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Sir,

Recurrent endophthalmitis caused by *Burkholderia cepacia*

Burkholderia cepacia, earlier named *Pseudomonas cepacia*, is a Gram-negative motile bacillus. It is an important opportunistic pathogen in certain compromised hosts, particularly those with cystic fibrosis or chronic granulomatous disease.[1] Postoperative endophthalmitis with *B. cepacia* is very rare. Till date two endophthalmitis cases, one following cataract surgery and the other following trauma are reported.[2,3] We report a case of recurrent *B. cepacia* endophthalmitis following cataract surgery. To our knowledge, this is the second documented case of post cataract surgery endophthalmitis caused by *B. cepacia* (Medline search).

Case report

A 53-year-old gentleman was referred with a diagnosis of left eye acute postoperative endophthalmitis. He had undergone cataract surgery and posterior chamber intraocular lens (IOL) implantation. At 1 month after the surgery he developed pain and redness. He was a known diabetic with poor glycaemic control at presentation. His best-corrected visual acuity (BCVA) in the left eye was perception of light with accurate projection of rays. On examination, oedematous lids, congested conjunctiva, and a 2 mm hypopyon were noted. An exudative membrane was seen on the IOL surface. Ultrasound B scan revealed multiple dot and membrane-like echoes in the vitreous cavity with an attached retina.

He underwent pars plana vitrectomy, IOL explantation, and intravitreal injections of vancomycin (1 mg/0.1 ml), amikacin (400 μg/0.1 ml) and dexamethasone (400 μg/0.1 ml). Vitreous microscopy showed Gram-negative bacilli. He was treated with topical 0.3% ciprofloxacin and1% betamethasone eye drops every hour, and topical 1% cyclopentolate three times a day. He was put on oral ciprofloxacin 750 mg twice daily. Vitreous culture on blood agar, chocolate agar, and brain heart infusion broth showed significant growth of *B. cepacia*, identified by API 20 NE (Bio Merieux, France). The organism was sensitive to ciprofloxacin and ceftazidime, but resistant to chloramphenicol, amikacin, gentamicin, and vancomycin by the Kirby Bauer disc diffusion method. As the organism was resistant to the initially injected intravitreal antibiotics and clinically the patient was worsening, we injected intravitreal ceftazidime (2.25 mg/0.1 ml) and dexamethasone (400 μg/0.1 ml) on the third postoperative day. Oral ciprofloxacin was continued for 10 days. At 3 days after the second intraocular antibiotic injection, vitritis decreased and fundus examination showed preretinal exudates overlying an attached retina.

However, he returned 11 days later with an increase in pain and worsening of vitreous opacification. On the same day he underwent another (the third) intravitreal injection of ceftazidime (2.25 mg/0.1 ml) and dexamethasone (400 μg/0.1 ml). During successive follow-up, the inflammation cleared considerably. At 1 month after the last injection, he returned with sudden increase in pain and loss of vision. On examination, a few fresh keratic precipitates with an increase in vitritis was noted. Due to the recurrence of infection, we considered vitreous lavage and another (the fourth) intraocular ceftazidime (2.25 mg/0.1 ml) with dexamethasone (400 μg/0.1 ml). A repeat vitreous biopsy grew *B. cepacia*, sensitive to ceftazidime and ciprofloxacin. The patient was subsequently lost to follow-up, but returned after 2 months with no perception of light and a phthisical eye.

Comment

The first reported case of *B. cepacia* endophthalmitis presented as an indolent inflammation 1 year after cataract surgery. Although the organism was multidrug resistant, the eye showed complete resolution of inflammation.[2] Irvine *et al*[3] have reported one case of *B. cepacia* endophthalmitis following trauma. Postoperatively the inflammation persisted, but subsequently cleared with good visual outcome.

In our patient, the endophthalmitis was acute in onset and the organism was multidrug resistant. The multidrug resistance of *B. cepacia* is due to rough lipopolysaccharide encasing the organism.[4] The organism produces lipopolysaccharide and beta lactamase that renders the antibiotics ineffective against it.[5] Resistance to aminoglycoside noticed in the previous two cases[2,3] was also noted in our patient.

Unlike the previous two cases, our patient had recurrent endophthalmitis. Recurrent endophthalmitis was treated with multiple intravitreal antibiotic injections. Recurrence could be due to insensitive antibiotics (amikacin and vancomycin) given the first time, Gram-negative bacillus, multidrug resistance, and inadequate exposure time to antibiotics.[6]

Treatment of infections with virulent organisms poses problems even if the intraocular space is sterilized with

appropriate antibiotics. A significant amount of bacterial debris and potentially toxic products remain to account for treatment failure. The treatment of bacterial endophthalmitis ideally should be directed to the simultaneous control of infections and inflammation.[7] Hence, we prefer to inject intravitreal dexamethasone with antibiotic.

In our patient, despite treatment, the vitreous remained culture positive, and the same organism with the same sensitivity pattern was grown. This could probably be due to phenotypic changes in the organisms within the eye, thereby inhibiting the effect of antibiotics[8] and causing recurrent infections. This ultimately resulted in phthisis bulbi.

In conclusion, endophthalmitis due to *B. cepacia* is potentially difficult to cure. Patients need close follow-up due to risk of recurrence of infection.

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Sir,

Purtscher-like retinopathy as an initial presentation of thrombotic thrombocytopenic purpura: a case report

Thrombotic thrombocytopenic purpura (TTP) is a rare haematological disorder in which there is microvascular thrombosis with increased platelet consumption.[1] Ocular manifestations of TTP includes retinal haemorrhages, oculomotor nerve palsies, pupil abnormalities, and choroidal and retinal vascular occlusions.[2] We report a case of TTP, which presented with a severe bilateral ischaemic Purtscher-like retinopathy several days before any haematological abnormalities were detected. The patient has been left with permanent visual loss.

Case report

A 24-year-old Nigerian woman, presented with a 2-week history of left-sided pleuritic chest pain. Clinical examination and chest X-ray demonstrated left lower lobe consolidation. Her blood test showed neutrophilia and eosinophilia. C-reactive protein measured 105.

After 2 days, the patient complained of bilateral loss of vision and was referred for an ophthalmic opinion. Her visual acuities were 6/24 in the right eye and 6/60 in the left best corrected. Dilated fundal examination revealed bilateral Purtscher's retinopathy with macular oedema (Figure 1). Fundus fluoroscein angiography demonstrated areas of capillary drop out and retinal nonperfusion at the posterior poles (Figure 2).

A provisional diagnosis of a systemic vasculitis was made, and treatment with intravenous methylprednisolone 1 g/day was commenced. After