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Enterobacter amnigenus endophthalmitis

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Abstract

Purpose—To describe a case of *Enterobacter amnigenus* endophthalmitis.

Methods—A 33 year-old, previously healthy man, presented with a unilateral hypopyon uveitis. Visual acuity in the affected eye was counting fingers. Initial laboratory work-up was negative. Vitreous cultures revealed the offending agent.

Results—The patient was treated with intravenous and intravitreal antibiotics and responded well. Final visual acuity was 20/30.

Conclusions—*Enterobacter* species have been rarely reported as causes of endophthalmitis. *E. amnigenus* is almost never pathogenic in humans and previous cases have been described in immunocompromised individuals. Our case demonstrates that appropriate treatment of *E. amnigenus* endophthalmitis in an immunocompetent individual can result in a good visual outcome.

Keywords

endophthalmitis; *Enterobacter*; trauma; uveitis

Introduction

Infectious endophthalmitis is a serious intraocular infection with potentially devastating visual sequelae. The majority of cases result from exogenous sources following penetrating trauma or intraocular surgery. Endogenous endophthalmitis occurs via hematogenous spread of bacteria from a remote primary focus. We present a case of *Enterobacter amnigenus* endophthalmitis, an organism that has not, to our best knowledge, been previously reported as a cause of endophthalmitis.

Case Report

A 33 year-old, previously healthy man, presented to the emergency room complaining of decreased vision and pain in his left eye. He reported a slow decline in vision over the previous two weeks and pain for two days prior to presentation. He was employed as a construction worker. He recalled that four weeks prior to presentation he felt a stinging sensation in his left eye while at work. However, the eye appeared normal, and this

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symptom resolved prior to the decline in vision. Review of systems was negative. His past medical history was significant only for hypertension. He denied intravenous drug use.

On examination, the patient was afebrile. His visual acuity was 20/15 in the right eye and counting fingers in the left eye. External examination was unremarkable with no evidence of periocular trauma. There was no afferent pupillary defect. Examination of the right eye was unremarkable. The left eye revealed a 3mm hypopyon. Of note, there was no evidence of globe injury. Dilated fundus examination of the left eye revealed 2+ anterior vitreous white blood cells. The optic nerve, macula, vessels, and peripheral retina all appeared normal.

Given the history of possible antecedent trauma, an orbital computed tomogram (CT) was obtained. No radiopaque foreign body was present, and the globe contour and appearance was normal. Laboratory data included a white blood cell count of 12.7 k/μl (4.8–10.8) with a normal differential.

The patient was diagnosed with unilateral hypopyon uveitis. Endophthalmitis was a concern given the patient's history of possible remote trauma. However, there was no evidence either by exam or CT to support a diagnosis of exogenous endophthalmitis. Furthermore, he had no signs or symptoms suggestive of an infectious nidus for endogenous endophthalmitis. A serologic work-up was initiated and included HLA-B27 and HLA-B5 typing. The patient was initiated on topical therapy with prednisolone acetate 1% hourly and cyclopentolate three times a day.

The following day, his vision had improved to 20/300, and the hypopyon had decreased to 1.5mm in size. The fundus examination was stable. He was continued on the same topical regimen while awaiting results of his serologies. He continued to deny systemic symptoms.

One week later, his vision had worsened to hand motions. His hypopyon increased to 3mm, and a dense vitritis obscured view of the posterior pole. B-scan ultrasonography showed only vitreous opacities. His laboratory testing had all returned as normal. Given his progression on topical steroids and negative laboratory testing, blood cultures were drawn, and a retina consultation was obtained. A chest x-ray was normal.

The patient underwent an anterior chamber tap and pars plana vitrectomy. Undiluted anterior chamber and vitreous samples were sent to microbiology. Intraoperative examination of the posterior pole and peripheral retina was normal with no foci of retinitis or vasculitis seen. A chalky-white material was found in the vitreous along the inferior arcade, and this was removed with vitrectomy. Intravitreal injections of Vancomycin 1mg/0.1ml and Ceftazidime 2mg/0.1ml were given.

On the first post-operative day, the patient's vision was 20/400. The hypopyon had decreased to 0.5mm. Gram stain showed moderate polymorphonuclear leukocytes but no bacteria. On the second post-operative day, cultures revealed moderate *Enterobacter amnigenus* in the vitreous aspirate. The remainder of the microbiologic and cytologic examination of the specimens was negative. The organism was sensitive to Ceftriaxone, and the patient was started on Ceftriaxone 2g intravenously every 12 hours. He was also given topical fortified Ceftazidime 50mg/ml drops to be used every 2 hours while awake. Blood cultures taken two days prior never grew any organisms.

On the fifth post-operative day, the patient's vision was 20/100. Slit lamp examination revealed no hypopyon, and the vitritis had lessened as well. However, a 360 degree peripheral retinal detachment was noted. The patient was taken to the operating room for surgical repair with scleral buckling, pars plana vitrectomy, 360 degrees of endolaser, and injection of 16% C_3F_8. No retinal breaks or tears were noted. Vitreous washings were

obtained during the case, and no organisms grew from the specimen. The patient was continued on systemic antibiotic therapy with Ceftriaxone for a two-week course.

The patient was presumed to have developed exogenous endophthalmitis from antecedent trauma. However, given the lack of clinical evidence for globe compromise, some suspicion remained for the possibility of an endogenous source. In order to further evaluate for a systemic source of infection, an abdominal CT and colonoscopy were arranged to be performed as an outpatient. These tests were negative.

Two months later, his visual acuity had improved to 20/80. He had a posterior subcapsular cataract but an otherwise normal ophthalmic examination. Cataract surgery and a YAG capsulotomy were performed subsequently. Following these procedures and six months from his initial presentation, his best corrected visual acuity was 20/30.

Discussion

To the best of our knowledge, this case represents the first report of *Enterobacter amnigenus* endophthalmitis. Our patient is believed to have acquired his infection exogenously from trauma while working on a construction site. The delay in presentation may explain the lack of external evidence for globe compromise. Presumably, the globe was penetrated in such a way as to leave no evidence of prior external trauma on examination four weeks later. The “chalky-white material” found on vitrectomy was suspicious for foreign body debris, but this could not be confirmed on histopathologic examination of the specimen. A comprehensive systemic work-up was performed and showed no evidence of a remote focus of infection. As such, a diagnosis of exogenous endophthalmitis was made. Of course, it is impossible to exclude the possibility of endogenous endophthalmitis with an occult source that responded to intravenous antibiotics. However, this was felt to be much less likely in a healthy young man without any systemic symptoms or risk factors for an endogenous infection.

Enterobacter species are commensal organisms of the gastrointestinal tract and are generally considered pathogenic only for patients with lowered resistance to infection or impaired immunity. Members of this genus have been reported as rare causes of endophthalmitis. The earliest case report of endophthalmitis occurring secondary to *Enterobacter* was a case of post-surgical endophthalmitis caused by *Enterobacter cloacae* published in 1966.[1] *Enterobacter cloacae* has also been reported as a causative agent of post-traumatic endophthalmitis, often as a part of mixed infections.[2] Ocular infections resulting from gram-negative organisms result in a poor visual outcome for the majority of patients. Infections with *Enterobacter* species are no exception. In 1994, Mirza et al. reported an epidemic of post-surgical *Enterobacter* endophthalmitis in seven patients. Four of the seven patients required evisceration, two developed phthisis, and only one patient retained any vision.[3] The subspecies *Enterobacter amnigenus* was classified in 1981.[4] *E. amnigenus* has been isolated in the environment from soil samples and thermal springs[5]. In humans, it has been cultured from sputum, blood cultures, stool samples, and wounds often without evidence of clear pathogenicity.[6] Capdevilla et al. reported five cases in which *E. amnigenus* was isolated from patients and believed to be the responsible organism.[7] In each of these cases, the patient had a chronic disease state which predisposed them to systemic infection. The scarcity of reports in the literature reflects the rarity in which *Enterobacter* species, and *E. amnigenus* in particular, produce infections in humans.

Endophthalmitis remains a devastating condition with high ocular morbidity. A high clinical suspicion must be maintained to achieve early diagnosis and prompt management. The present case of *Enterobacter amnigenus* endophthalmitis represents an organism not

previously reported to occur in the eye. Our patient responded well to medical and surgical therapy despite a delay in presentation.

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Summary Statement

To our best knowledge, this is the first report of *Enterobacter amnigenus* endophthalmitis. The patient presented with a unilateral hypopyon uveitis. Initial laboratory work-up was negative. Vitreous cultures revealed the offending agent. The patient was treated with intravenous and intravitreal antibiotics and responded well.