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Review Article

Infective keratitis: a concise overview of the clinical characteristics and treatment of keratitis caused by microbial agents

Mahesh Chandra[1]*, Jitendra Singh[2], Ayush Jha[1]

[1]Department of Optometry, Dr. Sushila Tewari Hospital and Government Medical College, Haldwani, Uttarakhand, India
[2]Clear Vision Eye Care Centre, Faridabad, Haryana, India

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***Correspondence:**
Dr. Mahesh Chandra,
E-mail: Mahesh.optometrist@gmail.com

ABSTRACT

The following document provides an overview of microbial keratitis, a well-known sight-threatening inflammation of the cornea that progresses to the corneal ulcer. Sometimes, such infection is difficult to identify and cure due to the involvement of multiple pathogens implicated in the specific disorders because of similar symptoms and immunological responses. Instead of fungi and protozoa, viruses and bacteria are the most prevalent pathogens that cause microbial keratitis. A virus contains protein-encased genetic material and may infect any living creature, including bacteria and fungi, by replicating inside the host's cell and infecting neighbouring cells. Bacteria are complicated pathogens that may thrive in any media and cause harm to host cells, often through the production of toxins. Fungi are far more difficult; they spread quickly and can cause harm to several organs at the same time if the immune system is compromised. Protozas are found freely in the environment and once invade the cornea, divide quickly and become difficult to identify as well as treat, because of their involvement or in conjunction with polymicrobials. These microbes show common symptoms after invading the cornea although; their common diagnostic procedures show different results to trace out their existence in the tissue. Up to some extent, specific treatment can cure the disease with certain conditions according to a load of microbes, therefore visual status gets hampered, otherwise total loss of the eye takes place due to the endophthalmitis.

Keywords: Bacteria, Virus, Fungi, Protozoa, DNA, RNA, Replication, Keratitis, Ulcer, Corneal graft, Hypopyon, Herpes, PCR

INTRODUCTION

Microbial keratitis may be caused by bacteria, fungi, viruses and parasites and is more commonly associated with the formation of corneal ulcers followed by ocular trauma, and underlying and surface disease of the cornea.[1] Keratitis is a corneal inflammation, marked by redness of the eye with moderate to severe pain, decreased vision, photophobia, and a 'gritty' sensation.[2,3] Post keratitis condition is often referred to as a 'corneal ulcer'. However, the term keratitis and corneal ulcer are not interchangeable, because a cornea may have an ulcer without an invading infection. The increased chance of keratitis is intended with the contact lens wearer, decreased immunity, diabetes, humid and warm climate, prolonged use of corticosteroid eyedrops, vegetative or chemical injury to the eye, and Fungal bloodstream infection.[4-6] The cornea is protected by the tear film to introduce natural defence mechanisms by the lysozyme, beta lysin and other protective proteins. (immunoglobulins and other antimicrobial compounds).

The bacteria invade the cornea in condition either due to the trauma, the immunity is compromised or the causative agent becomes very virulent. S*taphylococcus aureus* and *Pseudomonas aeruginosa* (librates enzyme to decay the cornea) are the commonest bacteria for infection. Others are *Pseudomonas pyocyanea*, *Streptococcus pneumoniae*, *Escherichia coli*, *Proteus* and *Klebsiella*.[7,8] *Staphylococcus*, *Streptococcus* and *Pseudomonas* species disruptes corneal epithelium and Neisseria gonorrhoeae, Neisseria meningitides, and Corynebacterium diphtheria invade through the intact epithelium. Bacterial keratitis can occur due to extended contact lens usage, inadequate lens care, or corneal damage (e.g., prior surgery, foreign body, chemical injury). Causes of ocular surface illness include dry eyes, lid malposition, and chronic blepharitis, as well as immunosuppression from medicines, immunodeficiency syndromes, and diabetes.[9]

Fungal corneal infection (keratomycosis) of the eye is a rare, typically slow and persistent disease, usually a result of an eye injury, especially by vegetation. It may turn very serious from keratitis (corneal infection) to endophthalmitis (infection of the interior of the eye) because of to injudicious use of antibiotics and steroids in recent years. The two types of endophthalmitis have been defined as the fungal spores that enter through an external source called exogenous endophthalmitis. In invasive eye procedures like a corticosteroid injection, the surgical procedure includes irrigation solution dye and contact lens solution. The bloodstream fungal infection causes endogenous endophthalmitis. For example candidemia spread.[10-15] Various types of fungi affect the eye, some of them are Aspergillus, Fusarium, (most common), Alternaria, Cephalosporium, Cladosporium, Curvularia, Penicillium and Rhizopus and the yeast Candida albicans and Cryptococcus generally found in mucous membranes. The invasion of corneal tissue by the filamentary fungi comes through ocular trauma, and yeasts in the immune-compromised patients respectively.[9,16,17] Fungi takes entry in the anterior chamber without perforation of the cornea, hence hypopeon could not be unsterile like bacterial keratitis (till the perforation). The commonest viral keratitis is due to the Herpes Simplex Virus (HSV) and Varicella Zoster Virus (VZV) also known as shingles (due to introduced symptoms). Herpes simplex keratitis develops as a result of HSV latency in the ophthalmic nerve, the branch of the trigeminal nerve. The inflammation of Herpes keratitis initially expresses as the dendritic infiltrate pattern in the corneal epithelium and thereafter turns to the clouding of the cornea.[1] Viruses can enter many organs at the same time in the body through mucous membranes, such as the eyes, nose, mouth, penis, vagina, and anus, as well as through skin breaks or mosquito or tick bites.

A serious keratitis that takes place with an amoebic infection caused by Acanthamoeba often affects contact lens wearers.[18,19] Acanthamoeba is the most common protozoa presenting increasing incidence with the difficult diagnosis and unsatisfactory treatment. Acanthamoeba *castellani* amoeba is freely found in soil, freshwater, well water, seawater, sewage and air. It exists in trophozoite and encysted forms, therefore, the infection comes through direct corneal contact with contamination mostly Contact lens wearers get affected using home-made saline from contaminated tap water, predisposing variables include minor trauma from contaminated vegetable substances, salty water diving, windblown harmful substances, hot tub use, organic matter trauma, and muddy water exposure. It may also occur in patients with herpetic keratitis, bacterial keratitis, bullous keratopathy and neuroparalytic keratitis.[20]

BRIEF DESCRIPTION OF PATHOGENS

Bacteria

Bacteria are microscopic, unicellular organisms classified by their shapes- Spheres or ball-shaped known as cocci, Rod-shaped known as bacilli and Spirals or helixes shape, called spirochetes (Figure 1). They are also classified by their genetic material. According to the staining property of their cell wall with gram stain, they are defined as gram +ve and gram -ve. Gram +ve bacteria take blue-purple stains, like Corynebacterium, Clostridium and Listeria, the gram -ve bacteria take red-pink stains, for as, *Pseudomonas*, *Proteus* and *Klebsiella*.[21]

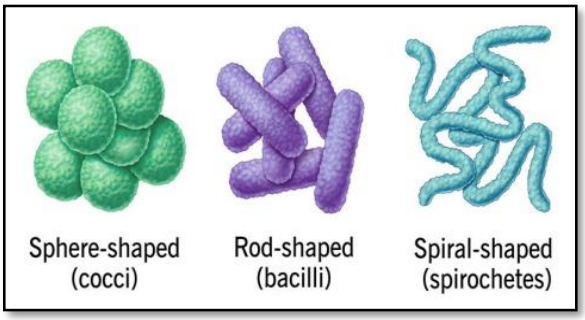

Figure 1: shape difference of bacteria.[21]

Fungi

Fungi grow with the structure of hyphae which become cylindrical, thread-like structures of a diameter of 2-10 µm and a length of several centimeters. Further, the process of branching of the Hyphae gives rise to a new one, and the interconnected network is called mycelium (Figure 2).[22,23] Hyphae can be either septate that divides into compartments including one or more nuclei separated by cross walls and coenocytic types of hyphae are uncomparted multinucleate supercells. [24,25]

Virus

Virus/virions are microscopic pathogens, that have genetic information (DNA/RNA) bounded by a protective shell, known as a capsid (necked virus) and sometimes an extra coat also found and named envelope. Viruses

provide instructions to host cells, which they employ to replicate themselves (Figure 3).[27]

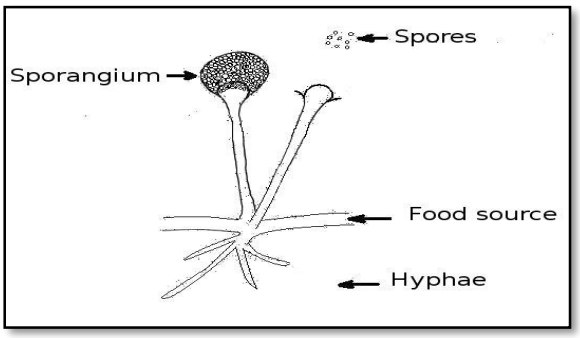

Figure 2: Microscopic structure of fungi.[26]

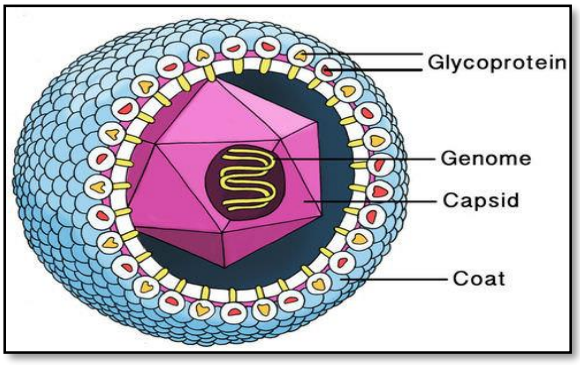

Figure 3: The virus.[28]

The reproduction process involves; attachment to the host cell. Entry can occur by receptor interaction with the host cell or direct fusion with bacteriophages, which transfer genetic material into bacterial cells (Figure 4).

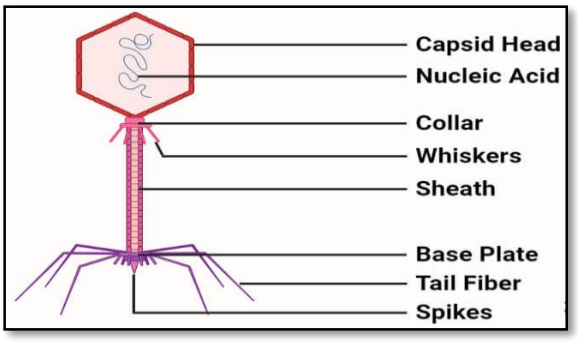

Figure 4: The bacteriophage.[28]

Replication of genetic material takes place through either the lytic cycle where host cells are used to produce various copies of them and the cell bursts to infect other cells or follow the lysogenic cycle where virion becomes dormant inside the host cell and reproduces along with the host cell till the trigger where host cell bursts. Assembly and then Release.[27] Viruses have a vast range according to size, molecular weight and their different external geometric structures, according to the shape they are; Icosahedral or polyhedral viruses are the commonest soccer ball-shaped. Helical viruses, look like a cylinder with spring coiled like genetic information inside. Spherical viruses are identical to helical or polyhedral viruses, except they have an envelope around them. Complex viruses incorporate several shapes. Bacteriophages are viruses that infect bacteria. They contain a polyhedral head and a helix body.[27] According to the genetic information of DNA or RNA, the genome can be categorized as linear or circular. Positive sense RNA (plus strand or positive strand) contains instructions for viral generation that do not require any additional steps. Negative sense (minus sense or negative strand) RNA viruses require certain enzymes to generate positive sense RNA. The majority of DNA viruses are regarded positively. Single or double-stranded DNA viruses can have either double strands (dsDNA) or a single strand of instructions (ssDNA) in their genetic material. Some RNA viruses are double-stranded, but most are single-stranded.[27]

Table 1: Classification of keratitis.

Classification	Types
Environmental Aetiology	Exposure keratitis, Photo keratitis, Contact lens acute red eye (clare)
Chronicity	Acute keratitis; Acute epithelial keratitis, Nummular keratitis, Interstitial keratitis, Disciform keratitis
	Chronic keratitis; Neurotrophic keratitis, Mucous plaque keratitis
Diseased Stages	Superficial punctate keratitis, Ulcerative keratitis
Infective	Bacterial, Fungal, Viral, Amoebic/parasitic

EPIDEMIOLOGY

Staph. aureus and streptococci are the commonest bacteria for keratitis and among them, *Pseudomonas aeruginosa* is responsible for 40% of keratitis infections in people who use contact lenses.[1] An outbreak of fungal keratitis was reported in 2005-2006 through the possible vector (cf. Fusarium) caused by the ReNu Moisture Loc contact lens solution of Bausch & Lomb.[16] The disabled and immunocompromised person was affected with yeast Candida albicans, as reported in North America. Although, these filamentous fungi are rarely reported in that terrain.[29] Corneal graft patients are more susceptible to the risk of fungal infection. In America, 50000 people have a corneal transplant and 4 to 7/10000 get infected with fungal infection. Onward 2007 to 2014, endophthalmitis was commonly countered twice in patients with corneal graft surgery. In the past, endophthalmitis was considered a bacterial infection but now fungi of candida species are estimated for two-thirds of infections of endophthalmitis.[13,30] A Brazilian report among 40% of samples regarding the traumatic infection

shows the effect of 67% of Fusarium sp., 10.5% of Aspergillus sp, and 10% of Candida sp.[31] Approx. 8-20% of cases of Varicella Zoster Virus (VZV) infect the eyes as herpes *zoster ophthalmicus* to form VZV keratitis in 13-76% of cases, after 1 month onset of symptoms. Initially, it forms a dendriform epithelial keratitis pattern which changes into the clouding of the cornea. In 50% of cases, its infiltrate involves the corneal stroma to cause corneal scarring.[1, 32]

A health advisory issued by the United States center for disease control for the increased risk of Acanthamoeba keratitis for the users of advanced medical optics complete moisture plus multi-purpose eye solution.[33] A retrospective study (December 2011 to January 2017) of pediatric patients of age from newborn to 16 years, diagnosed with traumatic infectious keratitis at a tertiary care centre in North India concludes 104 eyes of 104 children. The 88 (84.2%) eyes found affected on culture through the discharge/scrap of the eye indicate 54.2% for bacterial infection, 40.8% for fungal infection and 2.1% for acanthamoeba.[34] A prospective hospital-based study (January 1999 to June 2001) on 485 consecutive patients at Guru Nanak Eye Center, Maulana Azad Medical College, New Delhi, diagnosed mycotic keratitis in 191 (39%). The possibility of infection is counted as 42% through corneal trauma, 25% through contact lenses, and 21% through prolonged use of topical corticosteroids. The detected fungi were Aspergillus species in 78 (41%) and Curvularia species in 55 (29%).[35] A retrospective cross-sectional study for ten years includes Ninety-Three Patients between 1 to 60 months. The corneal scraping intended culture results most common risk factor for trauma (40.9%) followed by the use of contact lenses (8.6%). 28 (30.1%) patients were found negative for microbial growth and the positive were for *S. epidermidis* (10.8%) and *P. aeruginosa* (10.8%). Fluoroquinolone antibiotic (ciprofloxacin sensitivity 93.8%) was the most potent drug.[36]

SIGNS AND SYMPTOMS

The common signs and symptoms are redness/ swelling/pain or discomfort/lacrimation with discharge/ blurred vision or loss of vision/photosensitivity/feel of something stuck inside the eye.[7,37]

Bacterial keratitis

The bacterial keratitis presents lid swelling, degree of blepharospasm, conjunctival chemosis shows redness of the eye (circum-corneal/ciliary congestion of vessels), Pain and foreign body sensation (due to mechanical effects of lids and chemical effects of toxins), watering from the eye (reflex hyper-lacrimation), light intolerance (Photophobia), blurred vision, muddy Iris with constricted pupil, IOP may also rise (Figure 5). Later on, a corneal ulcer exist with yellow whitish colour established with hypopeon and the epithelial defects and infiltrates enlarge to develop stromal edema around the ulcer area. Some of the specific characteristics are oval, yellowish-white densely opaque ulcer surrounded by clear cornea- Staphylococcal aureus and streptococcus pneumonia. The semi-opaque area around the thick greenish mucopurulent exudates with hypopyon is represented by Pseudomonas species (perforate within 48 to 72 hours) and the shallow ulcer with greyish-white colour and diffuse stromal opalescence, Enterobacteria cause ring-shaped corneal infiltration (*E. coli*, *Proteus*, and *Klebsiella*).

Figure 5: Bacterial keratitis.[8]

Fungal keratitis

Fungal keratitis almost has the same symptoms as bacterial keratitis but some of the silent features are dry greyish-white with rolled margins of the ulcer. At the stromal layer, feathery finger-like projections are found. Multiple small lesion around the site of the ulcer is also found. Usually, Hypopyon is found even though with a smaller ulcer. Perforation is rarely found but vascularization is not found (Figure 6).

Figure 6: Fungal keratitis.[15]

Viral keratitis

Herpes simplex virus (HSV) is a DNA virus and its known natural host is human. It affects the skin of lids, the periorbital region and the lid margin (vesicular blepharitis). Acute follicular conjunctivitis with regional lymphadenitis. Epithelial keratitis either of coarse,

punctate or diffuse branching usually does not involve the stroma, thereafter dendritic ulcer is of irregular shape and branched with knobbed at their ending. The floor of the ulcer gets stained with fluorescein and the virus-laden cells at their margins are stained up with rose bengal. If the branching of the dendritic ulcer enlarges to some extent to form a large epithelial ulcer in a design of geographical or amoeboid pattern then named a geographical ulcer.

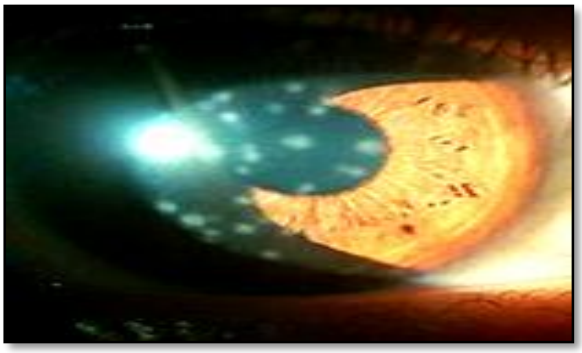

Figure 7: Adenoviral keratitis.[38]

The stromal keratitis concludes disciform keratitis is characterized by a focal disc-shaped patch of stromal oedema without necrosis, Descemet's membrane folds, keratic precipitates, a ring of stromal infiltrate (Wessley immune ring), diminished corneal sensations, increased Intra-ocular pressure (IOP) with mild anterior uveitis. Necrotizing interstitial keratitis (IK) is caused by active viral invasion of tissue followed by the destruction of tissue and the lesion appears as necrotic, blotchy, cheesy white infiltrates underlying the epithelial ulcer or present independently beneath the intact epithelium, mild iritis, (herpetic keratouveitis) with stromal vascularization may occur.

Figure 8: Acanthamoeba keratitis.[20]

Herpes Zoster Ophthalmicus is known as an acute infection of the Vth cranial nerve. Ocular involvement confirms itself if the nose tip or its side has a vesicle (nasociliary nerve engaged) called Hutchinson's Rule and the lesion is strictly limited to one-half of the face. Conjunctivitis is the most common consequence of herpes zoster, sometimes accompanied by mucopurulent petechial haemorrhages or severe follicular conjunctivitis with regional lymphadenopathy. Zoster keratitis comes in several kinds of epithelial keratitis. The condition might present as fine or coarse punctate epithelial keratitis with microdendritic ulcers, Nummular keratitis, caused by anterior stromal infiltrates, is characterized by many small granular deposits enclosed by a halo of stromal haze. Healing leaves nummular scars, and disciform keratitis is preceded by this condition (Figure 7). Episcleritis/scleritis and Iridocyclitis a frequent occurrences with hypopyon and hyphaema (acute haemorrhagic uveitis), Acute retinal necrosis may occur, anterior segment necrosis and phthisis bulbi resulting from zoster vasculitis and ischemia. Trabeculitis can cause secondary glaucoma in its early stages, followed by synechial angle closure in its later stages.

Acanthamoebic keratitis

The acanthamoebic keratitis first exhibits limbitis, radial kerato-neuritis, and epitheliopathy in the forms of punctate epitheliopathy/epithelial ridges/ pseudo or true dendrites/irregular epitheliopathy (Figure 8). At the advance of the disease, signs are patchy stromal infiltrates, satellite infiltrates, and central or paracentral ring infiltrates with overlying epithelial defects. At the severity hypopyon with ring abcess associates with stromal necrosis.

DIAGNOSIS

Clinical diagnosis is difficult and usually made by non-responsive patients who are treated for herpetic, bacterial or fungal keratitis. It is generally determined by an eye examination as well as signs and symptoms. Corneal scraping also plays an important role in evaluating it through microbiological culture. Initial treatment is not changed until the response is negative, then immediate change of drugs is as needed by the culture and sensitivity report.[1,7,37,39] Trauma with vegetative material and animal tails Secondary fungal ulcers, immune system suppression, very dry eye, bullous keratopathy, herpetic keratitis, or post keratoplasty, the late or non-healing chronic ulcer may lead to mycotic involvement. In recent trends, the faster diagnosis methods are Polymerase chain reaction (PCR) and confocal microscopy allow direct visualization of the hyphe and cysts etc. The scrapped sample is tested microscopically and with the standard diagnostic method of culture, carried out through wet KOH scrapping, Calcofluor white stain, which is a fluorescent brightener that stains the cysts of acanthamoeba as bright apple green under a fluorescence microscope. Lactophenol cotton blue-stained film is also useful for the demonstration of acanthamoeba cysts in the corneal scrapings. Grum's and Giemsa-stained films for fungal hyphae and culture on Sabouraud's agar medium. Culture on non-nutrient agar (*E. coli* enriched) may show trophozoites within 48 hours, which gradually turn into cysts. Prolonged use of medication causes symbiotic

disturbances between pathogens as antibiotics make disturbances between bacteria and fungi, and steroids make the disturbance between facultative pathogens. In the case of corneal infections/keratitis, Wessley's immune ring, triggered by corneal intrastromal responses, forms at the confluence of pathogen and host antibodies. The yellow demarcation develops at the adjoining area of fungal antigens and host antibodies. The stromal oedema signifies the junction between viral antigen and host antibody.

TREATMENT

The bacterial keratitis is treated with topical broad-spectrum antibiotic drops but if there is a corneal ulcer then specific treatment concludes, Fortified Cefazoline, 5% (50 mg/ml freshly prepared by adding sterile water to 500 mg powder to make 10 ml solution, and fortified tobramycin, 1.3%, (13.6 mg/ml prepared by adding 2 ml of tobramycin injection.

Table 2: Treatment of keratitis.

Pathogens	Species	Specific signs	Treatment
Bacteria	*Pseudomonas* *Staphylococcus* *Streptococcus*	Mucopurulent discharge, yellow-white infiltrate with clearly defined margins, hypopyon	Topical antibiotics, steroids and oral antibiotics
Virus	*Herpes simplex* Virus *Varicella Zoster* Virus	Clear discharge/epiphora, epithelial defect with linear branching and terminal bulbs (dendritic ulcer), reduced corneal sensation, hypopyon	Topical antivirals and cycloplegics
Fungi	*Aspergillus* *Fusarium* *Candida*	Mucopurulent discharge (less than bacterial), grey-white infiltrate with fluffy margins and satellite lesions, hypopyon	Topical antifungals and topical antibiotics and/or systemic antifungals
Protozoa	*Acanthamoeba*	Clear discharge/epiphora, ring-shaped infiltrate, reduced corneal sensation and hypopyon	Topical PHMB and chlorhexidine

Freshly prepared fortified vancomycin 5%, (50 mg/ml, prepared by adding sterile water to 500 mg vancomycin powder) and fluoroquinolones eye drops (0.3% ciprofloxacin. or 0.3% ofloxacin or 0.3% gatifloxacin or 0.5% moxifloxacin) are used. At any severity, Systemic antibiotics cephalosporin and an aminoglycoside or oral ciprofloxacin (750 mg twice daily) may also be used.[7, 21, 37, 39-42] Antibiotics work on the cell wall or DNA of bacteria and their prolonged use becomes resistant to the bacteria for example MRSA (methicillin-resistant *Staphylococcus aureus*). To reduce the ciliary spasm and to prevent posterior synechiae formation from secondary iridocyclitis cycloplegic drugs are used (1% atropine- increases the blood supply to anterior uvea by relieving pressure on the anterior ciliary arteries and so maximum antibodies pores out in the aqueous humour besides it exudation also reduces due to decrease off hyperaemia and vascular permeability) .

Systemic analgesics, anti-inflammatory drugs, Vit. A, B and C are used respectively to reduce pain, inflammation and early healing of an ulcer. The general measures are hot fomentation and dry heat can provide pain relief, decrease inflammation, and increase blood flow. Dark-coloured eyewear is used for relieving photophobia. Non-healing ulcers can be a result of elevated intraocular pressure, concretions, misdirected cilia, foreign bodies, dacryocystitis, inadequate therapy, incorrect diagnosis, lagophthalmos, and excessive vascularization. Systemic causes such as diabetes, severe anaemia, malnutrition, chronic debilitating diseases, and systemic steroids should also be considered. So Mechanical debridement of the ulcer is to be done for fast healing, and cauterisation of the ulcer with pure carbolic acid or 10-20% trichloracetic acid may apply, if the corneal vascularization is hindering the healing peritomy process is applied and the bandage soft contact lens may be applied over the cornea for faster healing. The fungal treatment for an eye depends on the type of fungus, the severity of the infection, and the affected area of the eye. The treatment is given through topical, oral, intravenous and direct injection routes. Sometimes surgery is also needed. The specific treatment includes topical antifungal drugs for a period of 6 to 8 weeks. Against Aspergillus und Fusarium fungi, effective drugs are Natamycin (5%), Amphotericin B (10-1.25 mg/ml), Fluconazole (0.2%)/miconazole (10 mg/ml)/ voriconazole (10%) eye drops initially instilled one hourly around the clock, then taper slowly over 6 to 8 weeks. Against Candida, the effective drug is Nystatin (35%) eye ointment, five times a day. The systemic antifungal drugs are tablet fluconazole or ketoconazole used for 2-3 weeks. The non-specific treatment is similar to that of bacterial keratitis. If the patient doesn't get better may need surgery, a corneal transplant and vitrectomy or, in extreme enucleation is needed. The antiviral drugs give a response within 4 to 10 days if it is not so, then the used drug is changed. Among the popular drugs is Aciclovir (Acycloquanosine) 3% ointment 5 times/day. Ganciclovir (0.15% gel), 5 times a day, Triflurothymidine 1% drops used two hourly, Vidarabine (Adenine arabinoside) 3% ointment 5 times/day are used and as the ulcer heals tapering is applied. The oral antiviral drugs Famcyclovir 250 mg, Valacyclovir 500 mg and Acyclovir 400 mg for a period of 10 to 21 days are also used. Sometimes mechanical debridement over the virus-laden cells is also applied. Protozoal keratitis is usually treated unsatisfactory but the non-specific

treatment concludes the same for the corneal ulcer and the specific treatment includes Multiple drug therapy of topical Antiamoebic agents are Diamidines- Propamidine isethionate (0.1%)/hexamidine (0.1%) and Biguanides- Polyhexa-methylene biguanide (0.02%) twice a day for a year is recommended /chlorhexidine (0.02%). Topical aminoglycosides- Neomycin, and Paromycin and topical imidazoles; Clotrimazole and miconazole. The oral medication may be added as ketoconazole 200 mg BD, or itraconazole 100 mg BD. Sometimes penetrating keratoplasty is also applied in non-responsive cases.

CONCLUSION

Keratitis and healed ulcers interfere with visual potential and some of the infections may cause corneal perforation and endophthalmitis resulting in loss of the eye, therefore within the time limit of starting the infection, the attention over the patient for the treatment makes the successful possibility for the restoration of the vision and loss of the eye. Acanthamoebic and fungal keratitis are difficult to treat and are associated with a poor prognosis so further research is required. Vaccination for zoster is highly effective for shingles, herpes *zoster ophthalmicus* and herpes zoster keratitis.

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