Answer to Ophthaproblem *continued from* **page 155**

1. Endogenous fungal endophthalmitis

Endogenous endophthalmitis is a relatively rare pathology, representing only 2% to 8% of all cases of endophthalmitis.[1] It is more common among patients with diabetes, immunocompromised or hospitalized patients, and parenteral drug addicts.

The sources from which the infectious agent spreads, in order of frequency, include liver abscesses, pneumonia, endocarditis, and urinary tract infections.[2] Endogenous fungal endophthalmitis has the same presentation rates as the endogenous bacterial endophthalmitis caused by *Candida albicans*, which is the most frequently found microorganism. *Candida* endophthalmitis is present in 10% to 20% of systemic candidiasis and is associated with increased mortality among critically ill patients.[2] The most common signs and symptoms at presentation include blurred vision, eye pain, hyperemia, hypopyon, corneal edema, and vitritis. Treatment should be initiated as soon as possible, as this is a systemic infection with a high mortality rate.

In this case, there was clinical suspicion of endogenous endophthalmitis, so blood cultures were taken. We admitted the patient and administered empirical antifungal treatment with systemic intravenous fluconazole and intravenous antibiotics (ceftazidime and vancomycin). The patient underwent full evaluation by the internal medicine department to search for other sources of infection and was also diagnosed with oral fungal infection; she remained on the same treatment. After several days of monitoring, oral corticosteroid treatment was added. We observed improvement in vitritis and retinochoroidal focus, and her visual acuity improved to 10/20 with the treatment. Vitreous analysis was not pursued because of both the patient's poor systemic condition and the good response to intravenous treatment. Subsequently, the blood culture was positive for *Candida*, confirming the fungal origin of the disease.

Diagnosis

In patients with possible symptoms of endophthalmitis, the most important thing is to guide the differential diagnosis; pathologies especially associated with chorioretinitis plates include toxoplasmosis, toxocara, and cytomegalovirus retinitis. Fundus examination should guide diagnosis of one disease over another. In our case, no associated chorioretinal scars were visible, thereby excluding toxoplasmosis as an option. There were also no heavy hemorrhages or necrosis that suggested cytomegalovirus chorioretinitis. We did find a plate of chorioretinitis bulging into the vitreous with associated vitritis, characteristic of fungal endophthalmitis. Endogenous fungal endophthalmitis is a condition that seriously compromises vision and that has been associated with mortality rates of up to 58%.[2]

Treatment

For good vital and visual prognosis, it is essential to complete early diagnostic orientation and to introduce empirical treatment before laboratory confirmation. It is important to note that in our case, it was the diagnosis of endophthalmitis that led to detection of, and treatment for, candidemia.

Unlike other forms of endophthalmitis, systemic treatment is the mainstay of endogenous endophthalmitis. For many years, amphotericin B and flucytosine were the only antifungals available for systemic treatment. Fortunately, the antifungal armamentarium has expanded in the past 2 decades with the emergence of new drugs such as itraconazole, fluconazole, lipid formulations of amphotericin B, voriconazole, and caspofungin, which are distinguished by their pharmacokinetic and pharmacodynamic spectrum.

In this case, we used intravenous fluconazole, an antifungal belonging to the triazole group, which, along with voriconazole, includes drugs that are achieving great results and that do not have the systemic side effects that accompany amphotericin B.[3]

Intravitreal treatment and vitrectomy depend on the evolution of the pathology, the virulence of the organism, and the resistance developed. Because the use of intravitreal antifungal therapy is not well established and carries a high rate of retinal toxicity, we decided to wait to see if the pathology evolved with intravenous treatment. The role of corticosteroids is also unclear. Although, according to the literature, corticosteroids have no effect on final visual acuity, they do have an effect on the speed of resolution. Thus, we opted to include them in our treatment, although not from the time of diagnosis.[3]

Indications for vitrectomy in endophthalmitis include visual acuity of light perception or less, total loss of fundus reflex, afferent pupillary defect, corneal infiltration ring, or worsening symptoms after 48 to 72 hours of medical treatment.[4,5] There is some controversy in this regard; recent studies point to benefit from an increase in early vitrectomy.[5] In this case, we decided to perform vitrectomy, if necessary, as a delayed treatment option because of the good results with systemic antifungals.

Conclusion

Endogenous fungal endophthalmitis is a systemic infection with high risk of mortality, and prognosis often depends on correct ophthalmologic diagnosis.

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Competing interests
None declared

References

1. Lemley CA, Han DP. Endophthalmitis: a review of current evaluation and management. *Retina* 2007;27(6):662-80. Erratum in: *Retina* 2007;27(7):table of contents.
2. Ness T. Endogenous endophthalmitis [article in German]. *Ophthalmologe* 2007;104(11):935-9.
3. Khan FA, Slain D, Khakoo RA. Candida endophthalmitis: focus on current and future antifungal treatment options. *Pharmacotherapy* 2007;27(12):1711-21.
4. Ness T, Pelz K, Hansen LL. Endogenous endophthalmitis: microorganisms, disposition and prognosis. *Acta Ophthalmol Scand* 2007;85:852-6.
5. Takebayashi H, Mizota A, Tanaka M. Relation between stage of endogenous fungal endophthalmitis and prognosis. *Graefes Arch Clin Exp Ophthalmol* 2006;244:816-20.