

# Postsurgical Endophthalmitis Clinical Presentation And Management

Essay

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Of Master Degree In Ophthalmology

By

Hanan Said Mahmoud  
M.B.B.CH., Faculty of Medicine  
Ain Shams University

Supervised by

Prof. DR /Ahmed Ibrahim Abo Elnaga  
Professor of ophthalmology  
Faculty of medicine  
Ain Shams University

DR/ Raafat Ali Rihan  
**Assistant Professor of ophthalmology**  
Faculty of Medicine  
Ain Shams University

Cairo  
Faculty of medicine  
Ain Shams University, 2006

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## List of Abbreviations

AIDS	Acquired immunodeficiency syndrome.
AOPE	Acute-onset postoperative endophthalmitis.
CPE	Chronic postoperative endophthalmitis.
CSF	Cerebrospinal fluid.
CT	Computed tomography.
ECCE	Extracapsular cataract extraction.
E V S	Endophthalmitis Vitrectomy Study.
FBE	Filtering bleb- associated endophthalmitis.
FUO	Fever of unknown origin.
GMS	Gomori-methenamine-silver.
ICCE	Intracapsular cataract extraction.
IIE	Intravitreal injection-associated endophthalmitis.
IOL	Intraocular lens.
IOP	Intraocular pressure
I.V	Intravenous.
LASIK	Lasre in-situ keratomileusis.
mg	Milligram.
MVR	A blade used for sclerotomies.
NSAID	Non-steroidal Anti-inflammatory drugs.
NTM	Non-tuberculous mycobacteria.
PAS	Periodic acid schiff.
PMMA	Polymethylmethacrylate.
PO	Per oral.
PPV	Powerful predicting visual outcome.
PVD	Posterior vitreous detachment.
UGH	Uveitis-Glaucoma-Hyphema syndrome.
Vs	Versus.
YAG	Neodymium yttrium-aluminium-garnet.

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## INTRODUCTION

Endophthalmitis is the term used for severe inflammation of the intraocular structures always involving the ocular fluids (either the vitreous, the aqueous humor or both). The cause of inflammation may be infectious or non-infectious. Non-infectious (sterile) endophthalmitis may result from various causes such as retained native lens material after an operation or from toxic agents. Panophthalmitis is inflammation of all coats of the eye including intraocular structure. (David, 2002)

Endophthalmitis is one of the most devastating eye complications that can occur following intraocular surgery. If the outcome is to be successful, it is essential that the diagnosis is recognised and not denied. Once the diagnosis is made, treatment should begin without delay to avoid further erosion of visual acuity. (Alfonso et al, 2005)

The two types of endophthalmitis are endogenous and exogenous. Endogenous endophthalmitis results from the hematogenous spread of organisms from a distant source of infection (e.g. endocarditis). Exogenous endophthalmitis results from direct inoculation as a complication of ocular surgery, foreign bodies, and/or blunt or penetrating ocular trauma. (Albert and Jakobiec, 2000)

Endophthalmitis is one of the most serious complications of cataract surgery. In the US, incidence after intraocular surgery is less than 0.1%. Incidence of culture-proven endophthalmitis is similar to that of extracapsular cataract extraction and phacoemulsification. Another recent report reviewed over 9,000 phacoemulsification procedures at a single eye center from (1997–2001) and found an incidence of endophthalmitis of one per 378 cases (0.26%). The majority of cases of postcataract extraction endophthalmitis show acute to subacute manifestations and develop within 6 weeks of surgery. Ninety percent of all postoperative cases of endophthalmitis are seen after cataract surgery. (Bernard et al, 2004)

If not properly treated, a risk of complete vision loss and the possibility of persistent ocular pain exist. Infection very rarely spreads beyond the confines of the sclera and tracks into surrounding tissue structures. (Alfonso et al, 2005)

The most common organism in acute onset endophthalmitis is Coagulase-negative organisms such as Staphylococci and Streptococcus, on the other hand, in delayed type Propionibacterium acnes, and Corynebacterium species or fungal, the common fungi are Candida and Aspergillus. **(Alfonso et al ,2005)** .

Postoperative endophthalmitis may also occur weeks to years following surgery. This delayed infection is likely due either to sequestration of low-virulence organisms introduced at the time of surgery or to delayed inoculation of organisms. In the former case, Propionibacterium acnes is the most common microorganism encountered, and clinically evident low-to moderate-grade inflammation may occur weeks to months after surgery .**(Samson and Foster, 2000)** .

Risk factors for development of postoperative endophthalmitis may include the following: uncontrolled diabetes , poor healing of the corneal wound , pre-existing local conditions such as chronic blepharitis or conjunctivitis, increased operative time, posterior capsule rupture/vitreous loss , retained lens fragments, inadequate sterilization of the operative field , contamination of surgical instruments .**(Raskin et al , 1993)** .

The main presenting symptoms are pain , defective vision, swelling or redness of the eye lids and discharge following surgery. Signs include: visual acuity decreased below the level expected, lid edema, conjunctival hyperemia, corneal oedema, flare and cells, keratic precipitates, hypopyon, fibrin clot in the anterior chamber, fibrin membrane formation, vitreous cells or abscess may be seen, loss of red reflex and retinal periphlebitis if view of fundus possible. When the signs are pronounced, the eye should be treated as if it were infected. **(Endophthalmitis Vitrectomy Study, 1995)** .

Preoperative meticulous examination of the eye , treatment of any local cause and infection elsewhere: (blepharitis, dacryocystitis, conjunctivitis, contralateral ocular prosthesis), prolonged use of topical corticosteroids and chronic urinary tract infection should caution the physician while planning any kind of intraocular surgery. Preparation of operative field with 5% povidone-iodine solution , meticulous draping technique to cover lashes and lid margins and prophylactic antibiotics are the main lines to decrease incidence of endophthalmitis. **(Paymen and Daun , 1994)** .

Accurate diagnosis of postoperative endophthalmitis can be confirmed by staining and culturing the pathogenic organisms present in the eye. Diagnosis by polymerase chain reaction analysis is a recent advancement, this method has a higher sensitivity and shorter detection time than conventional staining and culture. In case of acute postoperative endophthalmitis, both aqueous and vitreous humours should be cultured. For chronic postoperative endophthalmitis, all intra-ocular plaques should be cultured and stained. **(Sharma et al , 2002)** .

The Endophthalmitis Vitrectomy Study (EVS) identified that the use of periocular and intravenous antibiotics have no role in endophthalmitis following cataract surgery. The goals of pharmacotherapy are to eradicate the infection, to reduce morbidity, and to prevent complications. Intravitreal injection is the most effective route. Corticosteroids are important adjuvants to antibiotic therapy and vitrectomy in the management of endophthalmitis. Surgery may be needed to remove infected tissue from inside the eye, which may improve the chances of stopping the infection. **(Morlet et al , 2005)**.

Vitrectomy must be performed urgently except in the delayed onset category. Immediate vitrectomy is recommended in severe cases, cases in which gram-negative organisms are seen on a smear examination of vitreous aspirate and in cases showing no response to medical treatment (intravitreal injection). **( Pasricha ,2002)** .

Prognosis according to EVS, the visual outcomes of the patients with acute onset post-operative endophthalmitis were excellent and post-operative endophthalmitis is associated with a better visual prognosis than traumatic endophthalmitis. **(Endophthalmitis Vitrectomy Study , 1995)** .



## Epidemiology

Postoperative endophthalmitis is the most frequent category, accounting for more than 70% of cases. In a nosocomial survey (1995–2001) of 35,916 intraocular surgical procedures performed at a university-based hospital, acute-onset endophthalmitis occurred in 17 cases (0.05%). In this survey, the rates of endophthalmitis were highest after secondary intraocular lens implantation (1 of 485 cases; 0.2%) and glaucoma surgery (4 of 1970 cases; 0.2%), and lowest after pars plana vitrectomy (2 of 7429 cases; 0.03%). **(Doft , 1997 and Eifrig et al , 2002).**

In the endophthalmitis vitrectomy study (EVS), from 293 culture-positive identifications, gram-positive bacteria were isolated from 274 eyes (93.5%) and gram-negative bacteria from only 19 eyes (6.5%). The bacteria most often isolated were *Staphylococcus epidermidis* (about 40% of cases), followed by *Staphylococcus aureus* (about 20%) and *Streptococcus* sp. (about 15%). Among the gram-negative bacteria, *Pseudomonas* sp. and *Haemophilus influenzae* were most frequently isolated. Other isolated gram-negative bacteria were *Aerobacter* sp., *Proteus* sp., *Klebsiella* sp., *Escheria coli*, *Bacillus* sp. and *Enterobacter* sp. **(Irvine et al , 1992).**

The incidence of endophthalmitis was the same after extracapsular cataract extraction or phacoemulsification. Cataract surgery accompanied by anterior vitrectomy increased the one month risk for developing endophthalmitis to 0.41%, (more than a four fold increase) over that for cataract surgery without anterior vitrectomy. **(Powe et al , 1994).**

In the US: Postoperative endophthalmitis remains a rare complication of intraocular surgery. Of the 41,654 patients undergoing cataract extraction at the Bascom Palmer Eye Institute (BPEI) from 1984-1994, 34 (0.08%) developed endophthalmitis. During the same period at BPEI, the incidence of endophthalmitis was 0.37% after secondary intraocular lens (IOL) implantation, 0.05% after pars plana vitrectomy, 0.18% after penetrating keratoplasty, and 0.12% after glaucoma filtering surgery. **(Aaberg et al , 1998).**

Postoperative endophthalmitis has been reported following nearly every type of ocular surgery. It occurs most frequently following cataract surgery, the most commonly performed type of ocular surgery. The overall incidence

of post-cataract surgery endophthalmitis in the United States, using modern techniques of phacoemulsification and intraocular lens implantation, is about 0.1% .The incidence following other types of intraocular surgery has been reported to range between 0.05 to 0.37% . In general, those procedures with a higher risk for acute postoperative endophthalmitis (secondary intraocular lens implantation and penetrating keratoplasty [corneal transplantation]) are those with a greater potential for wound leaks with subsequent intraocular bacterial contamination .(**Schmitz et al, 1999**)

In the US: Endogenous endophthalmitis is rare, occurring in only 2-15% of all cases of endophthalmitis. Average annual incidence is about 5 per 10,000 hospitalized patients. In unilateral cases, the right eye is twice as likely to become infected as the left eye, probably because of its more proximal location to direct arterial blood flow from the right innominate artery to the right carotid artery. Since 1980, candidal infections reported in IV drug users have increased. The number of people at risk may be increasing because of the spread of AIDS, more frequent use of immunosuppressive agents, and more invasive procedures (eg, bone marrow transplantation). Most cases of exogenous endophthalmitis (about 60%) occur after intraocular surgery. In the United States, postcataract endophthalmitis is the most common form with approximately 0.1-0.3% of operations having this complication, which has increased over the last 3 years. (**Albert and Jakobiec , 2000**).

The incidence of this complication at one major ophthalmology center between 1995 and 2001 was .04% and after secondary lens implantation was 0.2%. Another recent report reviewed over 9,000 phacoemulsification procedures at a single eye center from 1997–2001 and found an incidence of endophthalmitis of one per 378 cases (0.26%). The majority of cases of postcataract extraction endophthalmitis show acute to subacute manifestations and develop within 6 weeks of surgery. Ninety percent of all postoperative cases of endophthalmitis are seen after cataract surgery. (**Eifrig et al , 2003**)

Post-traumatic endophthalmitis occurs in 4-13% of all penetrating ocular injuries. Incidence of endophthalmitis with perforating injuries in rural areas is higher when compared with nonrural areas. Delay in the repair of a penetrating globe injury is correlated with increased risk of developing endophthalmitis. Incidence of endophthalmitis with retained intraocular foreign bodies is 7-31%. (**Alfaro et al, 1994**) .

### **Source of infection and risk factors:**

Potential sources of infection include contaminated instruments or irrigation solutions, but the most common source is the patient's own ocular flora. Micro-organisms from the conjunctival sac can enter the eye during or after surgery through a wound. There are many risk factors underlying postoperative endophthalmitis. Preoperative risk factors include blepharitis, lacrimal duct obstruction, the wearing of contact lenses, the existence of an ocular prosthesis in the other eye and secondary intra-ocular lens implantation. Intra-operative risk factors include inadequate eyelid or conjunctival disinfection, surgery lasting more than 60 minutes, loss of vitreous humour, use of a prolene haptic intra-ocular lens and unapparent or unplanned ocular penetration. Postoperative risk factors include wound abnormalities, inadequately buried sutures, suture removal, vitreous incarceration in the surgical wound and the presence of a filtering bleb. Furthermore, clear corneal incisions during cataract surgery impart a higher risk of infection than scleral tunnel incisions, perhaps because a stable, self sealing incision is more difficult to construct in the cornea than in the sclera. The incision site of clear corneal incisions in the temporal region may also play a role. (**Chung and Lam , 2004**).

### **Demography**

No sexual predilection exists. , No racial predilection exists, No age predilection exists. (**Endophthalmitis Vitrectomy Study Group, 1996**)

**Predisposing factors:** risk factors that may increase the risk of endophthalmitis include rupture of the posterior capsule, retained lens material, and surgical procedure. Published studies have demonstrated an increased risk of endophthalmitis after placement of a secondary intraocular lens, possibly due to increased surgical time or ocular manipulation. Prolene haptic sutures also have been implicated as a possible risk factor for the development of endophthalmitis due to the surface properties of the material. Other factors that predispose to postoperative inflammation include past history of uveitis, problems with IOL implantation, pseudo-exfoliation syndrome, pigment dispersion during surgery and the use of intra-ocular miotic. (**Aaberg et al , 1998**).

## **Diabetes and Endophthalmitis:**

Diabetics were twice as likely to have growth than nondiabetics at 33% versus 15% according to studies. This probably reflects a more permissive environment for growth in diabetic eyes so that low virulence organisms when present in diabetics tend to grow in higher concentrations in the eye, thus being more readily detected at culture. In the nondiabetics these same cases may well end up as no growth or equivocal growth. Diabetics were more likely to have growth of gram-positive coagulase negative organisms than nondiabetics. Diabetics were slightly more likely to show growth of the most virulent organisms in endophthalmitis, the 'other gram positive organisms' group, which consisted of Strep, S. aureus. However there was almost no difference in frequency of gram-negative cultures between diabetic and nondiabetic patients. (Doft et al, 2001).

**Mortality/Morbidity:** Fortunately, postsurgical endophthalmitis, unlike endogenous endophthalmitis, rarely causes any extraocular complications. Rarely, untreated cases can lead to late panophthalmitis and orbital cellulitis, prompting need for enucleation. Morbidity associated with postoperative endophthalmitis can be substantial and is related not only to the acute process but to late sequelae. In general, the risk of severe visual loss in patients with acute endophthalmitis is higher in patients who develop infections from more virulent organisms and do not seek treatment promptly. Fortunately, 70-80% of patients with postoperative endophthalmitis have infections caused by coagulase-negative staphylococci, and the visual prognosis in these cases is usually good with rapid treatment. (Aaberg et al, 1998).

## Pathogenesis

### Pathophysiology:

Under normal circumstances, the blood-ocular barrier provides a natural resistance against invading organisms. In endogenous endophthalmitis, blood-borne organisms permeate the blood-ocular barrier either by direct invasion (i.e. septic emboli) or by changes in vascular endothelium caused by substrates released during infection. Destruction of intraocular tissues may be due to direct invasion by the organism and/or from inflammatory mediators of the immune response. Endophthalmitis may be as subtle as white nodules on the lens capsule, iris, retina, or choroid. It can also be as ubiquitous as inflammation of all the ocular tissues, leading to a globe full of purulent exudate. In addition, inflammation can spread to involve the orbital soft tissue. Any surgical procedure that disrupts the integrity of the globe can lead to exogenous endophthalmitis (eg. cataract, glaucoma, retinal, radial keratotomy). **(Albert and Jakobiec , 2000).**

The majority of cases of acute postoperative endophthalmitis (88%) appear within the first 7 days after the intervention. Delayed –onset postoperative endophthalmitis is diagnosed when signs of endophthalmitis are observed 4 weeks or more after surgery . The timing of clinical signs may give some clues to the causative agents.**(David 1, 2002).**

Infectious agents gain access into the eye during surgery or postoperatively through incision sites. These infectious agents may be cleared from the intraocular cavity or may induce a local inflammatory response culminating in endophthalmitis. The risk of developing endophthalmitis depends on a variety of factors including the size of the inoculum, virulence of the organism, access to the vitreous, and perioperative antibiotic use. The avascular vitreous appears to be a better culture medium than the aqueous. Proliferation of the infectious agents results in an inflammatory response from the surrounding vascular structures and breakdown of the blood-aqueous and blood-vitreous barriers. A well-formed vitreous gel and an intact anterior hyaloid or lens capsule may initially restrict the extension and spread of inflammation and exudates . In general, bacterial infections tend to cause diffuse liquefactive necrosis of the vitreous and proliferate rapidly. In acute bacterial infections there occurs invasion of polymorphonuclear cells that are seen in varying stages of degeneration, a hallmark of suppurative necrosis.**(Green , 1984).**

Most of the cases of postsurgical endophthalmitis are seen following cataract surgery. Changes in the surgical techniques for cataract removal have altered the pathogenesis of postsurgical endophthalmitis. The initial technique of intracapsular cataract extraction (ICCE) resulted in a direct communication between the anterior chamber and vitreous cavity with only an intact anterior hyaloid to prevent intraoperatively introduced microorganism from extending into the vitreous cavity. Retention of the posterior capsule following extracapsular cataract extraction (ECCE) while being associated with a reduced incidence of endophthalmitis has its own associated unique problems. Organisms of low virulence such as *Propionibacterium acnes* can become sequestered in the retained capsular sac resulting in chronic localised endophthalmitis. (Javitt et al, 1991).



**Figure 1:** Postsurgical endophthalmitis after ECCE and posterior chamber IOL implantation fibrin and cells on the lens.

*Online Journal of Ophthalmology, 2004*

The intraocular lens implantation (IOL) implantation has also increased the potential for postsurgical inflammation due to the IOL acting as an intraocular foreign body. Increased intraocular manipulations, ineffective sterilization techniques and chronic irritation of the iris and ciliary body are some other factors contributing to postsurgical inflammation. In addition, the electrostatic forces on the IOL may attract microorganisms from the periocular tissues during insertion. The IOLs, particularly those with polypropylene haptics seem to be associated with an increased risk of endophthalmitis. The IOL haptics may become embedded in the surrounding tissues. Histologically,

this erosion appears to be related to the size of the lens and elasticity of the haptics, rather than duration of the implant. The newer technique of sutureless wound closure leaves a potential route for entry of microorganisms. However, this technique has not proven to be associated with increased risk of postsurgical endophthalmitis.(Menikoff,1991).

In large clinical series, gram-positive organisms caused 56-90% of all cases of endophthalmitis. The most common organisms are Staphylococcus epidermidis, Staphylococcus aureus, and Streptococcus species. Gram-negative organisms like Pseudomonas, Escherichia coli, and Enterococcus are observed in penetrating injuries.(David 1, 2002).



**Figure 2:** Postsurgical pneumococcal endophthalmitis.  
*Online Journal of Ophthalmology, 2004*

Streptococci are gram-positive cocci occurring in chains or pairs. All are catalase-negative (degrade hydrogen peroxide, H<sub>2</sub>O<sub>2</sub>). Identification is based mainly on the type of hemolysis of red blood cells in agar. Alpha hemolytic streptococci only incompletely hemolyze red blood cells and include *S. pneumoniae*, and *S. viridans*. Beta hemolytic streptococci completely hemolyze red blood cells and form a green zone around colonies in agar. Beta hemolytic streptococci include enterococci (*S. faecalis*), *S. agalactiae*, and *S. pyogenes*. Staphylococci are gram-positive cocci occurring in clusters. All produce catalase, unlike streptococci. *S. aureus* and *S. epidermidis* are the most common ocular pathogens. The drug of choice for these strains is a B-lactamase resistant penicillin, such as methicillin. However, methicillin-resistant strains have developed and vancomycin remains the drug of choice, even though in 1997, vancomycin-resistant strains were isolated. Of the remaining gram-positive organisms, propionibacterium and bacillus are also found in post-operative endophthalmitis. Propionibacteria are pleomorphic,