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Chapter

Infectious Endophthalmitis: Ongoing Challenges and New Prospectives

Mohamed Al-Abri, Ahmed Al-Hinai and Nawal Al-Fadhil

Abstract

Endophthalmitis is a rare but potentially sight and organ-threatening ocular emergency characterized by marked intraocular inflammation. It can be categorized into two broad categories of exogenous and endogenous types. Exogenous endophthalmitis is caused by inoculation of the globe by either bacterial or fungal microorganisms from an external environment and most commonly occurs as a complication of intraocular surgeries or procedures and open globe injuries. Blurred vision and pain are the main symptoms, and gram-positive coagulase-negative organisms are the main etiology of exogenous endophthalmitis. Endogenous endophthalmitis is caused by the hematogenous spread of microorganisms from distant sites of the body into the globe. Both categories lead to subsequent intraocular inflammation and potentially severe visual and anatomical devastating consequences. In addition, they have different risk factors and causative microorganisms, and thus, require somehow different diagnostic and treatment approaches. In this review chapter, further review of infectious endophthalmitis in terms of risk factors, causative pathogens, clinical presentations, prognosis, prevention, and the latest therapeutic recommendations are discussed.

Keywords: endophthalmitis, exogenous, endogenous, causes, treatment, prognosis

1. Introduction

Endophthalmitis in general is classified into infectious and non-infectious categories. Furthermore, infectious endophthalmitis is subclassified into exogenous and endogenous (Box 1) [1–5].

Although the term endophthalmitis refers to an infectious or non-infectious inflammation, its use often refers to an infectious origin. Exogenous endophthalmitis is caused by inoculation of the eye by either bacterial or fungal microorganisms from an external environment and most commonly occurs as a complication of ocular surgery, traumatic open globe injuries, or intravitreal injections [2]. Acute postoperative endophthalmitis occurs within 6 weeks of the intraocular surgery or procedure [3].

The incidence of endophthalmitis after various intraocular procedures is low, and it is illustrated in Table 1 [6–15].
Eye Diseases - Recent Advances, New Perspectives and Therapeutic Options

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Percentage (%)</th>
<th>Pathogen</th>
<th>Percentage (%)</th>
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<tbody>
<tr>
<td>Gram-positive bacteria</td>
<td>75–85%</td>
<td>Gram-negative bacteria</td>
<td>10–15%</td>
</tr>
<tr>
<td>Staphylococcus epidermidis</td>
<td>43%</td>
<td>Pseudomonas</td>
<td>8%</td>
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<td>Staphylococcus spp.</td>
<td>57%</td>
<td>Proteus</td>
<td>5%</td>
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<tr>
<td>Staphylococcus aureus</td>
<td>15%</td>
<td>Hemophilus influenzae</td>
<td>Less than 1% (higher in late-onset bleb-associated)</td>
</tr>
<tr>
<td>Propionibacterium acnes</td>
<td>Rare</td>
<td>Klebsiella</td>
<td>Less than 1%</td>
</tr>
<tr>
<td>Bacillus cereus</td>
<td>1%</td>
<td>Coliform spp.</td>
<td>Less than 1%</td>
</tr>
<tr>
<td>Fungi</td>
<td>Rare</td>
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<tr>
<td>Candida parapsilosis</td>
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<td></td>
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<tr>
<td>Aspergillus</td>
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<td></td>
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<tr>
<td>Cephalosporium spp.</td>
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Table 2. Microorganisms causing infectious endophthalmitis.

Box 1. Classification of Endophthalmitis.

Procedure | Incidence of endophthalmitis |
--- | --- |
Cataract extraction | 0.053% to 0.145% |
Pars Plana Vitrectomy | 0.03% to 0.05% |
Glucoma filtering surgery | 0.06% to 1.32% |
Penetrating Keratoplasty | 0.11% to 0.16% |
Intravitreal injections | 0.04% to 0.08% |

Table 1. Different intraocular procedures and incidence of endophthalmitis.
Causative microorganisms of infectious endophthalmitis in general are listed and summarized in Table 2 [15]. Intraocular inflammation caused by viruses, protozoa, or helminths is usually named uveitis.

2. Acute postoperative infectious endophthalmitis

The onset of acute postoperative infectious endophthalmitis (APIE) onset is within 6 weeks from the intraocular procedure [3]. It has a typical presentation of severe ocular pain and reduction in vision. In addition; eyelid edema is observed in around one-third of the patients. The conjunctiva is congested and corneal edema is commonly present. Intraocular inflammatory signs (cells, fibrin, and hypopyon) are the main findings and these involve both anterior chamber (AC) and the vitreous cavity. The red reflex is usually absent in the affected eye. Hypopyon and fibrinous reaction in the AC are typical features of APIE. B-scan ultrasound, usually, reveals vitreous hyper echoic opacities due to vitritis (Figure 1).

The APIE can be also subclassified into early (within 7 days after surgery) or late (8 days to 6 weeks after surgery) [16]. The early type is usually caused by gram-negative bacteria and usually has worse visual acuity at presentation and carries a poor visual prognosis. However, the late type is usually caused by gram-positive bacteria (frequently coagulase-negative staphylococci) and the presenting visual acuity is usually better than the early type with better visual prognosis.

Early diagnosis and prompt intervention of APIE are crucial. The guidelines from Endophthalmitis Vitrectomy Study (EVS) are usually adopted to treat patients with APIE based on the visual acuity on presentation. If the affected eye has a visual acuity of hand motion or better than a tapping-injection procedure is recommended. Pars plana vitrectomy with vitreous specimen collection and intravitreal injection of antibiotics, is recommended for an eye with visual acuity of light perception or worse. According to EVS, tapping of the vitreous yields more positive microbial results than tapping from the anterior chamber. Intravitreal injection of antibiotics should cover both gram-positive and gram-negative bacteria. The most common intravitreal antibiotics being used are vancomycin (1 gm/0.1 ml) and ceftazidime (2.25 mg/0.1 ml).

Figure 1. Acute postoperative (phaco + PCIOL) endophthalmitis secondary to Serratia marcescens. Anterior segment photo of the right eye shows diffuse lid edema, diffuse and severe conjunctival chemosis, diffuse corneal cloudiness, organized hypopyon, iris details not visualized (1a) and B-scan shows diffuse vitreous hype echoic opacities and attached retina (1b).
Amikacin (0.4 mg/0.1 ml) can be used as an alternative for ceftazidime. The visual prognosis for an eye with APIE is promising after early and appropriate intervention. After 9–12 months of follow-up of treated eyes; 53% of eyes had visual acuity of 6/12 or better and only 11% had worse than 6/240 [3].

3. Delayed-onset postoperative infectious endophthalmitis

Delayed-onset postoperative infectious endophthalmitis occurs beyond 6 weeks after the intraocular procedure [3]. It is a synonym that is usually used to describe chronic postoperative endophthalmitis (CPE) [17]. This type of endophthalmitis has a diagnostic and therapeutic dilemma and requires a high index of suspicion because other causes may mimic its presentation. Hence, patients with CPE are frequently treated with corticosteroids due to the masquerading presentation of autoimmune intraocular inflammation. CPE was first described in the 1980s and the microorganisms that are responsible for CPE are somewhat different from that of APIE. They are usually low virulent and indolent. Specific groups of bacteria and fungi are considered to be causative pathogens for CPE (Table 3) [17–21].

It was observed that CPE has a lower incidence rate than APIE. Its incidence is around 0.017% after cataract surgery [20]. Some studies reported a ratio of acute to chronic postoperative endophthalmitis between 5:1 and 2:1 [17].

Propionibacterium acne, which is a gram-positive, non-spore-forming anaerobic bacillus bacteria, and found usually in skin and hair follicles; is considered to be the most common causative pathogen for CPE. Around two-thirds of the CPE, cases are found to have Propionibacterium acne [21].

The clinical features of CPE are less aggressive than that of APIE. It occurs 6 weeks to months after the intraocular procedure. Usually, the affected eye shows a persistent or recurrent low-grade indolent inflammation [22, 23]. The inflammation is often granulomatous with large keratic precipitates. The ocular pain is usually not severe, but reduced vision is common in almost all patients. Hypopyon is not a frequent

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>Fungi</th>
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<tr>
<td><em>Propionibacterium acne</em></td>
<td><em>Aspergillus</em> species</td>
</tr>
<tr>
<td><em>Staphylococcus</em> species</td>
<td><em>Candida</em> species</td>
</tr>
<tr>
<td><em>Corynebacterium</em></td>
<td><em>Curvularia lunata</em></td>
</tr>
<tr>
<td><em>Nocardia</em></td>
<td><em>Fonseca pedrosi</em></td>
</tr>
<tr>
<td><em>Cephalosporium and Acremonium</em></td>
<td><em>Paecilomyces</em> species</td>
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<td><em>Paecilomyces</em></td>
<td><em>Acremonium strictum</em></td>
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<td><em>Ochrobactrum anthropioc</em></td>
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<td><em>Hafnia alvei</em></td>
<td></td>
</tr>
<tr>
<td><em>Sphingomonas paucimobilis</em></td>
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<tr>
<td><em>Mycobacterium chelonae</em></td>
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<tr>
<td><em>Pseudomonas stutzeri</em></td>
<td></td>
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<tr>
<td><em>Achromobacter</em></td>
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Table 3. Microorganisms that are associated with CPE.
finding, but microhypepopyon may present. Other associated clinical features include conjunctival congestion and photophobia. An important feature in CPE is the presence of white plaques in the capsule. These plaques are associated with a different variety of microorganisms that causes CPE and not only P. acne (Figure 2) [20].

Management of CPE is somewhat challenging due to the ambiguous presentation. Many patients with CPE are treated initially with steroids and which may result in a partial response. However; the indolent inflammation persists and recurs after tapering down or discontinuation of the steroids. Furthermore, patients with fungal etiology might get worsening signs if steroid treatment is commenced. There is no standard treatment protocol known for CPE like the one which is known for APIE (i.e., EVS recommendations). However, in real-life practice, treatment may include intravitreal injection of antibiotics alone as an initial treatment or with pars plana vitrectomy (PPV) along with partial or total capsulotomy and removal or exchange of the intraocular lens (IOL) is performed [17]. In addition, sending the IOL with the capsule to a microbiology lab is recommended in the management plan of CPE. The use of empiric intravitreal antibiotics is recommended when treating chronic endophthalmitis. Patients with highly suspected or culture-proven fungal endophthalmitis to be monitored and treated with anti-fungal therapy, such as intraocular amphotericin (5–10 μg in 0.1 ml).

The outcome after treating CPE depends on the causative pathogen. In general, visual acuity of 20/40 or better can be achieved in 29–50% after treatment. Eyes with CPE caused by P. acne or other gram-positive bacteria has a better outcome [17]. However, CPE caused by fungus carries poor visual outcome [20].
4. Bleb-associated postoperative infectious endophthalmitis

Endophthalmitis is a sight-threatening rare complication of glaucoma surgery. Mostly delayed in onset. The rates of endophthalmitis associated with glaucoma filtering surgeries vary with surgical technique and use of antimetabolite agents. A study based on 5-year, retrospective data showed a 0.55% risk of blebitis and 0.45–1.3% risk of endophthalmitis after glaucoma filtering surgery [21]. The tube versus trabeculectomy study (TVT) reported endophthalmitis developed in 1 of 107 in the tube group and 5 of 105 in the trabeculectomy group over five years [22].

Early bleb-associated postoperative endophthalmitis is rare with a reported incidence of 0.1% [22]. Majority of bleb-associated postoperative endophthalmitis occurs months to years after the original procedure.

There are multiple reported risk factors under this entity. Among these are; cystic bleb, which may create direct access of pathogens to the eye either by the high permeability of the cystic wall or by the relatively frequent coexistence of a conjunctival leak [23]. Use of antimetabolites, such as mitomycin C and 5-fluorouracil, changes the thickness, cellularity, and vascularity of the overlying conjunctiva, which increases the risk of developing endophthalmitis. Furthermore, the use of these agents weakens the barrier against the migration of bacteria across the bleb wall. Patients with recurrent or persistent bleb leak are more at risk of developing endophthalmitis [24]. Hence, screening for bleb leakage following each visit is recommended among patients with trabeculectomy surgery. Finally, an inferiorly located bleb is more likely to lead to endophthalmitis than a superiorly located one probably due more to exposure of the bleb to the bacteria-rich tear film [23].

Patients with bleb-associated postoperative endophthalmitis may present with a drop in vision, redness, and eye discharges. It is often preceded by the accelerated prodromal syndrome of brow ache, ocular pain, and headache, which progress rapidly over a short period of few hours [3].

Clinically, the visual acuity is significantly reduced and anterior segment evaluation may reveal discoloration or mucopurulent discharge within the bleb described as a “white on red” appearance against conjunctival erythema. The anterior chamber may show the cellular reaction of cells, flare, and/or hypopyon. Seidel test may identify early bleb leak. Posterior segment evaluation may show vitreous cells and filaments, however, if dense vitritis, fundus view might be obscured, and hence, the B-scan ultrasonography is indicated to confirm vitreous involvement and rule out complications, such as retinal detachment.

True bleb-associated postoperative endophthalmitis must be differentiated from blebitis. In blebitis, there is a chalky white bleb surrounded by conjunctival injection and might be associated with a bleb leak and mild anterior chamber cellular reaction without significant vitritis. The diagnosis is based on a clinical exam and confirmed by standard adequate vitreous or aqueous sample for culture and sensitivity.

The array of organisms associated with bleb-related endophthalmitis differs from those associated with cataract surgery. Bleb-related endophthalmitis is mostly preceded by bleb infection, therefore, clinicians must be more aggressive in the treatment of blebitis. This includes oral and hourly topical fourth-generation antibiotics and daily monitoring for signs of progression, which includes assessment of the bleb, anterior chamber, and anterior vitreous face until a positive response to therapy is observed [25].

Intravitreal antibiotics, the combination of vancomycin (for gram-positive coverage) and ceftazidime (for gram-negative coverage) commonly administered during vitreous
tap & inject. Intravitreal amphotericin B and voriconazole are considered if a fungal infection is suspected and/or confirmed. The use of corticosteroids is controversial since their effect on the visual outcome is not yet known [26].

Following the endophthalmitis vitrectomy study (EVS) guidelines; patients presented with hand motion or better vision may be treated with tap and inject; on the other hand, patients with light perception or worse should be considered for immediate pars plana vitrectomy (PPV) and intravitreal antibiotics. However; the population EVS study did not include patients with post-glaucoma surgery endophthalmitis. Therefore, it might be fair to consider early vitrectomy in patients with more virulent organisms, and it has been shown that vitrectomy in such cases may produce more favorable outcomes [24, 26].

Although bleb-associated endophthalmitis carries a poor visual prognosis; early recognition and treatment are necessary for optimizing the outcome of filtering surgery. Therefore, it is important to inspect the bleb or tube at each visit for any evidence of leak or erosion and if endophthalmitis is suspected, immediate aggressive and appropriate treatment is initiated.

5. Endophthalmitis Post Pars Plana Vitrectomy (PPV)

Endophthalmitis post-PPV is relatively rare. The incidence has decreased from 0.3–0.14% to 0.0180.076% with improvement in VR instrumentations and techniques [27].

There are several reported predisposing factors under this entity; insufficient wound closure, hypotony from a sclerotomy leak, and vitreous incarceration at a sclerotomy site, are some risk factors as it allows migration of microorganisms into the vitreous cavity from the ocular surface [28–30].

Patients may present with symptoms and signs similar to endophthalmitis following cataract surgery in particular hypopyon and dense vitritis (Figure 3).

Vitreous samples should be obtained if endophthalmitis is suspected. A combination of broad-spectrum intravitreal antibiotics is injected intravitreally.

Multiple studies have reported several bacteria under such entity which include coagulase-negative staphylococci, Pseudomonas species, Propionibacterium, enterococci, and Bacillus species. However, coagulase-negative staphylococci are the most common organism [31].

Figure 3.
Intraoperative photos of post PPV endophthalmitis. Intraoperatively photos showing dense vitritis suggestive of aggressive inflammation (3a) and clear vitreous after silicone oil removal (3b). (Courtesy of Dr. Hemant Trehan).
EVS did not enroll patients with post-PPV endophthalmitis, therefore, the results do not directly apply to the management of such entity. Nevertheless, certain principles apply.

On average, about 70% of all samples across studies have shown positive microbiology cultures. In a large multicenter study, Cohen et al. reported 16 culture-positive cases out of 18 cases (89%) of endophthalmitis post-vitrectomy [32].

Despite treatment, the visual outcome varies. As described by Park et al., a significant proportion of cases have poor visual outcomes, with vision ranging from 20/200 to no light perception [33]. However, some cases achieved 20/40 vision [34, 35].

6. Endophthalmitis post-intravitreal injections

The use of intravitreal agents (e.g., Anti-VEGF and steroids) to treat various retinal diseases has increased significantly since the 1990s.

The incidence rate of infectious endophthalmitis after intravitreal injection is low when compared to other post-surgical infectious endophthalmitis, it varies between 0.038% and 0.053% [36].

The most commonly causative pathogens are Streptococcus or Staphylococcus species suggesting the commensal flora of the ocular adnexa and oropharynx as the source. Published reports confirmed that Streptococcus is significantly more frequent after intravitreal injection than after other intraocular surgeries [37, 38].

The use of post-injection antibiotics does not decrease the frequency of subsequent endophthalmitis; however, it may possibly cause drug-resistant bacteria in the nasopharynx [39].

Post-intravitreal infectious endophthalmitis should be differentiated from sterile endophthalmitis, a well-recognized condition that can occur after intravitreal injections. In the latter patients present with painless reduction in vision, no or minimum redness, anterior chamber cells, fibrin, and/or hypopyon. In the MARINA trial, there was a 1% rate of serious inflammation in patients who received intravitreal injection with ranibizumab [40].

In contrary, the presentation post-intravitreal infectious endophthalmitis is more aggressive similar to the presentation seen after other intraocular procedures; this may include significant painful loss of vision, marked anterior chamber cells, fibrinous reaction, hypopyon, and/or vitritis [41].

The outcome is variable. In a large series of post-intravitreal infectious endophthalmitis; most of the eyes that developed (15 of 23) returned to baseline vision within 3 months after treatment and there was no significant difference in the rate of endophthalmitis between the types of anti-VEGF injected [42].

7. Endophthalmitis post-penetrating keratoplasty

Endophthalmitis following penetrating keratoplasty (PK) occurs at a slightly higher rate compared to other intraocular procedures. The incidence ranges from 0.2% to 0.4% [43, 44].

Risk factors include; large circumferential incision, wound dehiscence, suture abscess, and contaminated donor tissue [44]. In addition, chronic use of topical steroids which are known to decrease immune defense may increase the risk of endophthalmitis.
Symptoms are similar to other types of postoperative endophthalmitis. The causative agents are predominantly gram-positive bacteria (Streptococcus and Staphylococcus). On the other hand, gram-negative bacteria account for nearly 20% of cases. The outcomes can be devastating with severe visual loss in 50% of cases and often a requirement of repeat keratoplasty [45]. Overall, the frequency of endophthalmitis in modern corneal transplant surgery is expected to decrease significantly with the advancement in lamellar keratoplasty. In a study conducted by Heinzelmann et al., it was apparent that the outcome of lamellar endothelial keratoplasty and Descemet membrane endothelial keratoplasty is superior to PK without serious complications, such as exogenous endophthalmitis [46].

8. Post-traumatic infectious endophthalmitis

Post-traumatic endophthalmitis is an uncommon yet devastating complication of open globe injuries. It has a poorer outcome compared to postoperative endophthalmitis due to concomitant ocular tissue damage, presence of more virulent pathogens, and possibly due delay in diagnosis and treatment. The reported incidence of infectious endophthalmitis following open globe injuries ranges from 3.1% to 11.9% of open globe injuries in the absence of an intraocular foreign body (IOFB) [47–52]. The incidence increases from 3.8% to 48.1%, in the presence of an IOFB, with higher infection rates with retained IOFBs contaminated with organic matter from a rural setting [53–57].

The following factors are associated with an increased risk of post-traumatic endophthalmitis: intraocular foreign bodies (IOFB) [58–62], violation of the lens capsule [58, 60–62], contamination of the wound [59, 61] and delayed primary repair [58, 61, 63]. Interestingly, the existence of hyphema or iris prolapse was associated with lower rates of endophthalmitis [64].

Recognizing symptoms of post-traumatic endophthalmitis might be challenging due to the presence of injury-induced inflammation and the disruption of ocular structures [65]. Pain and decreased visual acuity often occur with ocular injury with or without infection. Presence of worsening symptoms of photophobia, tearing and pain are suggestive of infectious endophthalmitis. Pain from ocular injury can be distinguished from that of endophthalmitis if it is progressive and out of proportion to the degree of injury [66–70]. Inflammatory signs are almost similar to other types of endophthalmitis (i.e., eyelid edema, chemosis, corneal edema, hypopyon, variable degree of vitritis, etc.), however, purulent discharge from the site of injury is a feature of post-traumatic endophthalmitis. Inflammation that progresses slowly following primary repair may be indicative of fungal endophthalmitis [67, 69].

The broad spectrum of pathogens causing post-traumatic endophthalmitis, with gram-positive cocci the most frequently identified causative organism, followed by Bacillus species, fungi, and mixed infections. In a large cohort of post-traumatic endophthalmitis cases, Long et al. have reported that 38.1% of cases of post-traumatic endophthalmitis were culture-positive, and 3.2% showed mixed infections (gram-negative bacilli and fungi). Culture proven pathogens included gram-positive cocci (41.9%), gram-negative bacilli (29.1%), gram-positive bacilli (12.3%), and fungi (16.8%). In the same reported series, the coagulate-negative staphylococcal (CNS) species S. epidermidis (21.8%) and S. saprophyticus (12.0%) were the predominant pathogens, followed by Bacillus subtilis (8.7%), Pseudomonas aeruginosa (7.8%), and...
and Escherichia coli (6.4%). Delayed repair over 24 hours and metallic injury were significantly associated with a positive culture of CNS. The most frequent fungal species were Aspergillus (33%), followed by yeast-like fungi (30%) [51]. In another cohort of post-traumatic endophthalmitis; Clostridium perfringens were isolated in 16.6% of culture-positive cases [71].

To reduce the risk of post-traumatic endophthalmitis, it is widely accepted common practice to immediate initiation of empirical prophylactic systemic antibiotics (either intravenous or oral administration) at initial presentation for all open globe injuries and an injection of intravitreal antibiotics at the time of primary repair for high-risk patients [72–75].

In a meta-analysis report, it has been noted that the incidence of endophthalmitis post open globe injuries has reduced significantly when intravitreal/intracameral antibiotics were used intraoperatively without additional benefit on the final visual outcome [76].

The threshold for vitrectomy in cases of post-traumatic endophthalmitis is low. Vitrectomy should be performed if there is no response to intravitreal antibiotics within 24 hours or if associated with complications like retinal detachment or intraocular foreign body (Figure 4).

In general, the visual prognosis of post-traumatic endophthalmitis is worse than that of post-operative endophthalmitis [77, 78]. The virulence of the microorganisms in post-traumatic endophthalmitis, for example, Bacillus cereus carries a very high risk of progressing to a final visual acuity of NLP [79]. A delay in diagnosis and initiation of appropriate therapy is an important risk factor that contributes to poor visual prognosis. Moreover, the presence of an afferent pupillary defect, perforating injury, expelled lens, corneoscleral wound (vs corneal wound), and retinal detachment at the time of ocular injury is associated with a poor visual prognosis even in the absence of endophthalmitis [80].

9. Endogenous endophthalmitis

Endogenous endophthalmitis (EE), also called metastatic endophthalmitis, occurs as a result of the hematogenous spread of microorganisms from the body to the eye. The microorganism primarily spreads through the posterior segment vessels. The right
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Eye is more commonly affected due to dominant and direct blood flow from the right carotid artery, however, approximately 25% of cases have a bilateral presentation [81]. The reported incidence of EE is 2 to 8% [82, 83]. However, it has been reported that the causative pathogens have geographical variations. They can be fungal or bacterial, with fungal pathogens being the commonest [82]. According to reported cases from Asia, fungal EE accounts for nearly 11.1 to 17.54% of total cases [84]. In bacterial EE, gram-positive streptococcus and staphylococcus are the most common pathogens. Gram-negative bacterial EE, however, is more common in Asian countries [85]. Among fungal species, Candida albicans account for many cases being the most common yeast, and among the molds, Aspergillus flavus is the commonest [86]. The risk factors for EE are primarily systemic conditions or medications which reduce immunity. Among these are diabetes mellitus, malignancies, asplenia, cardiac or renal transplant, and severe infections (e.g., pneumonia, urinary tract infection, vertebral osteomyelitis, liver abscess, and acquired immune deficiency syndrome), chronic alcoholism, intravenous catheters, and drug abuse and long-term use of steroids or other immunosuppressants [87–90].

The patients with the aforementioned risk factors and associated with ophthalmic symptoms require a thorough ophthalmic assessment to rule out EE. Such symptoms vary from eye pain, redness, photophobia, blurred vision, floaters, and flashes. The clinical signs are similar to other types of endophthalmitis, however, in EE the vitreous involvement, for example, vitreous haze, vitreous cells and floaters as well as subretinal membranes and exudates are the most important associated findings to look for [91, 92]. Aspergillus flavus is known to cause yellow/white exudates in the vitreous, which vary from focal to diffuse. The hallmark feature of Candida EE is the presence of fluffy cotton wool-like white retinal exudates or colonies along with vitritis (Figure 5) [92].

The management of EE should be focused on systemic evaluation and management of underlying causes. In addition to clinical evaluation, a B scan ultrasound may delineate choroidal abscess, which appears as a dome-shaped elevation on B scan similar to choroidal detachment [93].

Blood cultures and urine cultures are important adjuvant diagnostic modality in the diagnosis of possible systemic infection. It was reported that blood culture has shown a higher culture positivity than the vitreous sample, probably due to the...
sample volume obtained. Moreover, culture at extraocular sites has yielded a 21–100% positivity rate as per previous reports [94].

Medical management includes topical and systemic antibiotics or antifungals, intravitreal antibiotics or antifungals, and pars plana vitrectomy (PPV). PPV is recommended for non-resolving vision-threatening EE cases. PPV serves both diagnostic and therapeutic purposes. Sato et al. suggested early vitrectomy in Candida EE cases [95]. Yoon et al. suggested early PPV in Klebsiella endophthalmitis, which may result in better visual effects [96]. The collaboration of the treating ophthalmologist or vitreoretinal surgeon, microbiologists, pathologists, and the critical care physician plays a vital role in determining the patient's final systemic and ocular outcome [82].

Prognosis of EE similar to other endophthalmitis depends on the duration of the condition, the extent of the ocular structures involved, the virulence of the causative agents, and the timing of initiation of treatment as well as the response to the treatment. Reported studies have shown that yeasts have a better prognosis, followed by bacteria followed by molds, which have the worst prognosis.

10. Conclusion

Endophthalmitis is a devastating ophthalmic emergency that may lead to total loss of vision and possibly the eye if it is not recognized and managed promptly. In addition to eye caregivers, patients undergoing intraocular surgeries must be counseled about this rare condition as well as the potential further sequela that might occur with or without treatment are critical. With the advancements in VR instrumentations and techniques, the threshold of diagnostic and therapeutic PPV and appropriate intravitreal antibiotics and/or antifungals in the management of endophthalmitis is very low. Finally, frontline health care providers must be aware and critical if they encounter patients with suspicious of endophthalmitis as early recognition, prompt referral, and timely treatment are very crucial aspects for better visual prognosis.

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Author details

Mohamed Al-Abri*, Ahmed Al-Hinai and Nawal Al-Fadhil
Department of Ophthalmology, Sultan Qaboos University Hospital, Muscat, Sultanate of Oman

*Address all correspondence to: msalabri@squ.edu.om

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