

Endophthalmitis due to *Delftia acidovorans*: An unusual ocular pathogen

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Endophthalmitis is a dreaded postoperative complication of cataract surgery. *Delftia acidovorans* is usually nonpathogenic and an unusual ocular pathogen. Isolated reports of *delftia*-associated sepsis, otitis media, endocarditis, keratitis, etc. exist in literature. We report a rare and unique case of *delftia*-related endophthalmitis in a 67-year-old male diagnosed 2 weeks after uneventful cataract surgery. He was treated successfully with core vitrectomy and intravitreal antibiotics. Microbiological evaluation of vitreous sample identified the

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causative organism as *Delftia acidovorans*. Post-vitreotomy fundus evaluation at 1 week revealed the presence of retinal vascular sheathing and sclerosis along with few retinal hemorrhages. Final visual recovery was poor due to the presence of macular edema, *epiretinal* membrane, and temporal disc pallor.

Key words: *Comamonas acidovorans*, *Delftia acidovorans*, hemorrhagic occlusive retinal vasculitis, postoperative endophthalmitis, retinal vasculitis

Endophthalmitis is a rare but the most dreaded postoperative complication of cataract surgery. *Staphylococcus* species is the most common organism associated with postoperative endophthalmitis.^[1] *Delftia acidovorans*, also known as *Comamonas acidovorans*, is a gram-negative, aerobic, nonfermenting, nonpathogenic organism. Rare reports of its isolation from respiratory tract infections, catheter-associated sepsis, otitis media, etc. do exist in literature but clinically it is seldom significant.^[2,3] We, hereby, report a case of post-cataract surgery endophthalmitis due to *Delftia acidovorans* in a 67-year-old male with a poor visual outcome. To the best of our knowledge, this is the first case of postoperative endophthalmitis reported due to *Delftia acidovorans*.

Case Report

A 67-year-old male patient presented with a history of sudden onset gross diminution of vision, pain, and redness in right eye (RE) 2 weeks following uneventful phacoemulsification surgery. His best-corrected visual acuity (BCVA) on postoperative Day 1 was 20/40 in RE with clear cornea, 1+ cells, and a clear media with normal fundus. Two weeks following the surgery, he had history of rubbing the operated eye due to dustfall in the eye. This was followed by redness, pain, and decreased vision in the eye. He had no systemic comorbidities. His BCVA at presentation was hand movement close to face in RE and 20/80 in the left eye (LE). Slit-lamp examination of RE showed

circum corneal ciliary congestion, cells 4+, 2 mm hypopyon, and fibrinous membrane over the intraocular lens [Fig. 1a] obscuring view of the fundus. LE examination showed immature cataract and normal fundus. Ultrasound B (USG B) scan of RE showed heterogenous echogenicities in vitreous cavity suggestive of vitritis due to endophthalmitis [Fig. 1b]. Vitreous tap was done and intravitreal vancomycin (1 mg/0.1 mL) and ceftazidime (2.25 mg/0.1 mL) were injected. Gram stain of vitreous sample showed gram-negative bacilli [Fig. 2d]. The patient was started on intravenous (IV) ciprofloxacin 200 mg twice a day, topical moxifloxacin drops hourly, atropine drops thrice a day, and prednisolone acetate drops every two hourly. Evaluation after 48 h showed no clinical improvement and USG B scan showed increased vitreous echogenicities [Fig. 1c]. Although core vitrectomy was planned due to the observed clinical deterioration, it had to be deferred till the next day morning (i.e., 60 h from initial presentation) due to logistic reasons. Second dose of intravitreal antibiotics—vancomycin (1 mg/0.1 mL) and ceftazidime (2.25 mg/0.1 mL)—were injected in the same day evening while core vitrectomy was scheduled on a subsequent day. Meanwhile, the culture of the vitreous sample showed nonhemolytic, gray moist colonies on 5% sheep blood agar and nonlactose fermenting colonies on MaConkey agar. The colonies were identified as *Delftia acidovorans* by an orange indole reaction and Vitek 2 System (98% probability) (Biomérieux, USA). The organism was sensitive to ceftazidime, ceftriaxone, levofloxacin, cefoperazone/sulbactam, meropenem, and chloramphenicol. Core vitrectomy was done subsequently with repeat intravitreal ceftazidime (2.25 mg/0.1 mL) injection alone based on the drug sensitivity report. Intraoperative retinal examination showed dense white exudates in the vitreous cavity, thick exudates overlying the disc and macula, and perivascular exudates. Based on the sensitivity pattern, the patient was started on IV ceftazidime 2 g twice a day and topical fortified ceftazidime drops hourly. Post vitrectomy, serial B scans of RE showed resolving vitritis. Gradual media clearing was noted on daily fundus

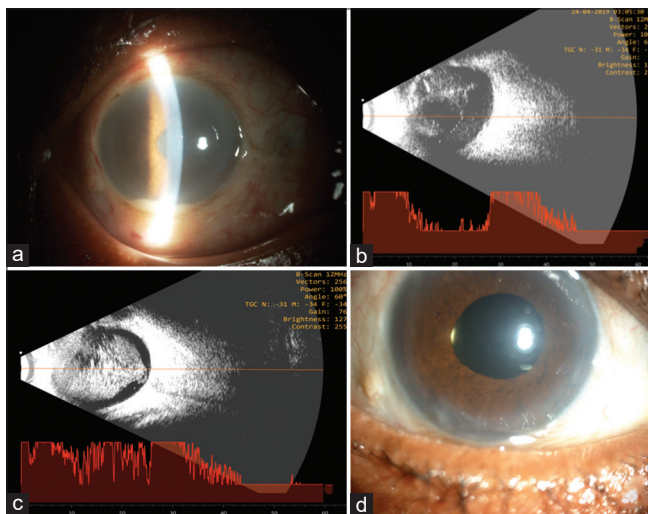


Figure 1: (a) Anterior segment image of right eye at presentation showing circum ciliary congestion and hypopyon. (b) Ultrasound B scan image of the right eye at presentation showing heterogenous vitreous echogenicities suggestive of endophthalmitis. (c) Ultrasound B scan image of the right eye at 48 h after presentation showing increased vitreous echogenicities. (d) Anterior segment image of right eye at 1 month showing clear cornea, occasional cells in the AC, normal iris color and pattern, and PCIOL *in situ*

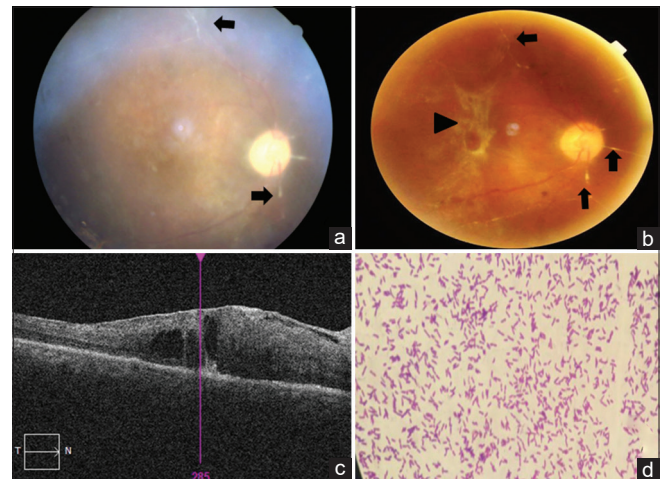


Figure 2: (a) Fundus image of right eye at 1-week post vitrectomy showing sheathing and sclerosis of vessels nasal and inferior to disc, and along the superotemporal arcade (black arrows). (b) Fundus image of right eye at 1 month post vitrectomy showing temporal disc pallor (red arrow), sclerosis and sheathing of vessels (black arrows) with ERM (triangle). (c) OCT image of right eye showing CME and ERM. (d) Gram stain of vitreous sample showing gram-negative bacilli which on culture had grown *Delftia acidovorans*

evaluation. At 7 days post vitrectomy, fundus examination revealed sclerosed retinal vessels along the superotemporal arcade, below and nasal to the disc [Fig. 2a], and few retinal hemorrhages through a hazy media. Sclerosed vessels were seen in the areas corresponding to perivascular exudates documented during intraoperative retinal examination. At 1-month post vitrectomy, slit-lamp examination showed clear cornea, occasional cells in the anterior chamber (AC), normal iris color and pattern (no evidence of neovascularisation), and posterior chamber intraocular lens (PCIOL) *in situ* with a fairly clear media [Fig. 1d]. Fundus examination showed temporal disc pallor; sclerosis and sheathing of retinal vessels [Fig. 2b]; and cystoid macular edema (CME) with epiretinal membrane (ERM), confirmed on optical coherence tomography (OCT) [Fig. 2c]. Visual acuity in the RE improved to 20/320 which was maintained till last follow-up visit.

Discussion

Delftia acidovorans was previously identified by the names of *Comamonas acidovorans* or *Pseudomonas acidovorans*.^[3] It is a gram-negative, nonfastidious, aerobic rod and a member of *Pseudomonas* RNA type III group. *Delftia* is usually isolated from soil, water, raw milk, animal infections, etc. They only seldom cause human infections, viz., endocarditis, otitis, nosocomial sepsis, peritonitis, urinary tract infections, and keratitis. Notwithstanding few reported cases of *delftia*-associated keratitis, *D. acidovorans* is still contemplated as a highly unusual ocular pathogen.^[4,5] To the best of our knowledge, our case is the first reported case of *delftia*-associated endophthalmitis. As only a handful reports of *delftia*-associated ocular infections exist in literature, the clinical characteristics and the antimicrobial resistance patterns of the organism are relatively unfamiliar. Study of the clinicomicrobiological profile, antibiotic sensitivity pattern, and treatment response of our case is, therefore, essential to understand the risk factors, clinical presentation, and outcome of *delftia*-related endophthalmitis.

Our patient presented with dense fibrinous hypopyon uveitis, had no preexisting ocular or systemic comorbidities. In our case, there was an initial lack of response to intravitreal ceftazidime prior to core vitrectomy. This can be explained probably by the high virulence of the organism, high initial bacterial and toxin loads which had reduced subsequently after core vitrectomy. The patient, therefore, had shown response with clearing of media to intravitreal, topical, and IV ceftazidime. There was no subsequent recurrence of infection till last follow-up.

Intraoperative retinal examination showed perivascular retinal exudates. Follow-up evaluation of the fundus at 1 week and at 1 month showed features of retinal vasculitis in the form of sheathing and sclerosis of vessels, few retinal hemorrhages, etc. Retinal vasculitis has been described as a rare manifestation of bacterial endophthalmitis secondary to *staphylococcus* and *streptococcus* species.^[6-9] In all these cases, diagnosis of endophthalmitis was heralded by retinal hemorrhages and vasculitis as early hallmark of the diagnosis. Extensive retinal perivasculitis can appear clinically as vascular sheathing as seen in our case. Although such a presentation in endophthalmitis is rare, the visual outcome is often poor. Early intervention in such cases in the form of vitrectomy is beneficial as it reduces

bacterial toxins and white blood cells that produce harmful proteolytic enzymes and cause tissue destruction.^[6] In hindsight in our case, an earlier vitrectomy would probably have been helpful in improving final outcome but dense vitreous exudates had precluded initial fundus evaluation.

Another important differential diagnosis to consider in our case based on the fundus findings is coexistent hemorrhagic occlusive retinal vasculitis (HORV). HORV is described as a type III hypersensitivity or delayed immune reaction akin to leukocytoclastic vasculitis or Henoch-Schönlein purpura. It is caused by deposition of immune complexes in vessel walls, with subsequent activation of complement pathway and numerous cytokine cascades. It usually presents between 2 days to 2–3 weeks following intraocular use of vancomycin (either intracameral or intravitreal) in the form of retinal vasculitis and small retinal hemorrhages. Retinal hemorrhages in HORV are mostly clustered around occluded venules. Other uncommon manifestations in HORV can be large retinal hemorrhages (>1 disc diameter), cuffing or sheathing of the venules, macular edema, and whitening. Peripheral retinal involvement is more common.^[10] In our case, intraoperative perivenous exudates were noted during initial core vitrectomy on the third day after intravitreal vancomycin and vessel sheathing in the corresponding vessels was documented in fundus photograph on day 7. Retinal hemorrhages in our case were also very few as compared to extensive retinal hemorrhages seen in classical HORV. Vessels around the disc as well as along the temporal arcades showed sheathing and sclerosis in our case while in HORV, involvement of peripheral venules is more common. HORV can occasionally be a dose-dependent response to the drug.^[10] Our patient had received two doses of intravitreal vancomycin prior to core vitrectomy which could possibly cause a dose-dependent HORV. However, the onset of clinical findings and spectrum of retinal manifestations in our case are more in favor of endophthalmitis-associated vasculitis. We do acknowledge that HORV cannot be ruled out completely due to overlapping clinical features. Complexity of the diagnosis in our case lies in the intermingled manifestations of both endophthalmitis and HORV.

Fundus fluorescein angiography (FFA) can show vascular occlusion corresponding to the areas of hemorrhage in HORV. Also, it can show the status of macular perfusion. Due to logistic reasons, FFA could not be done in our case which is a certain drawback here.

In HORV, the initial inflammatory response is followed by the onset of an ischemic drive which results in retinal neovascularization and neovascular glaucoma. Antivascular endothelial growth factor (Anti-VEGF) injections and panretinal photocoagulation may be useful at this stage to prevent these complications of severe retinal ischemia associated with HORV. Rapid development of iris and angle neovascularization leading to secondary neovascular glaucoma is uncommon in postoperative endophthalmitis-associated retinal vasculitis. Hence, close follow-up of our patient to detect these signs at the earliest is recommended. However, till last follow-up, our patient had no evidence of neovascularization or glaucoma.

ERM, CME, and temporal disc pallor seen in our case were responsible for poor post surgery visual outcome. Visual evoked potential (VEP) can be done to assess optic nerve function. Optic atrophy will show reduced amplitude and

normal latency of P100 in pattern-reversal VEP. Also macular perfusion status can be studied using either FFA or optical coherence tomography angiography (OCTA). Visual outcome for ERM peeling surgery will depend on the macular perfusion status in FFA/OCTA and optic nerve function in VEP.

D. acidovorans usually shows susceptibility only to broad-spectrum cephalosporins, trimethoprim-sulfamethoxazole, ureidopenicillins, fluoroquinolones, and tetracyclines but it is often resistant to aminoglycosides. Therefore, timely intervention, isolation of the organism, identification of the species, and antimicrobial sensitivity testing are all mandatory to decide the most appropriate antimicrobial therapy.^[2,3] On biochemical testing, the organism isolated in our case was urease nonproducer, kligler iron agar nonfermenter and showed an orange indole reaction test. On addition of Kovac's reagent in nutrient agar, the colonies turned orange. This occurs due to production of anthranilic acid from tryptophan on addition of Kovac's reagent which subsequently imparts the characteristic "pumpkin orange" color to the media.^[3] Further identification test using VITEK2 colorimetric card (BioMerieux) confirmed the organism as *Delftia acidovorans*. The organism in our case showed sensitivity to ceftazidime, ceftriaxone, levofloxacin, cefoperazone/sulbactam, meropenam, and chloramphenicol; and responded to treatment with intravitreal, intravenous, and topical ceftazidime after core vitrectomy. There was no recurrence of infection till last follow-up.

Conclusion

Delftia acidovorans is an unusual ocular pathogen and can rarely cause endophthalmitis. Differential diagnosis of retinal vasculitis as well as HORV should be considered in any endophthalmitis case presenting with vascular sheathing, sclerosis, and retinal hemorrhages. Final visual outcome may be poor in such cases due to associated sequelae of CME, ERM, macular hypoperfusion, etc. as seen in our case.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published

and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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