Of note, vasculitis has also been reported in association with posterior scleritis.[8,9] As a corollary, although there was no ultrasonographic evidence of posterior scleritis in our patient, the inflammatory focus in the superior intraconal space could have spread to the adjacent superior hemiretinal vein. Additionally there was evidence of optic nerve sheath inflammation on CT scan, although this was not evident clinically or ultrasonographically. Since the superior hemiretinal vein, like the central retinal vein, crosses the subarachnoid space around the optic nerve, there is a high probability of this optic nerve sheath inflammation to spill over to the vein.

To the best of our knowledge, this is the first case report of HCRVO associated with pseudotumour orbit as per medline search. The possibility of the occurrence of such vascular occlusions due either to direct compression or spill-over inflammation should be borne in mind in cases of visual loss due to pseudotumour orbit.

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Sir,

An unusual appearance of limbal conjunctival follicles in a patient on brimonidine and dorzolamide

Conjunctival follicles are known to occur as a result of ocular allergy to topical glaucoma medication, and are located mainly in the inferior bulbar and palpebral conjunctiva. Ocular allergy to topical treatment is associated with symptoms of burning and stinging; however, these symptoms settle and the conjunctival follicles disappear after discontinuing the offending agent. We report a case of bilateral multiple limbal and palpebral conjunctival follicles in a glaucomatous patient.

Case report

An 85-year-old Caucasian man presented in 1998 with hand movement (HM) vision in both eyes. A diagnosis of advanced open angle glaucoma with age-related macular degeneration (AMD) was given and he was registered blind. He had a presenting intraocular pressure (IOP) of 31 and 35 mmHg in the right and left eye, respectively. Retinal examination showed bilateral 0.95 cup/disc ratios with extensive AMD. He had a history of asthma, thus beta-blockers were contraindicated. After a 3-week trial, he became intolerant of Latanoprost 0.005%, which caused symptomatic blurring of vision. He was subsequently treated with topical dorzolamide 2% tds and brimonidine 0.2% to both eyes. No further blurring occurred, and IOP remained stable for 4 years. At this stage the right IOP control deteriorated, and the corresponding visual field showed progressive field loss. An augmented

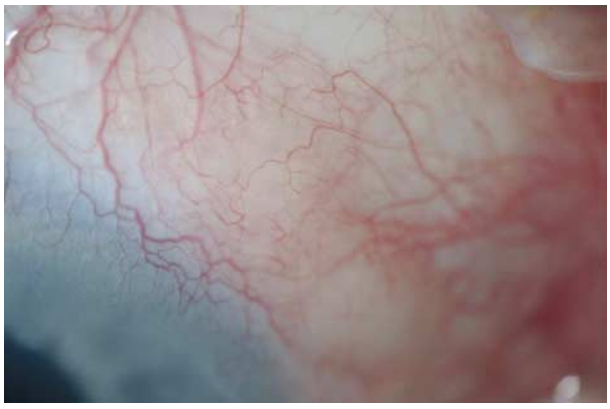

Figure 1 Anterior segment showing conjunctival limbal follicles.

microtrabeculectomy was planned. Surgery at the time of preoperative assessment was postponed however, due to the presence of bilateral, multiple limbal conjunctival follicles (Figure 1). The follicles were noted to occur on the superior and the inferior aspect of the bulbar conjunctiva without any other signs of ocular allergy such as injection or corneal erosions. The patient denied any ocular discomfort or irritation.

Visual acuity remained at HM in each eye, and anterior segment examination was unremarkable with no vitreous or retinal inflammation. The IOP was 19 mmHg in both eyes. The dorzolamide and brimonidine were stopped, and pilocarpine 2% preservative free and acetazolamide SR 250 mg po commenced. Chest X-ray, serum angiotensin converting enzyme, calcium, C reactive protein, and autoimmune screen were all normal. The erythrocyte sedimentation rate was 46 mm/h.

After 3 weeks, the follicles were still present albeit in a reduced number, and a right-sided inferior conjunctival biopsy preformed. Histology showed nonspecific infiltrate of lymphocytes and plasma cells without any granulomas. The conjunctival follicles gradually disappeared over the next 3 months. The IOP was maintained on the pilocarpine and acetazolamide. The patient has remained symptom-free throughout.

Discussion

Unlike oral carbonic anhydrase inhibitors that rarely cause ocular side effects, topical dorzolamide is known to cause various effects: stinging, burning, tearing, and blurring of vision. A 4% incidence of lid and/or conjunctival allergy has been reported.[1] Brimonidine is also known to cause ocular adverse effects such as hyperaemia, pruritus, foreign body sensation, blurred vision, and stinging sensation. Ocular allergy has been reported in 9.6% of cases.[2] In our case the patient was completely asymptomatic, and only on preoperative examination were the presence of bulbar conjunctival follicles noted. Unusually, these were located 360° around the limbus and not just in the inferior aspect of the conjunctiva, thus prompting a search for a systemic cause. Hypersensitivity to either one or both the topical antiglaucoma agents was suspected. In many patients with allergic reactions, the adverse effect is due to the preservative rather than the antiglaucoma agent[3] and, considering both agents contain benzalkonium chloride, sensitivity to this preservative cannot be excluded. To confirm our suspicions, ideally the patient would need to be re challenged with the suspected antiglaucoma agents and benzalkonium chloride. It was deemed inappropriate, however, to re-challenge the patient. Withdrawal of the topical agents led to gradual follicle resolution, and since no systemic cause was found and histology confirmed nonspecific inflammation the conjunctival reaction was labelled a probable adverse drug reaction using the criteria proposed by Naranjo *et al*[4] to assess causality of adverse events by drugs.

We recommend that patients about to undergo glaucoma surgery be carefully examined for any signs of ocular allergy even if the patient is asymptomatic.

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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