# A rare case of *Lecythophora* endogenous endophthalmitis: Diagnostic and therapeutic challenge

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Fungal endogenous endophthalmitis (EE) secondary to contaminated intravenous fluid infusion is frequently seen in developing countries. Molds and yeasts are commonly implicated as the causative agents. Dematiaceous fungi such as *Lecythophora* have been linked to exogenous endophthalmitis but have never been reported to cause EE. We report a case of *Lecythophora* EE that was successfully managed with pars plana vitrectomy along with intravitreal and systemic voriconazole. Endogenous endophthalmitis (EE) is a potentially devastating intraocular infection caused by intraocular spread of pathogens through blood stream. It generally accounts for 2%–16% of all reported endophthalmitis cases.[1] Predisposing risk factors include diabetes mellitus, malignancies, intravenous drug use, organ abscess, immunosuppressive therapy, indwelling catheters, urinary tract infection, organ transplant, end-stage renal or liver disease, and endocarditis.[2] It may occur in patients with no overt signs of systemic infection, particularly in the setting of contaminated intravenous fluid infusion in a rural setting.[3] Among the three broad categories of pathogens responsible for EE-bacteria, yeast, and molds, cases caused by molds are most infrequent and have the worst outcomes.[4] While *Candida* and *Aspergillus* are the most common species among fungal causes of EE, *Lecythophora* has been rarely reported as a cause of endophthalmitis due to exogenous causes.[5-8] We, herein, report a case of EE caused by *Lecythophora* species.

**Key words:** Endogenous endophthalmitis, Lecythophora, intravitreal voriconazole

## Case Report

A 26-year-old male presented with pain and redness in the left eye for 15 days with rapid and progressive decrease in vision. The visual acuity was 6/9 and counting fingers (two feet) in the right and left eyes, respectively. Intraocular pressure was 16 and 14 mmHg in the right and left eyes, respectively. The right eye was normal. The left eye anterior segment showed corneal endothelial dusting, 3+ anterior chamber cells, moderate flare, and pigment on anterior lens surface. Fundus evaluation of the left eye showed dense vitritis with vitreous membranes and a submacular abscess [Fig. 1a]. There was no history of ocular trauma or surgery. There was history of hospitalization and intravenous infusion of normal saline for fever and dehydration 1 month ago. There were no features suggestive of any systemic illness. With a probable diagnosis of contaminated intravenous infusion-related EE of the left eye, the patient was admitted for systemic workup. Systemic examination by an internist did not reveal any focus of infection, and the patient was afebrile. Complete blood counts and liver and renal function tests were within normal limits, and blood and urine cultures were sterile. A diagnostic pars plana vitrectomy (PPV) was performed in the left eye with intravitreal amphotericin B (5 µg/0.1 ml) + dexamethasone (400 µg/0.1 ml). The vitreous fluid samples were sent for bacterial and fungal smears and cultures. The potassium hydroxide (KOH) mount of vitreous sample revealed septate hyphae with branching at acute angles, and the panfungal polymerase chain reaction was positive [Fig. 1b and c]. Oral itraconazole 200 mg BD was started. Following an initial improvement, and after five injections of intravitreal amphotericin B with dexamethasone, his vision deteriorated with worsening of vitritis and an increase in the size of submacular abscess at 4 weeks [Fig. 2a]. The culture showed growth of a fungus which was not sporulating. Molecular sequencing of this growth was done with partial internal transcribed spacer-1 gene as the target gene. Direct sequencing of the amplicon was done using bidirectional Sanger's method. The sequence was checked with public database of NCBI, which found a 100% match with *Lecythophora* species. Oral voriconazole 200 mg BD was started in place of itraconazole. Five weekly injections of intravitreal voriconazole (100 µg) were given. Oral voriconazole was stopped after 3 months. At 6-month follow-up, the visual acuity was counting fingers at 2 meters, with a quiet eye and macular scar [Fig. 2b].

## Discussion

Ocular involvement by *Lecythophora* has been reported in the form of bleb-related endophthalmitis,[5] after corneal cyanoacrylate glue application and after deep anterior lamellar keratoplasty.[6,7] *Lecythophora* is a mold that lacks a known sexual state and is thus categorized under Fungi Imperfecti. It is generally classified as a dematiaceous fungus which is mainly attributed to its darkly colored fungal body due to the presence of melanin in the cell walls of its conidia, hyphae, or both and is ubiquitous in nature.[9] *Lecythophora* species (*L. mutabilis, Lecythophora hoffmannii*, and *Lecythophora lignicola*) are cosmopolitan and are occasionally isolated from soil and from plant debris. *L. mutabilis* has been described

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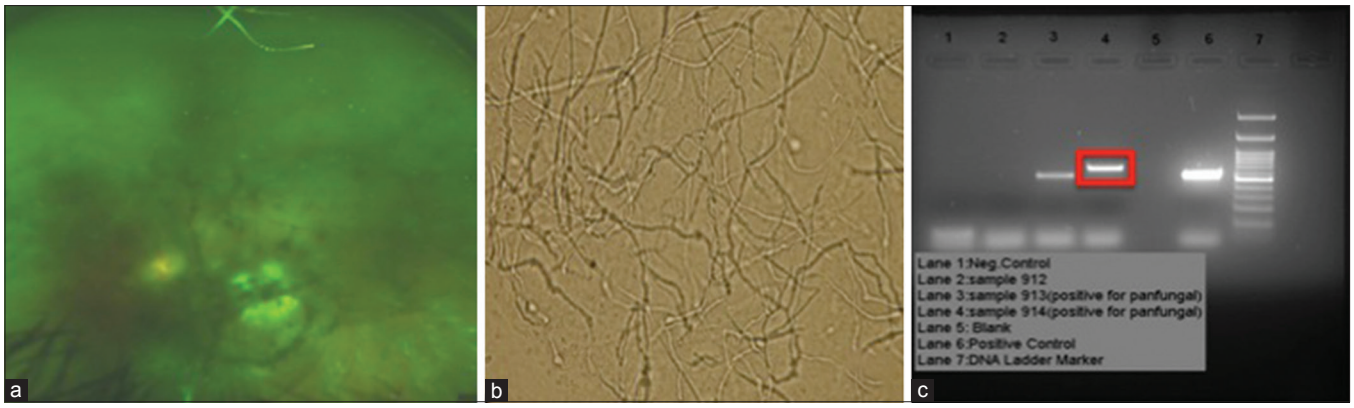


**Figure 1:** (a) Fundus photograph of the left eye at presentation, showing media clarity of Grade 3 with vitreous membranes and a 2–3 DD yellowish submacular abscess with indistinct margins, (b) potassium hydroxide mount showing septate hyphae with branching at acute angles, (c) picture of nitrocellulose gel showing positivity of panfungal polymerase chain reaction from the vitreous of our patient in Lane 4 (red square box), negative control in Lane 1, positive control in Lane 6, and DNA ladder marker in Lane 7 was present

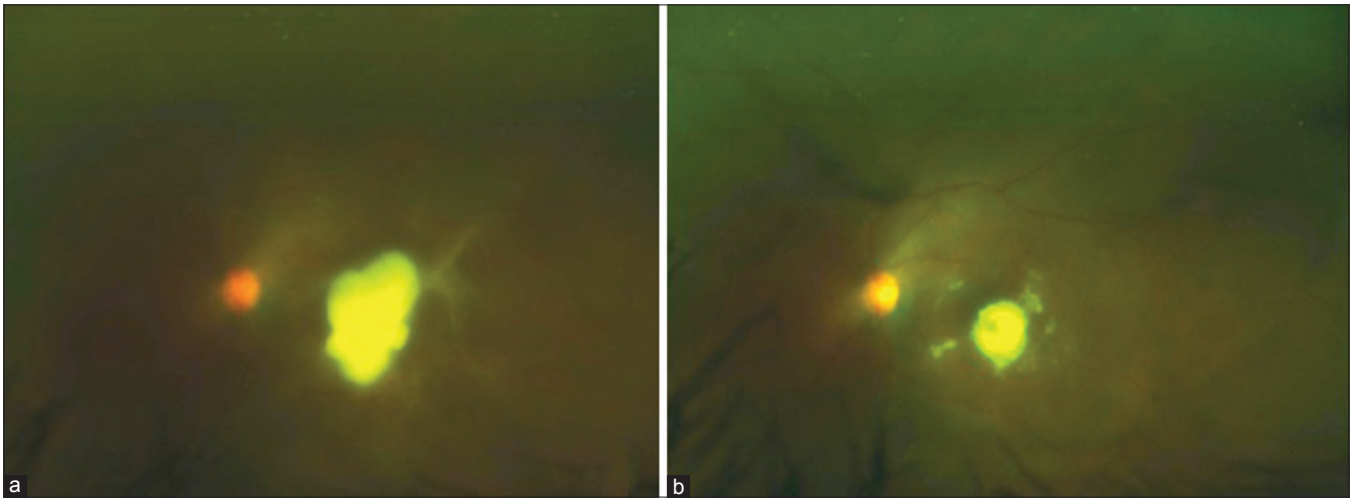


**Figure 2:** (a) Fundus photograph of the left eye 1 month after pars plana vitrectomy, showing media clarity of Grade2/3 with increase in size of the submacular abscess to 4–5 DD, (b) fundus photograph at 6-month follow-up, showing media clarity of Grade 1/2 with a submacular scar of 1–1.5 DD in the area of the abscess

to be the causative agent of human peritonitis, endocarditis, endophthalmitis, and keratitis. It is sensitive to voriconazole (systemic and intravitreal) and resistant to amphotericin B.[10]

## Conclusion

Besides the clinical and diagnostic challenges of other EE (fungal or bacterial), this case posed significant diagnostic and therapeutic challenges despite an early KOH report suggesting a fungal cause of EE. First, this is a rare fungus and was never considered in the differential diagnosis in our case as it has never been reported to cause EE. Second, it is difficult to detect because of poor growth on culture. Third, its resistance to conventional empirical antifungal therapy with amphotericin B poses treatment issues. Therefore, a high index of suspicion, as in any case of EE, should be kept while dealing with cases with severe infection in the eye, a recent history of hospitalization or intravenous infusion, an immunocompetent patient, and a persistent infection in the eye despite an early antifungal treatment. In the absence of culture showing any definitive results, a more aggressive approach by the microbiologist in the form of molecular sequencing can salvage the eye by revealing a rare pathogen causing EE.

### 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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Nil.

### Conflicts of interest

There are no conflicts of interest.

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