the edema and diplopia re-
solved. On the seventh day of medi-
cal treatment, there were no ocular
signs or symptoms. The patient was
discharged from the hospital on a
regimen of identical doses of am-
photericin B and itraconazole. He
had no orbital recurrences and died

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3 months later from complications of his leukemia.

**Comment.** *Scedosporium apiospermum* is an opportunistic fungus commonly found in soil and decaying vegetation. In 1944, Emmons demonstrated that *S apiospermum* is the asexual form of *Pseudallescheria boydii*. Prior to 1944, *P boydii* was known as *Allescheria boydii*. This organism can, in cases of penetrating injury, infect healthy individuals. Here, it produces an indurated lesion at the site of the injury. In immunocompromised patients, aggressive spread is common. Systemic sites of infectious mycoses in the immunosuppressed include the lungs, joints, central nervous system, sinuses, and ears.

Orbital involvement of this organism has rarely been reported. In 1977, Gluckman et al reported orbital extension of pansinusitis of *S apiospermum* in a diabetic host. The patient had both liver and renal failure. He was treated with surgical drainage and systemic amphotericin B but died 3 months later from unrelated complications. In 1984, Anderson et al described the successful treatment of a 4-year-old boy who had suffered a penetrating orbital injury with orbitocranial involvement of the infection. His therapy included multiple surgical interventions and intravenous amphotericin B and miconazole.

In cases of localized disease, it seems as if local debridement of as much of the fungus as possible, combined with systemic antifungal medication, provides the best chance of cure. Both itraconazole and miconazole (ie, imidazoles) have shown greater effectivity in vitro and may be used as monotherapy. Combined treatment with amphotericin B and an imidazole seems reasonable.

In our patient, needle drainage of the involved maxillary sinus performed for diagnostic purposes also provided some therapeutic decompression. This, combined with amphotericin B and itraconazole, resolved the signs and symptoms of orbital infection. Surgical debridement may still be the treatment of choice in this disease; however, antifungal therapy alone may be reasonable in patients unwilling or unable to undergo aggressive surgery. Ideal dosage and duration of treatment are unknown, although this patient’s clinical course may provide helpful information.

**Figure 1.** Computed tomographic scan shows complete opacification of the maxillary sinuses bilaterally. Left, Periosteal thickening of the frontal and sphenoid sinuses and opacification of the left ethmoid sinus were also present (not shown). Right, A subperiosteal abscess was noted involving the left medial orbital wall.

**Figure 2.** *Scedosporium apiospermum*, the asexual form of *Pseudallescheria boydii*, consists of branched hyphae with ovoid conidia (lactophenyl-cotton blue, original magnification ×240).

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