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Review Article

Water related ocular diseases

Syed Shoeb Ahmad

Abstract

A number of ocular diseases can be attributed to contaminated water and we have coined a term ''Water-related ocular diseases (WRODs)'' to denote this wide-spectrum of conditions. WRODs are directly related to human contact with water and can occur through toxic, allergic, inflammatory or infective mechanisms. The non-infective causes can include chemicals used to clean swimming pools, oil spills and water-sport related injuries. Similarly, a number of infective organisms causing ocular diseases are transmitted through water. Since, these conditions can occasionally prove devastating, a review was done with the following aims: (i) To study the epidemiology of WRODs (ii) To assess the clinical presentation and current management of WRODs (iii) To highlight the future challenges and possible solutions to these problems.
The online search was conducted utilizing search engines such as PubMed, Google Scholar, ClinicalKey and the Virtual Library of the Ministry of Health, Malaysia for relevant terms such as water-borne, swimming pool and eye infections.

Keywords: Chloramines, Acanthamoeba keratitis, Ocular toxoplasmosis, Leptospirosis, Adenovirus

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Introduction

Water related ocular diseases (WRODs) attempts to be a broad-based term encompassing all ocular diseases occurring as a result of water exposure. The contaminants and pollutants in water can be infectious, toxic or allergic in nature. Although the condition is mild and self-limiting in most cases, in situations such as immunocompromise or extremes of age, WRODs can prove devastating and blinding.

Data released by the United States Environmental Protection Agency show that every year 1.8–3.5 million persons are affected by recreational water-borne illnesses (RWI) in the USA alone. RWI being attributed to contamination of water bodies by infectious agents from sewer overflows.[1]

Apart from infectious agents, ocular diseases can also be caused by mechanical trauma, chemicals and toxins. A continuous exposure to these substances can affect the quality of life of individuals exposed to them. The Disability Adjusted Life Years (DALY) lost from WRODs is proving to be a global burden on public health. For example, in the Netherlands a DALY of 2400 was calculated from ocular toxoplasmosis alone.[2]

Non-infectious conditions

Swimming pool related

Individuals exposed to ocean or swimming pool water commonly develop allergic conjunctivitis. This leads to ocular irritation, forcing the individuals to rub their eyes aggressively and causing corneal abrasions or secondary infection. There have also been reports of outbreaks of short-incubation ocular illnesses following exposure to swimming pools. The symptoms reported were burning, tearing, photophobia and blurred vision.[3] The Centers for Disease Control (CDC) conducted a study to assess illness and injuries caused by swimming pool disinfectants and chemicals in the USA. Data from the Sentinel Event Notification System for Occupational Risk (SENSOR)-Pesticide surveillance program and from the National Electronic Injury Surveillance System

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Department of Ophthalmology, Queen Elizabeth Hospital, Kota Kinabalu, Malaysia
e-mail address: syedshoebahmad@yahoo.com

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(NEISS) was used for analysis. According to this study, as many as 33% cases from SENSOR and 42% from NEISS had ocular illnesses attributable to swimming pool additives.[4] A study on indoor swimming pool workers in Italy also reported red eyes in 48.9% and itchy eyes in 44.4% of the respondents.[5]

The pathogenesis of such WRODs is due to exposure to irritants such as chloramines, specially nitrogen trichloride or trichloramine (NCl_3). A number of chlorine containing agents (such as Calcium- or Sodium-hypochlorite and Chlorinated isocyanuric acid) are used to disinfect swimming pool water.[6] The free chlorine reacts with nitrogen-containing matter introduced into the pool water by swimmers (urine/urea, sweat, skin squama, cosmetics) leading to the formation of a number of ''disinfection by-products'' (DBPs) including trihalomethanes and chloramines.[7,8] Trichloramine is regarded as the main causal agent responsible for ocular irritation.[6–8]

Swimming pool related incidents can be reduced by prophylactic measures aimed at minimizing DBPs such as: controlling all technical parameters during the water disinfection process, regulation of water quality, ensuring good public hygiene (showering before entering swimming pools and wearing proper clothing) and undertaking regular public education and awareness campaigns.[8]

Water sports related

WRODs during water sports can have multiple mechanisms. They can be chemicals from pollutants or mechanical injuries from sport equipments, such as masks and diving boards. In February 2015, some of the competitors in a kayaking contest in New Zealand developed sudden ocular pain and reduced vision. The condition was suspected to be due to exposure to dishwashing liquid or fire-retardant residues in the river water.[9] In a water-jet related incident, high pressure from the vehicle was reportedly responsible for Descemet's membrane ruptures in an individual.[10]

Oil spills

In 2007 an oil spill occurred in Daesan, South Korea. A study of 442 individuals involved in the cleanup operations showed that ocular symptoms were present for the longest period of time (9.7 months), compared to others like headache (8.4 months), skin symptoms (8.3 months), neurovestibular (6.9 months), respiratory (2.1 months) and back pain (1.8 months).[11] Subsequently, in 2010, an explosion on the Deepwater Horizon oil drilling rig in the Gulf of Mexico led to release of oil and dispersants into the water. A number of individuals subsequently had ocular complaints like burning pain and irritation.[12]

These non-specific conditions are usually self-limiting and resolve with minimal sequelae.

Specific water-borne ocular infections

A large number of pathogens are present in water bodies as normal inhabitants or contaminants. Herein, we take a concise look at the common infective agents causing WRODs.

Acanthamoeba

Five species of these free-living amoebae have been reported to cause keratitis. These are: A. polyphaga, A. castellani, A. hatchetti, A. culbertsoni and A. rhysodes. Acanthamoebae are present in fresh water, brackish water and sea water bodies. They are also found in swimming pools, hot tubs, drinking water systems, heating-ventilating-air-conditioning (HVAC) systems and humidifiers.[13] The occurrence of Acanthamoeba in water resources has an added danger since these amoebae can harbor pathogenic microorganisms such as Legionella, Pseudomonas and Helicobacter.[13–17]

Poor contact-lens hygiene is a significant risk factor for developing Acanthamoeba keratitis. Wearing of contact-lenses during swimming or showering can significantly increase the risk of infection from Acanthamoebae.[15,18–20]

Acanthamoeba keratitis is characterized by an initial limbitis with anterior stromal and perineural infiltrates (radial keratoneuritis). There can be overlying punctate or pseudodendritic keratitis. The infiltrates enlarge and join to form a central or paracentral ring abscess. Subsequently, satellite lesions, stromal opacification, scleritis and corneal thinning with descematocele formation may occur.[21,22] Occasionally, corneal perforation may lead to endophthalmitis (see Fig. 1).

Acanthamoeba keratitis is treated with 4 groups of chemotherapeutic agents[14] (see Table 1).

Public health awareness, e.g. warning signs in the area, to educate the public against water-related activities while using contact lenses is necessary to prevent Acanthamoeba infections.[15,23] Chlorine conventionally used for water treatment does not eliminate Acanthamoebae.[24] Thus, improved purification processes such as correct filter pore sizes are crucial factors in eliminating Acanthamoebae from water sources.

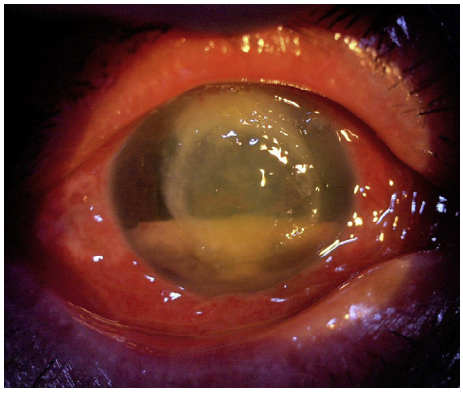

Fig. 1. Anterior segment image of a 57-year old patient who developed Acanthamoeba keratitis. Subsequently, she had corneal perforation requiring corneal gluing.

Giardiasis

This is an infection caused by the protozoa Giardia lamblia (sy. G. intestinalis or G. duodenalis). Giardiae occur in 2 stages: cysts and trophozoits. Cysts are excreted in feces and can survive for 2–3 months under ideal conditions. The common mode of transmission is by ingestion of cysts in contaminated water or directly through the fecal/oral route.

Table 1. Chemotherapeutic agents to treat Acanthamoeba keratitis.

Cationic antiseptics	Inhibit membrane function	(1) Polyhexamethylene biguanide (PHMB) (2) Chlorhexidine
Aromatic diamidines	Inhibit DNA synthesis	(1) Propamidine isethionate (Brolene) (2) Pentamidine (3) Hexamidine
Aminoglycosides	Inhibit protein synthesis	(1) Neomycin (2) Paromycin
Imidazoles	Destabilize cell walls	(1) Clotrimazole (2) Fluconazole (3) Ketoconazole

A few outbreaks of Giardiasis have been reported due to mass exposure to contaminated water or direct contact with the infected patients (e.g. in child care centers).[25]

Ocular changes reported include: iridocyclitis, choroiditis, retinal hemorrhages, retinal vasculitis and a peculiar ''salt and pepper'' retinal degeneration.[26–29]

Giardiasis can be diagnosed by direct observation of the cysts or trophozoites in fecal samples. Antigen tests include ELISA, immunochromatogenic tests, direct immunofluorescence and PCR assays.[25,26]

Treatment of Giardiasis is with metronidazole, tinidazole, albendazole, furazolidine or paromomycin.[25–27]

Toxoplasmosis

This condition is caused by Toxoplasma gondii, an obligate intracellular parasite. Ingestion of water contaminated with oocytes from the feces of infected cats can cause infection in humans.[30,31] In 1995, British Columbia, Canada, witnessed the world's largest outbreak of waterborne toxoplasmosis.[32,33] A severe form of clinical toxoplasmosis related to ingestion of T. gondii oocysts in food or water has also been reported.[34]

The definitive hosts for T. gondii are felids, especially domestic cats. Warm blooded animals like mice, livestock and humans act as intermediate hosts. There are 3 infective stages of T. gondii: (i) Sporocysts (Oocysts), which get excreted in cat feces. (ii) Bradyzoites, which remain encysted in tissue. (iii) Tachyzoites, the proliferative phase responsible for tissue destruction and inflammation. The oocysts can wash into water bodies from soil and hosts acquire infection by exposure to water contaminated by them.[22,32,34–37]

Active disease occurs in the form of ''Focal retinitis'', which appears as a solitary inflammatory focus, often near a healed chorioretinal scar. There is dense vitritis, making visualization of the lesion difficult. However, it usually stands out in the haze, giving a ''headlight in the fog'' appearance. In some cases, inflammation surrounding the optic nerve head may lead to optic disc inflammation itself (Jensen choroiditis).[22]

Laboratory investigations available for diagnosis of T. gondii are: Sabine Feldman Dye test; Indirect Fluorescent antibody Assay (IFA); direct agglutination test; Latex Agglutination Test (LAT); Microparticle Enzyme Immuno Assay (MEIA); Enzyme-linked Fluorescent Assay (ELFA) and ELISA.[37,38] Classically, Ocular toxoplasmosis has been treated with ''Triple Therapy'' consisting of Pyrimethamine (loading dose of 50–100 mg, then, 25–50 mg once daily), Sulfadiazine (2–4 g loading dose, then 1 g 4 times daily) and oral corticosteroids (0.5–1.0 mg/kg body weight daily). In ''Quadruple Therapy'' Clindamycin (300 mg four times daily for 3 weeks) is added to the above. (This medication is combined with Sulfadiazine to prevent pseudomembranous colitis). Other alternatives are: Cotrimoxazole twice daily for 4–6 weeks; Atovaquone 750 mg three times per day; or Azithromycin 500 mg per day for 3 days. In pregnant patients Spiramycin 3 g per day can be used in the first trimester and triple therapy thereafter. Intravitreal Clindamycin (1.0/0.1 ml–1.5 mg/0.1 ml) is also favored currently.[39–43]

Gnathostomiasis

Ocular Gnathostomiasis is an uncommon parasitic infection caused by accidental infection by the third stage larvae of spiruroid nematode Gnathostoma spp (G. spinigerum, G. hispidum, G. doloresi and G. nipponicum).[44,45] Humans are accidental hosts, but can also act as second intermediate hosts by drinking water contaminated with copepods infected with the second-stage larvae. The commonest presentation is anterior uveitis, iris holes and direct visualization of the intraocular parasite. The other manifestations include: eyelid oedma, conjunctival chemosis, hyphema, retinochoroiditis, vitreous hemorrhage, central retinal artery occlusion and retinal detachment.[46]

Laboratory investigations include: An ELISA test for L3 immunoglobulin G (IgG) antibody and an Immunoblot to detect the 24-kDa band, specific for Gnathostoma.[46] The condition is treated with Albendazole, Thiabendazole, Praziquentel, Metronidazole, Diethylcarbamazine, Quinine or Ivermectin.[44,46–48]

Coenurosis

It is a parasitic infection caused by Coenurus cerebralis, the cystic larval stage of the dog tapeworm. Four species of this parasite are known to cause infection in humans: Taenia multiceps, T. Serialis, T. glomerata and T. brauni.[49–51]

The adult tapeworms develop in the intestines of canids (dogs, foxes). The infected carnivore then passes the tapeworm eggs (gravid proglottids) in feces. Humans are incidental intermediate hosts due to inadvertent ingestion of water contaminated by the mature eggs. The eggs subsequently release the oncospheres which penetrate the intestinal wall to enter the blood stream and then to the eye. As the cyst matures, it excites an inflammatory reaction. Toxins released by the cyst induce iritis, vitritis, optic neuritis or neuroretinitis. The cysts often rupture, causing severe uveitis and secondary glaucoma. The chronic inflammation ultimately leads to a painful and blind eye.[49,50,52]

Diagnosis of the condition is made by direct visualization of coenuri. CT scan studies show the viable cysts as lucent lesions with surrounding contrast-enhanced rim. Treatment options include: Praziquentel (50 mg/kg body weight/day in divided doses for 14 days). Surgical removal of the cyst is mandated in cases which do not improve with 1 week of medical treatment.[50,53]

Pseudomonas

An ubiquitous gram-negative bacillus, Pseudomonas aeruginosa is widely found in the environment. In lake and reservoir waters, the concentration can reach 10/100 ml to

>1000/100 ml. Ocular infection can be acquired following exposure to inadequately chlorinated hot tubs, whirlpools or swimming pools. A severe form of infection leading to corneal ulcers is seen in contact lens users who use home-made saline solutions. Previous history of refractive surgery, associated trauma, ocular surface disease and immunosuppression are additional risk factors.[54,55]

Keratitis starts with redness, pain and decreased vision. On examination, an epithelial defect with infiltrate and often a hypopyon is present. The condition may progress to corneal perforation and eventual endophthalmitis. A characteristic feature of Pseudomonas infection is a diffuse epithelial graying, away from the main lesion.[56]

Laboratory tests include Gram's stain and culture-sensitivity of scrapings from the corneal infiltrate. Samples from the contact lens solution and lens case should also be taken for microbiologic workup.

Treatment of choice is governed by the sensitivity results. However, empirical treatment with a newer Fluroquinolone (e.g. gatifloxacin) or combination therapy with a cephalosporin and an aminoglycoside can be started while waiting for the results. In a study, P. aeruginosa was found to be highly sensitive to ceftazidime, ciprofloxacin, and amikacin.[57]

Melioidosis

It is a rare ocular infection caused by the gram negative bacillus Burkholderia pseudomallei.[58,59] The organism is often found in stagnant water and gains entry through percutaneous inoculation, a professional hazard for paddy field workers.[58–61] It can also be transmitted by ingestion of contaminated water.[62] The systemic illness can vary from a self-limited flu to acute fulminant septicemia or a smoldering, chronic infection.[58,61] Septicemic illness which can prove fatal is seen in patients having co-morbidities such as diabetes mellitus, chronic lung/renal disease, alcoholism, prolonged steroid use, thalassemia and cancers. It is presumed that these systemic conditions affect neutrophil function which plays a significant role in the pathogenesis of Melioidosis.[60,63,64]

Melioidosis can lead to corneal ulcers (usually with a history of ocular injury), subconjunctival abscesses, orbital cellulitis and endophthalmitis.[59–61,63]

The gold standard for diagnosis is the isolation of the organism from bodily fluids. Culture of the fluids can be done on Ashdown's selective agar or on B.pseudomallei selective agar (BPSA). Other tests such as HIA, latex agglutination, ELISA, immunoflurosecence and PCR techniques are also being made available. C-reactive protein is usually elevated and shows recovery within 2 days of successful treatment.[58,60,63]

Melioidosis is treated during the acute phase by Ceftazidime (50 mg/kg; up to 2 gm, every 6–8 hourly) or a carbapenem (Meropenem: 25 mg/kg; up to 1 gm, every 8 hourly. Imipenem: 25 mg/kg; up to 1 gm, every 6 hourly). The acute phase treatment is given for 10–14 days, followed by the oral eradication therapy for 3–6 months. The drug of choice during this phase is Co-trimoxazole. In children and pregnant patients Amoxicillin-Clavunate can also be used.[58,63,64]

Leptospirosis

This is regarded as the most widespread zoonosis in the world. The prevalence ranges from 0.1 to 1/100,000 per year in temperate areas to 10 or more/100,000 per year in the humid tropical areas. During outbreaks, e.g. after floods and in high-risk groups, 100 or more/100,000 persons may be infected.[65]

Leptospirosis is caused by pathogenic spirochaetes of the genus Leptospira such as L.interrogans. It occurs as a zoonosis in rodents, dogs, cattle, pigs and wild animals. The leptospires are excreted from the reservoirs in kidneys, leading to contamination of water bodies. The organism can survive in water for several months. Humans are infected by drinking or bathing in contaminated water. In humans the organisms usually gain entry through abraded skin or intact mucosa. This may lead to either a self-limiting anicteric syndrome or icteric leptospirosis (Weil's disease).[66,67]

Ocular manifestations of leptospirosis include: non granulomatous anterior uveitis, hypopyon, cataract, interstitial keratitis, vitreous inflammatory reaction and membranes, retinal vasculitis, papillitis and often, panuveitis. In severe systemic infection, the eyes characteristically show circumciliary congestion and yellowish sclera[68–71] (see Fig. 2).

Laboratory tests include: Microscopic Agglutination Test (MAT), ELISA, macroscopic agglutination, indirect haemagglutination, lepto dipstick, microcapsule agglutination tests and lateral flow assay. PCR tests are also being available recently.[69]

Treatment is with oral doxycycline, amoxicillin or ampicillin in mild to moderate illness. In cases with severe infection, the drugs of choice are intravenous Penicillin G, Cefotaxime or Ceftriaxone.[69,71,72]

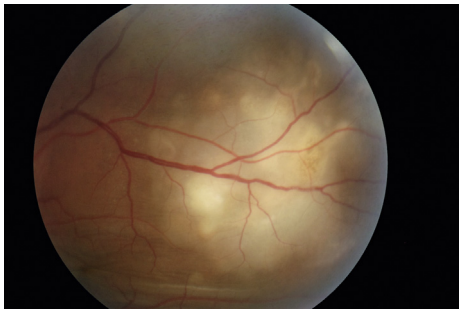

Fig. 2. Fundus image of a 32-year old soldier who had Leptospira chorioretinitis.

Toxocariasis

Toxocariasis occurs as a result of human infection with the larvae of Toxocara canis, Toxocara cati, Ascaris suum or some new species being reported recently.[73–75] Humans are paratenic hosts, who are infected accidentally by ingestion of invasive eggs through contaminated water.

Ocular larva migrans syndrome (OLM) is a localized manifestation of Toxocara infection, usually caused by a single second-stage larva. OLM is unilateral in 90% of the cases. It can present as Chronic endophthalmitis (CE), posterior pole granuloma (PPG) or a peripheral granuloma (PG).[76] CE is usually seen in children 2–9 years of age. There is a pan-uveitis, with the peripheral retina covered with dense greyish-white exudates. PPG occurs in older children (6–14 years). It

presents as a round, yellowish-white, solid granuloma on the posterior pole. A PG appears as a white, hemispherical granuloma in any quadrant of the fundus. There can be vitreous bands seen, causing dragging of the disc and macula and tractional retinal detachments[75–78] (see Fig. 3).

Other complications of OLM include: fulminant endophthalmitis, papillitis, secondary glaucoma and choroidal neovascular membranes.[80]

Laboratory diagnosis is done by an ELISA test. It is nonspecific and any positive titre can be considered as significant in association with clinical findings. Other tests include: an ELISA test for toxocara excretory-secretory antigen (TES-Ag) and Toxocara Goldmann-Witman (GW) coefficient analysis of aqueous and serum.[74] Treatment includes: Thiabendazole (25 mg/kg body weight, up to 3 gm/day for 5 days), Albendazole (800 mg twice daily for 6 days) or Mebendazole (100–200 mg twice daily for 5 days). Argon laser can be utilized to kill live larvae seen in the retina. Complications of the infection, such as retinal detachment, persistent vitreous opacification and epi-retinal membrane formation, require surgery.[75,77–80]

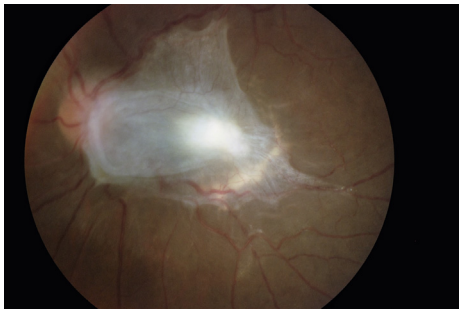

Fig. 3. Fundus image showing posterior pole fibrosis in a 23-year old with Toxocariasis.

Adenoviruses

Nearly 75% of the adenoviral ocular infections in humans are asymptomatic. These viruses usually cause self-limiting conjunctivitis and keratitis. Water-borne transmissions are usually reported in swimming pool outbreaks. Human adenoviruses have only been found in human feces, thus it is presumed that the occurrence of the virus in natural aquatic environments is due to presence of untreated or poorly treated human feces.[81]

Adenoviruses can cause Pharyngoconjunctival fever (PCF) or Epidemic Keratoconjunctivitis (EKC). In general, these conditions are characterized by follicular conjunctivitis, minimal watery discharge and pre-auricular lymphadenitis. PCF is caused by serotypes 3, 7 and 14. It may lead to keratitis in 30% patients but is seldom severe enough to cause symptoms. EKC is caused by serotypes 8, 19 and 37. The keratitis is commoner (in 80% cases) and can be severe.[22,82–85]

Steroid eyedrops are indicated if the eye is uncomfortable or the vision is decreased from anterior stromal corneal opacities.[22]

Other organisms

A number of other organisms have been rarely implicated in causing WRODs. Rathinam et al. have reported a possible endemic, water-borne trematode infection in children exposed to pond or river water. These patients were found to develop subconjunctival nodules and occasionally a granulomatous uveitis, leading to formation of anterior chamber nodules. Clinic-pathologic studies of the subconjunctival nodules revealed necrotizing granulomas containing tegumental and internal fragments of a parasite. PCR amplification of rDNA ITS regions of DNA from cercaria larvae and DNA from the granuloma tissue of patients, identified the fluke as Procerovum cheni. [86,87]

Other causative organisms include Legionella, Coxsakie B virus and intra-operative fungal infections during ocular surgeries due to contamination of irrigating fluids. A case of exogenous endophthalmitis caused by Enterococcus casseliflavus following a toy water gun injury has been reported. Uveitis from the trematode fluke Procerum varium from snails has been reported from a case series in South India. There is also a report of Mycobacterium chelonae infections from contaminated humidifiers.[88–92]

Conclusion

WRODs are a constant threat due to the increasing contamination of water bodies by various chemicals and pathogens world-wide. Climate change is bringing about new hazards in water bodies which can lead to ocular illnesses. There is also a risk of development of resistant species of organisms which may be difficult to manage. In such a scenario it is worth revisiting the common causes of such water-borne ocular diseases.

Conflict of interest

The authors declared that there is no conflict of interest.

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