

Endogenous Panophthalmitis: The Traitor Within!

SharanyaSukumar¹, Uma Kulkarni²

¹(Department Of Ophthalmology, Yenepoya Medical College Hospital, Yenepoya University, Mangalore, Karnataka)

²(Department of Ophthalmology, Yenepoya Medical College Hospital, Yenepoya University, Mangalore, Karnataka)

Abstract:

Aim: To report a rare case of endogenous panophthalmitis in a diabetic with chronic kidney disease undergoing haemodialysis.

Methods: A 54 year old male diabetic and hypertensive with chronic kidney disease (CKD) on maintenance haemodialysis through a tunnelled internal jugular vein (IJV) catheter with IJV catheter infection was being treated with parenteral Imipenam. He complained of sudden pain in the left eye with discharge and difficulty in opening the eyelids since 2 days. Examination revealed tense oedematous eyelids, purulent discharge, chemotic congested conjunctiva, hypopyon, proptosis, restriction of ocular movements and progressive diminution of vision. A clinical diagnosis of endogenous panophthalmitis was made and was supported by investigations.

Result: With uncontrolled diabetic status, despite intensive systemic antibiotics the condition progressed rapidly. The patient was treated, high local and systemic antibiotics and maintenance haemodialysis. Evisceration was inevitable.

Conclusion: Endogenous panophthalmitis is rare. Early intervention is the key to preventing visual loss, but prognosis is poor. Management is challenging in the presence of multiple risk factors.

Key Words: Endogenous panophthalmitis, gram negative organism, haemodialysis, IJV catheter

I. Introduction

Endophthalmitis is a serious ocular condition with an incidence of 0.09% in India.^[1] It refers to the inflammation in the anterior and/or posterior segment of the eye, which may be concurrent with only partial-thickness involvement of the adjacent ocular wall.^[2] Although it is attributable to bacterial or fungal infections following exogenous or endogenous spread, cases of sterile endophthalmitis^[3] are described in which infection is suspected, but return negative culture results.

Exogenous endophthalmitis^[4] results from the spread of causative organisms from surgical or non-surgical wounds. Spread through surgical wound occurs following cataract surgery, glaucoma filtering surgery, pars plana vitrectomy, penetrating keratoplasty or pneumatic retinopexy. Postoperative endophthalmitis has a reported incidence in the range of 0.04% to 0.41%.^[1]

Non-surgical causes occur following penetrating ocular trauma and constitute about 2-7% of cases.^[5] Incidence of posttraumatic endophthalmitis can be up to 30% when associated with intraocular foreign body and in rural setting.^[1] Studies reported that among patients treated with vitrectomy and intraocular antibiotics for endophthalmitis, 48% achieve final visual acuities better than or equal to 20/400 and in those treated with parenteral, topical and subconjunctival antibiotics alone, 38% achieve the same final visual acuity.^[6]

Endogenous (metastatic endophthalmitis) is rare and constitutes 2 to 6%^[7] of all cases of endophthalmitis. It results from haematogenous spread of pathogens in patients with extra-ocular foci of infection. It is associated with immunocompromised states like diabetes mellitus, human immunodeficiency viral infection and systemic chemotherapy,^[8] debilitating diseases like systemic malignancy; invasive procedures such as extensive gastrointestinal surgery, endoscopy and dental procedures. According to studies, 53.1%–63.6% of endogenous endophthalmitis cases result in no light perception, phthisis bulbi, and evisceration or enucleation, and in 69%, the final visual acuity is worse than counting fingers.^[9] Endogenous endophthalmitis may require surgical intervention in the form of vitreal, retinal, and/or choroidal biopsies and culture if there is no obvious primary source and blood cultures and other studies are negative.^[2]

Panophthalmitis is a more extensive ocular inflammation with involvement of all coats of the eye including sclera and extending to the orbit as well.^[10] It presents with marked lid oedema, proptosis, hypopyon, limitation of ocular movements and a high intraocular pressure, eventually leading to loss of vision. Depending on the virulence of the pathogen and the systemic status of the patient, panophthalmitis may be life threatening. The mortality rate due to this condition is 30%–50% since it usually develops in chronically ill patients.^[11]

1.1. Causative organisms

Gram positive bacterial endogenous endophthalmitis is known to occur following endocarditis, cutaneous infections and intravenous drug use, whereas gram negative cases are known to be associated with enteric infections, liver abscesses and meningitis. The common organisms include streptococci, staphylococci, bacillus cereus, propionibacterium acnes, E. coli, Klebsiella and Neisseria and pseudomonas.^[7] The common fungi which cause endogenous endophthalmitis are Candida, Aspergillus, Cryptococcus, Histoplasma and Coccidioides. Fungal endophthalmitis is reported to have the worst visual prognosis.^[12]

1.2 Prognosis

Endogenous endophthalmitis is associated with poor visual outcome as compared to exogenous. About 70% of the cases with endogenous endophthalmitis have a final visual acuity less than counting fingers, whereas 70% cases of exogenous endophthalmitis have a final visual acuity better than 6/6.^[13] The poor visual outcome is attributed to the delay in diagnosis, use of inappropriate antibiotics, diffuse infection of the vitreous and retina, panophthalmitis or virulent and Gram negative infection.^[9,14] It is also associated with a significant mortality rate of 5-10%,^[7,13] whereas endogenous endophthalmitis is not associated with mortality.^[13]

II. Materials and methods

2.1. Clinical history

A 54 year old man was referred to the department of ophthalmology from the nephrology unit for complaints of sudden onset left ocular pain of severe intensity, purulent ocular discharge and difficulty in opening the left eyelids. It was associated with fever and malaise. The symptoms rapidly progressed over the next two days. He denied any visual complaints prior to this presentation. He was a poorly controlled diabetic since 2 years and a hypertensive since 2 years. He was diagnosed to have CKD and was on maintenance hemodialysis through a tunnelled IJV catheter since 3 years. He was presently hospitalized for the management of IJV catheter infection and was receiving intravenous Imipenam 500mg QID. There was no history of ocular surgery or other ocular morbidity in the past.

2.2. Examination

He was conscious, well oriented, febrile but appeared ill. The left eyelids were oedematous, tense, erythematous, and tender with a local rise in temperature. There was purulent discharge along the lid margin. The left eye showed a relative proptosis of 3mm with grossly restricted and painful ocular movements in all directions. The conjunctiva was severely chemosed and congested and obscured the view of the sclera. Cornea appeared slightly hazy. A dense hypopyon of 4mm height was present (Fig.1) The pupil was mid-dilated with a sluggish reaction to both direct and consensual light. Lens appeared clear. Visual assessment revealed only perception of light with faulty projection of rays. The intraocular pressure appeared to be high. A detailed examination of the right eye revealed a normal anterior segment, no signs of diabetic retinopathy, no evidence of exudation or choroiditis. BCVA was 6/9.

2.3. Investigations

Ultrasound B-Scan of the left eye showed vitreous detachment, extensive retino-choroidal thickening with four large pockets of hyper echoic areas which appeared as localized moundings, in the equatorial region and an 'extensive' T sign. (Fig.2)

Gram staining of the ocular discharge on smears revealed gram negative bacilli. Blood culture showed gram negative bacilli and microbiological culture of the ocular discharge revealed gram negative bacilli, i.e., Enterobacteriaceae species. The organisms were sensitive to Gentamycin, Imipenam and Amikacin and resistant to other antibiotics. Blood investigations showed increased total WBC count (19.7 cells/mm³), blood urea (48mg/dl) and serum creatinine (3.2mg/dl) were increased. His blood sugar levels were maintained within normal limits (87-128mg/dl) during hospital stay with insulin therapy.

2.4. Diagnosis

A clinical diagnosis of panophthalmitis was made assuming an endogenous etiology secondary to the IJV catheter infection confirmed on culture of blood and ocular discharge.

2.5. Management

Intravenous Imipenam 500mg QID was continued and intravenous Gentamycin 80mg BID and oral Acetaminophen 650mg SOS were started. Intensive topical antibiotic medication with Ciprofloxacin and fortified Tobramycin was instituted. Antidiabetic medication was continued and blood sugars were fluctuating from normal to moderately high.

2.6. Course of the disease

The ocular condition rapidly worsened over the next 24 hours; proptosis, lid oedema and ocular pain increased in severity and perception of light became negative. Fever persisted.

III. Result

In view of rapid progression of inflammation to the orbit and the risk of its complications despite intravenous antibiotics in a diabetic patient, the option of evisceration was considered and discussed by the team of ophthalmologists and communicated to the patient and his family members.

After counselling and an informed written consent, evisceration was done under general anaesthesia. The following findings were noted during evisceration. After peritomy, an area of the scleral necrosis was seen in the supero-temporal quadrant adjoining the limbus. After removal of the clear cornea and lens, about 5-6 pockets of yellowish pus were seen around a relatively pus-free but blood tinged vitreous (Fig.3). After scooping the contents of the eyeball, the necrotic sclera was excised and the healthy sclera and conjunctiva were sutured in layers. Orbital implant was deferred.

Post operatively, patient was stable and asymptomatic. He was started on Gentamycin eye drops and Moxifloxacin eye ointment and continued on systemic antibiotics for 5 days.

IV. Discussion

The management of endogenous panophthalmitis was difficult. Despite systemic antibiotics progression of the condition was rapid. Several factors may have contributed; the severity at the time of presentation, the virulence of the organism, the presence of diabetic status and the compromised renal status preventing the liberal administration of higher antibiotics.

Our case had diabetes; indwelling IJV catheter and its associated infection are known risk factors for endophthalmitis. Several challenges were experienced in the management of this case.

4.1. The presentation

This case presented to the ophthalmologist when panophthalmitis had already developed and not at the stage of endophthalmitis. The progression was rapid. The case presented to us at a late stage probably because the pockets of retinochoroidal abscesses as witnessed during evisceration were painless and no ocular complaints were experienced by the patient at this stage. The visual complaints remained unnoticed in the absence of pain. Hence a diagnosis was not possible at the stage of endogenous endophthalmitis. He presented only after the infection had spread to the anterior segment and anterior sclera causing pain and extra-ocular manifestations like lid oedema and proptosis, when the condition had already progressed to the stage of panophthalmitis. In contrast to endogenous cases, exogenous endophthalmitis presents early owing to the initial manifestations in the anterior segment later on spreading to the posterior segment.

4.2. The organism

The causative organism in this case was Enterobacteriaceae, gram negative rods which are the natural inhabitants of the gastrointestinal tract.^[15] They are generally considered pathogenic only for patients with lowered resistance to infection or impaired immunity. Systemically, they have been known to cause meningitis, pneumonia, bacillary dysentery, typhoid and food poisoning. Members of this genus have been reported as rare causes of endophthalmitis.^[16] Enterobacter species develop resistance rapidly to antibiotics due to their capacity to produce extended spectrum beta-lactamases. Carbapenems or, alternatively, fluoroquinolones are the common choice of antibiotics for enterobacter infections.^[17] Gram-negative bacteria accounted for 50% of all causative organisms in endogenous endophthalmitis. There has been a rising trend of gram-negative organisms causing endogenous endophthalmitis recently, especially in the South East Asia.^[18]

4.3. The endogenous spread

In endogenous panophthalmitis the organism predominantly reaches the eye through haematogenous spread from septic foci anywhere in the body, in this case the tunnelled IJV catheter. Endogenous endophthalmitis typically occurs in sepsis; in immunocompromised states such as acquired immunodeficiency syndrome and immunosuppressive therapy, including corticosteroid use or long-term antibiotic use or malignancy; due to the presence of an indwelling urethral or intravenous catheter; or intravenous drug abuse. Treatment of metastatic bacterial endophthalmitis is difficult, due to poor systemic antimicrobial penetration into the vitreous humor by the blood-retinal barrier, as retinal vessels lack fenestration and inflammation has little effect on the integrity of this barrier.^[19] When suspected, urgent ophthalmologic evaluation and treatment are needed to reduce the risk of losing vision in the affected eye.^[20] In our case, the patient presented when the condition had progressed to a stage of panophthalmitis.

4.4. The diabetic status

Patients with diabetes mellitus are known to have a higher incidence of postoperative endophthalmitis secondary to infection with gram-negative organisms than patients who are not diabetic.^[21] Studies have demonstrated that up to 42% of endogenous endophthalmitis patients had underlying diabetes mellitus.^[19] In our case, diabetes mellitus was present since two years with fluctuating blood sugar levels, which is a significant contributory factor for endogenous endophthalmitis.

4.5. Renal condition and indwelling catheter infections

Renal failure and indwelling intravenous catheters are significant risk factors for endogenous endophthalmitis. In our case, the Stage 5 chronic kidney disease and haemodialysis for the past 2 years were identified as risk factors. This chronic immunocompromised state probably was the risk factor for indwelling catheter infection which made this case a potential target for endogenous endophthalmitis. Metastatic bacterial endophthalmitis is a rare complication of dialysis catheter-related bacteraemia and only 3 case reports have been documented so far, comprising of a total number of 6 cases.^[19]

V. Figures

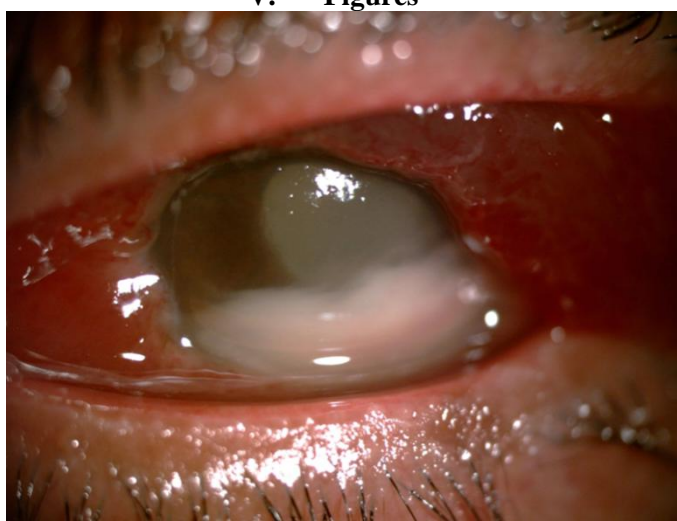


Fig.1. Anterior segment image of the left eye

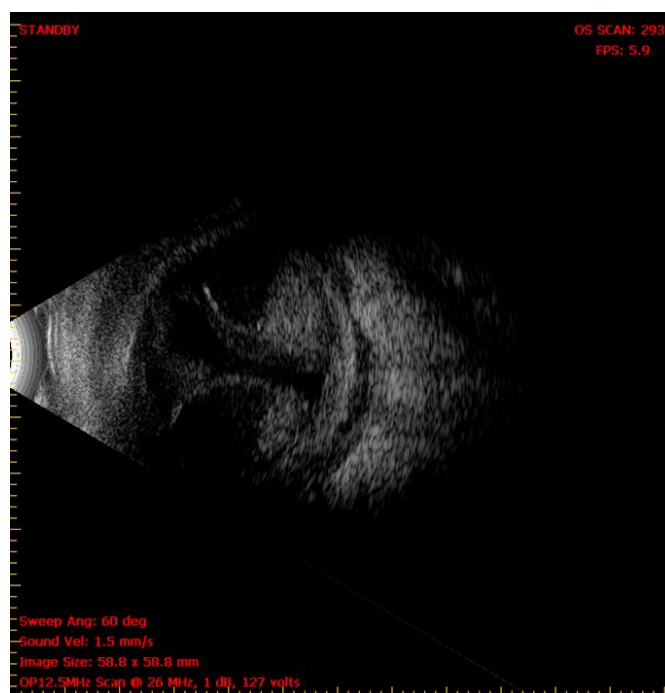


Fig.2. Left eye B scan image, showing vitreous detachment and 'T' sign



Fig.3.Intraoperative image of the left eye, showing pus pockets in vitreous

VI. Conclusion

Endogenous panophthalmitis is a vision threatening, serious complication of infections of indwelling catheters especially in the presence of a diabetes mellitus and chronic renal disease. Progression is rapid despite systemic administration of those antibiotics, to which the organism shows sensitivity on microbiological studies. Early intervention may help, but prognosis is poor in the setting of fluctuating diabetic status and IJV catheter infections leading not only to ‘blindness’ but to ‘loss of the eye’.

References

- [1]. Ravindran RD, Venkatesh R, Chang DF, Sengupta S, Gyatsho J, Talwar B. Incidence of post-cataract endophthalmitis at Aravind Eye Hospital: Outcomes of more than 42,000 consecutive cases using standardized sterilization and prophylaxis protocols. *J Cataract Refract Surg.* 2009;35:629–36.
- [2]. Russell W Read. Endophthalmitis. In: Duker SJ, editor. *Myron Yanoff and Jay S. Duker Ophthalmology.* 3rd ed. (China: Elsevier, 2009) p.815
- [3]. Moorthy RS, Rao PK, Read RW, Van Gelder R, Vitale A, Bodaghi B et al. Endophthalmitis. Intraocular inflammation and uveitis. *American Academy of Ophthalmology.* (Singapore:Elsevier,2012)pg 271.
- [4]. Miserocchi E. Endogenous Endophthalmitis. *Ocular immunology and uveitis foundation.* Vol. V No.3 Mar 2000.<http://www.uveitis.org/document/category/case-studies>
- [5]. Regillo C, Holekamp N, Johnson MW, Kaiser PK, Schuber HD, Spaide R et al. Posterior Segment Manifestation Of Trauma *Retina and Vitreous.American Academy of Ophthalmology.*(Singapore: Elsevier,2012)pg 327.
- [6]. Nobe JR, Gomez DS, Liggett P, Smith RE, Robin JB. Post-traumatic and postoperative endophthalmitis: a comparison of visual outcomes.*Br J Ophthalmol.*1987;71:614-7.
- [7]. Jackson TL, Eykyn SJ, Graham EM, Stanford MR. Endogenous bacterial endophthalmitis: a 17-year prospective series and review of 267 reported cases. *Surv.Ophthalmol.*2003;48:403-23.
- [8]. Regillo C, Holekamp N, Johnson MW, Kaiser PK, Schuber HD, Spaide R et al. Focal and diffuse choroidal and retinal inflammation. *Retina and Vitreous.American Academy of Ophthalmology.*(Singapore: Elsevier, 2012)pg 206.
- [9]. Wong JS, Chan TK, Lee HM, Chee SP. Endogenous bacterial endophthalmitis: an east Asian experience and a reappraisal of a severe ocular affliction. *Ophthalmology.* 2000;107:1483-91.
- [10]. Eagle RC. Inflammation. In ed: Pine JW.*Eye Pathology: An Atlas and Text.* (China: Lippincott Williams & Wilkins, 2012) pg. 33.
- [11]. Krėpštė L, Žemaitienė R, Barzdziukas V, Miliauskas A. Bilateral endogenous bacterial panophthalmitis. *Medicina (Kaunas).* 2013;49:143-7.
- [12]. Al-Mezaine HS1, Al-Assiri A, Al-Rajhi AA Incidence, clinical features, causative organisms, and visual outcomes of delayed-onset pseudophakic endophthalmitis. *Eur J Ophthalmol.* 2009;19:804-11.
- [13]. Kanski JJ, Bowling B. Uveitis. In ed: Nischal k, Pearson A. *Clinical Ophthalmology: A Systematic Approach.* 7th edition.(Elsevier. 2011) pg.401.
- [14]. Greenwald MJ, Wohl LG, Sell CH: Metastatic bacterial endophthalmitis: a contemporary reappraisal. *Surv.Ophthalmol.*1986;31:81–101.
- [15]. Saxena S, Meyer CH, Ohji M, Akduma L. In ed: Modi D, Ernst BJ, Feman SS. Endophthalmitis. *Vitreoretinal Surgery.*1st edition.(New Delhi:Jaypee brothers medical publishers. 2012)pg 347.
- [16]. Seal DV, Pleyer U. Microbiology. *Ocular Infection: Investigation and Treatment in Practice.* 2nd edition.(USA:CRC Press, 2013)pg 31.
- [17]. Sanders WE, Jr, Sanders CC. Enterobacterspp: Pathogens poised to flourish at the turn of the century. *ClinMicrobiol Rev.*1997;10:220–41.
- [18]. Wu ZH, Chan RP, Luk FO, Liu DT, Chan CK, Lam DS, Lai TY. Review of Clinical Features, Microbiological Spectrum, and Treatment Outcomes of Endogenous Endophthalmitis over an 8-Year Period.*J Ophthalmol.*2012;26:5078.
- [19]. Saleem MR, Mustafa S, Drew PJ, Lewis A, Shah Y, Shankar J, Ahmed W. Endophthalmitis, a rare metastatic bacterial complication of haemodialysis catheter-related sepsis. *Nephrol Dial Transplant.* 2007;22:939-41.
- [20]. de Lima LM, Cecchetti SA, Cecchetti DF, Arroyo D, Romão EA, Dantas M, Neto MM. Endophthalmitis: a rare but devastating metastatic bacterial complication of hemodialysis catheter-related sepsis. *Ren Fail.* 2012;34(1):119-22.
- [21]. Phillips WB 2nd, Tasman WS. Postoperative endophthalmitis in association with diabetes mellitus.*Ophthalmology.*1994; 101:508-18.