Ophthalmology and Eye Diseases

CASE REPORT

Severe Acute Endogenous Endophthalmitis with Staphylococcus Epidermidis in a Systemically Well Patient

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Abstract: Endogenous endophthalmitis is a devastating intraocular infection caused by haematogenous spread of bacteria or fungi, usually in people with immune deficiency. This case report is unusual as a systemically well patient with normal immune function and no identifiable locus of infection rapidly developed a fulminant endogenous endophthalmitis from an organism which does not usually produce aggressive virulence determinants. Endogenous bacterial endophthalmitis is an ongoing diagnostic and therapeutic dilemma for ophthalmologists as it is relatively rare, often initially presents as uveitis, and requires a high index of suspicion for prompt diagnosis and treatment. The treatment of endogenous endophthalmitis is still controversial due to a lack of clinical trials. Potential treatments include systemic antibiotics, peri-ocular injections, intravitreal injection of antibiotics and possibly corticosteroids, pars plana vitrectomy, or a combination of some of these.

Keywords: endogenous endophthalmitis, pars plana vitrectomy, retinal necrosis, uveitis

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Introduction

Endogenous endophthalmitis (EE) is a relatively rare intraocular infection caused by haematogenous spread of bacterial or fungal organisms from a distant primary source. It accounts for 2% to 8% of all cases of endophthalmitis.[1] It usually affects people with immune deficiency, such as diabetes mellitus, renal failure, AIDS, malignancies, as well as immunosuppressive treatment, intravenous drug use and invasive surgery.[2] Case series in the literature have reported 90% of patients with endogenous bacterial endophthalmitis (EBE) to have severe predisposing disorders, but the condition can also occur in apparently healthy individuals.[2,3] The gold standard for identification of causative organism is positive culture. Organism identification rate (including cultures from non-ocular sites) has been reported as 96% in one study.[1]

Gram-positive bacteria such as *Staphylococcus aureus*, *Streptococcus pneumoniae* and other streptococcal organisms are the most common causes of EBE in the Western world.[1] Though organisms such as *Listeria monocytogenes,*[4] *Aspergillus*,[5] *Pseudomonas aeruginosa*[6] and *Klebsiella*[7] have been identified in cases of EE in healthy people. The source of infection is identified in the majority of EBE cases (93%), most commonly as endocarditis or a gastrointestinal tract infection.[1]

Case Report

A 45 year-old woman presented with a one day history of right eye redness, mild photophobia and stabbing eye pain. She was otherwise healthy with no past ophthalmological problems, autoimmune disease, no immune compromise, and no relevant family history. Systems review revealed no recent illness, weight loss, nausea, diarrhoea, abdominal pain, fever, rigours, headache, hearing loss, skin wounds, or ulcers of mouth or genitalia. There was no history of recent travel, recent surgery, intravenous drug use or other risk taking behaviour. On examination her right visual acuity was only slightly reduced measuring 6/7.5 compared with 6/6 in the left eye. There were anterior chamber cells and flare present, but no hypopyon. A diagnosis of anterior uveitis was made, and treatment started with hourly prednisolone/phenylephrine 1%/0.12% eye drops and homatropine 2% eye drops three times a day. On review the following day she complained of reduced vision in the affected eye and on examination her right vision was now only LP, there was increased anterior chamber activity, a small hypopyon measuring 1.4 mm, dense vitritis and no view to the right fundus. Examination of the left eye was normal, as was general physical examination.

She was admitted to hospital, received 100 mg oral prednisolone, and ciprofloxacin eye drops were added to her treatment. Ocular ultrasonography suggested extensive vitreous debris with a flat retina (Fig. 1). A vitreous tap was done and revealed a cloudy looking vitreous. Intravitreal injection of foscarnet (2.4 mg/0.1 mL), vancomycin (1 mg/0.1 mL) and amikacin (0.4 mg/0.1 mL) was performed. Immediate gram stain identified moderate gram positive cocci, extensive polymorphonuclear cells, and the vitreous culture subsequently grew Staphylococcus epidermidis sensitive to flucloxacillin, dicloxacillin and vancomycin. As the patient was allergic to penicillin, intravenous vancomycin, 1 gram twice a day, was started.

Further investigations and management was done in consultation with infectious disease specialists. Initial blood tests revealed a normal full blood count, a slightly raised CRP at 16.2 mg/L (normal < 3.1) and ESR at 22 mm/hr (normal 1–12). There were no pus cells seen on urine microscopy. 3 sets of blood cultures, urine culture, a chest x-ray, orthopantomogram (OPG) x-ray, lumbar spine x-ray and CT scan of orbits and sinuses revealed no significant pathology. A transthoracic echocardiogram was also normal.

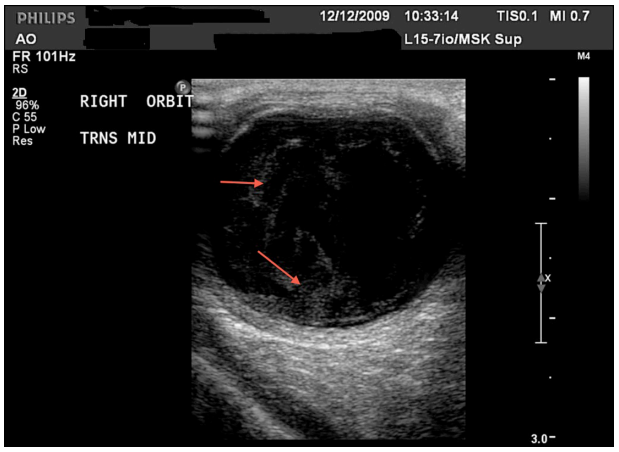

Figure 1. Ocular B-scan ultrasonography on admission shows extensive vitreous debris (arrows), but no abscess or retinal detachment is seen.

Blood tests for autoimmune screening (ANCA, ANA, ENA, HLA-B27, Rheumatoid factor) were all negative, and virology testing was negative for hepatitis B, hepatitis C, syphilis, CMV, with evidence of past exposure to EBV, VZV and Toxoplasma gondii (Polymerase chain reaction to Toxoplasma gondii, EBV and HSV negative). She was not tested for HIV.

One day after the vitreous tap her right visual acuity was still only vague HM with dense vitritis and no fundus view on examination. A pars plana vitrectomy (PPV) was performed to further decrease microbial load and repeat the intravitreal antibiotic. There were extensive vitreous bands, pus extending into the zonules, and large areas of retinal necrosis present, complicated by a suprachoroidal haemorrhage. A core vitreous biopsy confirmed persistent staphylococcus epidermidis. Further intravitreal vancomycin, ceftriaxone and heavy liquid was injected.

A subsequent PPV and lensectomy was done 16 days after the initial operation. The lens and large amounts of fibrotic tissue was removed. There was extensive intra- and sub-retinal haemorrhage, fibrosis across the ciliary body and ora serrata with a consequent detachment. She received 360 degrees laser treatment and silicone oil tamponade. Postoperative visual acuity was HM at 1 meter with an intraocular pressure of 5 mmHg.

Discussion

This case is unusual in that a patient who was systemically well with completely normal immune function went on to develop an acute fulminant case of *Staphylococcus epidermidis* EE which rapidly progressed to causing extensive retinal and other intra-ocular damage. Staphylococcus epidermidis is a gram-positive, coagulase-negative skin-colonising cocci, which does not usually produce aggressive virulence determinants, and usually requires an obvious breach in the host's defence mechanism to cause severe infection.[8] No locus of infection was identified in this case despite extensive investigation.

EE is an ongoing diagnostic and therapeutic dilemma for ophthalmologists as it is relatively rare, often presents like uveitis, and requires a high index of suspicion for prompt diagnosis and treatment. The treatment of EE is still controversial due to a lack of clinical trials. Potential treatments include systemic antibiotics, peri-ocular injections, intravitreal injection of antibiotics and possibly corticosteroids, pars plana vitrectomy, or a combination of some of these.

The patient described in this case report received the first dose of intravitreal treatment 3 days after initial symptoms appeared, and had a PPV and further intravitreal treatment 4 days after initial symptoms started. A previous review concluded that PPV and intravitreal antibiotics gave no benefit over intravenous antibiotics,[9] but recent reports have been encouraging; A major review of EBE found patients who had a PPV would benefit significantly in terms of higher likelihood for CF vision or better, and a smaller likelihood of needing evisceration or enucleation.[10] Reports have found that 25% of eyes with EBE end up eviscerated or enucleated.[1,10] Only 32% of patients retain a visual acuity better than CF.[10]

The greatest prognostic factors in EBE seems to be the infecting bacteria and the timing of initiating treatment. High clinical suspicion, early diagnosis and prompt aggressive treatment are imperative to minimise visual loss and the risk of losing the eye. Unfortunately, as this case shows, EBE can occasionally mimic typical idiopathic anterior uveitis, and it can be extremely difficult to preserve the eye or any significant level of vision despite appropriate treatment.

Authors' Contributions

EW conceived the study concept and design, was involved with patient care and drafted the manuscript and literature review. EH, AD: Involved with formation of the study concept and design, patient care and drafting the manuscript and literature review. All authors have read and approved the final version of the manuscript.

Disclosures

Author(s) have provided signed confirmations to the publisher of their compliance with all applicable legal and ethical obligations in respect to declaration of conflicts of interest, funding, authorship and contributorship, and compliance with ethical requirements in respect to treatment of human and animal test subjects. If this article contains identifiable human subject(s) author(s) were required to supply signed patient consent prior to publication. Author(s) have confirmed that the published article is unique and not under consideration nor published by any other publication and

that they have consent to reproduce any copyrighted material. The peer reviewers declared no conflicts of interest.

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