suggests that the immune response is directed to an inciting agent in the eye. The presence of positive titers for herpes simplex, varicella, rubella, cytomegalovirus, Epstein-Barr virus, and antistreptolysin O in some patients affected by frosted-branch angiitis suggested that viral or bacterial infection can be the triggering antigen. The prompt response to systemic steroids also indicates a probable immune-mediated mechanism.

In frosted-branch angiitis associated with viral disease like cytomegalovirus and human immunodeficiency virus, it has been theorized that viral antigens form immune complexes and deposit in retinal vessels causing vasculitis. A direct viral invasion may also be responsible for the pathogenesis. Immune complexes are also responsible for retinal vasculitis secondary to autoimmune disease. Direct infiltration of retinal vessels by malignant cells is believed to be the cause of frosted-branch angiitis among patients with leukemia and lymphoma.

Diagnosis is mainly by ophthalmoscopy and fluorescein angiography. Ophthalmoscopy shows the typical sheathing of retinal vessels mostly the veins giving the appearance of frosted-branches of a tree. Fluorescein angiogram shows normal blood flow but with late staining, leakage of dye from vessels, and optic-disc hyperfluorescence. Laboratory examinations usually do not show abnormalities and are mostly useful to rule out the possibility of associated systemic diseases.

Despite the severe retinal appearance, the prognosis is usually good, with rapid recovery of visual acuity after prompt steroid treatment to suppress intraocular inflammation and prevent visual loss and long-term complications such as capillary nonperfusion, retinal neovascularization, neovascular glaucoma, macular scarring, and retinal detachment. Recovery usually starts 2 to 3 weeks after treatment is initiated. Steroid-sparing agents may be used when there is significant steroid toxicity or persistent relapse at high dose of steroids. The ones commonly used are cyclosporine and azathioprine.[5]

Our patient was started on high-dose oral prednisone at an initial dose of 1 mg/kg/day for 5 weeks and responded well with improvement in visual acuity and fundus findings after 2 weeks. Funduscopy showed decreased vitreous haziness and opacities, less dilatation and tortuosity of the veins, and decreased perivascular sheathing. Prognosis is relatively good.

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A mysterious case of bilateral stromal keratitis

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ABSTRACT

Objectives

To describe a rare case of bilateral stromal keratitis and demonstrate the effectiveness of penetrating keratoplasty in the management of toxocara keratitis.

Method

This is a case report.

Results

A 53-year-old male farmer had a 10-month history of bilateral corneal opacity, photophobia, redness, foreign body sensation, and eye pain. The diagnosis was central microbial keratitis with the following etiologies considered: Epstein-Barr virus, herpes simplex, fungal, syphilis, tuberculosis (TB), myobacteria other than TB, and acanthamoeba. Despite treatment with topical steroids and antibiotics, both eyes worsened. Penetrating keratoplasty markedly improved the patient's visual acuity. Histopathology of the left corneal button revealed toxocara keratitis.

Conclusion

Good history taking, complete systemic and ocular examinations, and a histopathology of the corneal tissues are vital to the diagnosis of toxocara keratitis. Penetrating keratoplasty was shown to be effective in its management. Emphasis is given on prevention to decrease the incidence of the disease.

TOXOCARA keratitis is one of the many presentations of ocular toxocariasis. It results from invasion of the eye by the roundworm toxocara canis, a parasite that completes its life cycle in dogs and other canids, via the hematogenous route.

Only 2 cases of toxocara keratitis have been reported. Baldone and colleagues reported the presence of a nematode larva with the morphological appearance of toxocara in the corneal stroma.[1] However, no histopathologic examination was done because the larva was moving too swiftly to be surgically removed. The second case was the only one in a study by J. Altcheh et

al.[2] of 54 toxocariasis cases confirmed by enzyme immunoassay (ELISA).

We present the case of a 50-year-old male who had a 10-month history of eye redness, pain, blurring of vision, foreign-body sensation, tearing, photophobia, and corneal opacity in the right eye, also observed later in the left eye. There was no history of previous eye disease, surgery, or trauma. The patient had consulted several ophthalmologists, but chronic treatment with topical corticosteroids did not resolve the problem.

Visual acuity was 6/21 improved to 6/7.5 in the left eye (OS) and counting fingers (CF) in the right eye (OD). There was moderate congestion in OD with white central-corneal-stromal infiltrates, indistinct borders, and stromal edema. Severe congestion was present in OS with central multifocal to confluent stromal infiltrates, indistinct borders, stromal edema, and superior micropannus. No other abnormalities were seen. Corneal sensation was intact in both eyes (OU). Systemic physical examination was normal except for slightly distant breath sounds in the left hemithorax, consistent with pulmonary tuberculosis. Initial assessment was central stromal keratitis OU.

Differential diagnoses include herpes simplex/herpes zoster virus, Epstein-Barr virus, stromal keratitis, and Padi keratitis, which were not primary considerations in this case because these conditions respond well to topical corticosteroids. Moreover, they usually present with nummular or disciform stromal edema, with or without keratic precipitates, and rarely cause infiltration of the corneal stroma. Fungi can cause chronic corneal stromal disease but would worsen with steroid treatment, leading to corneal necrosis and rupture. Interstitial keratitis due to tuberculosis or syphilis, would have responded to steroids, but both conditions have a propensity for deep corneal-stromal neovascularization, which was not seen in this case. Corneal infections secondary to atypical mycobacteria other than TB are usually seen as wound infections after ocular surgery or trauma, which was not present in this patient.

The patient was initially treated as a rare bilateral acanthamoeba keratitis. Work-up included Gram's and Geimsa staining, bacterial and fungal cultures of corneal scrapings, and corneal biopsy, which showed no remarkable findings.

The patient was placed on the following regimen in OU: ketorolac (Acular, Allergan, Waco, TX, USA) every hour, atropine sulfate (Isopto Atropine, Alcon-Couvreur, Puurs, Belgium) TID, and ciprofloxacin (Ciloxan, Alcon-Couvreur, Puurs, Belgium) QID. After one month of treatment, OS worsened and developed a large epithelial defect with dense infiltrates. After 2 months, the left cornea showed signs of necrosis, melting, and eventual desmetocoele formation. An autoimmune etiology was considered. Additional work-up included antinuclear antibody, erythrocyte sedimentation rate, complete blood chemistry, and VDRL (Venereal Disease Research Laboratory) test, which were all within normal values. Chest X-ray showed findings consistent with pulmonary tuberculosis (PTB). Oral prednisone 60 mg per day was started, but was later discontinued with resultant increased inflammation in OD. Stromal infiltrates increased and hypopyon developed. Anterior-chamber tap showed marked eosinophila.

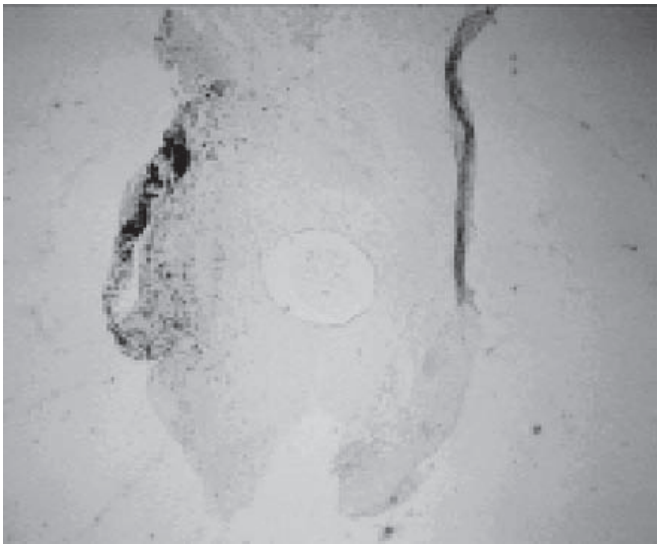

Figure 1. PAS stain of left corneal button.

Optical penetrating keratoplasty (PKP) was done for OS. Histopathology of the corneal button revealed a cavity lined with membranous material in the midstroma, containing an immature *Toxocara sp.* (Figure 1). Stool examination showed brown, formed ascaris and hookworms. Patient was started on mebendazole (Antiox, Janssen, Beersem Belgium) 250 mg BID for 5 days. Inflammation in OS resolved after PKP. Progressive cataract developed after two months and that eye underwent extracapsular cataract extraction with posterior-chamber lens implantation with postoperative visual acuity (VA) of 6/12, improved to 6/7.5.

The right eye also had PKP with postoperative VA of 6/15 improved to 6/9 with pinhole. There was mild nuclear sclerosis.

Chronic contact with dogs has been implicated in toxocariasis. J Altcheh et al. showed that in 54 patients who tested positive for toxocariasis based on ELISA, 92.6% had exposure to dogs for less than 6 months, 67% had a history of geophagia, 67% lacked basic hygiene, and 13% had a history of onychophagia.[2] This patient reported keeping dogs at home and eating cooked dog meat.

Toxocariasis is caused by ingestion of toxocara eggs. After ingestion, the third-stage larvae are released, penetrate the small intestines, and reach the liver via the

portal circulation. The larvae may gain access to the eye via choroidal, ciliary, and central retinal arteries. In the cornea, the larvae may elicit an immune response, resulting in migration of neutrophils and eosinophils from limbal vessels. These eventually invade the corneal stroma with disruption of corneal clarity. [3, 4]

A definitive diagnosis is dependent on the detection of toxocara larvae in tissue sections.[2] In this case, a section of the nematode was found in several slides.

ELISA is the most definitive serologic test for the detection of toxocara infection. Pollard et al. showed a specificity of 91% and sensitivity of 90% in 41 patients.[5] Shields showed 82% sensitivity and 100% specificity in 22 patients.[6]

The treatment for toxocara keratitis has not been reported. This case illustrates that penetrating keratoplasty may be effective since it removes the inflammatory foci from the body.

Prevention is still the key. This may be done by frequent handwashing and avoiding ingestion of or contact with eggs or larvae. It is also advised that dogs be treated with antihelminthics.

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Chopstick splinter: A rare cause of bilateral frozen orbits

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ABSTRACT

Objective

To report an unusual case of frozen orbit caused by an unsuspected intraorbital foreign body.

Method

This is a case report.

Results

A 31-year-old Chinese man presented with a 6-month history of painless progressive right caruncular growth with mucoid discharge. He also had bilateral progressive reduction in ocular movements. His best-corrected vision was 6/9 bilaterally. A conjunctival granuloma arising from the right caruncle and extending to the cornea and associated with ophthalmoplegia was also present. Orbital computed tomography showed a dense rod-like structure traversing the nasal area, extending from the superomedial wall of the right orbit to the apex of the left orbit with surrounding inflammatory reaction but sparing the optic nerve. The intraorbital foreign body, a 6 cm chopstick splinter, was successfully removed via right lateral rhinotomy.

Conclusion

Intraorbital foreign bodies are not uncommonly seen and, generally, the diagnosis is straightforward. Nevertheless, diagnosis could be missed if it is not suspected.

A MENTALLY challenged Chinese male presented with a 6-month history of painless progressive caruncular growth in his right eye associated with mucoid discharge. He was previously treated for conjunctivitis. His family members also noted progressive inability to move his eyes. He initially denied any history of trauma to his eyes, but later disclosed an alleged assault 5 years prior with supposedly no eye injury, ocular pain, or bleeding from the incident.

His best-corrected visual acuity was 6/9 bilaterally. Slit-lamp examination revealed a 4 x 5 mm pedunculated granulation tissue arising from the right caruncle and extending 3 to 4 o'clock of the right corneal limbus. The granulation tissue was mobile with areas of early keratinization. There was bilateral marked restriction of gaze in all directions. No ptosis, proptosis, or orbital cellulitis was present in either eye. The pupillary reflexes, intraocular pressures, and fundi of both eyes were normal.

Conjunctival swab failed to detect any organism. A nasal endoscopy revealed presence of granulation tissue in the right middle meatus with no obvious foreign body. Orbital and brain computed tomography (CT) revealed the presence of a hyperdense rod-like structure traversing the nasal area, extending from the superomedial wall of the right orbit to the apex of the left orbit with surrounding inflammatory reaction. The optic nerves were spared. The sinuses and nasopharyngeal spaces were clear (Figures 1 and 2).