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INFECTIOUS KERATITIS – A REVIEW

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ABSTRACT

Infectious keratitis is an important problem in the Indian context, and epidemiological studies suggest a higher prevalence of disease as compared to the developed world. Various aspects of the problem, including the host defence, ocular and systemic risk factors and microbial mechanisms that enhance pathogenesis have been discussed. The clinical and microbiological approach to the infected eye and patient and the relevant tests have been outlined. The major classes of medications that are used the methods of delivery are specified. Finally, the approach to patients with non-responsive keratitis that requires surgical manoeuvres is also highlighted. Clinical experience is important in distinguishing an infective from an inflammatory corneal pathology. The role of an experienced microbiologist is inevitable, for good clinical outcome. Therapeutic keratoplasty has to be done at the right time for better clinical outcomes.

Key words: Infectious keratitis, non healing corneal ulcer, bacterial keratitis, fungal keratitis.

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INTRODUCTION

The epidemiology of blindness in the recent years has shifted to important causes, such as cataract, corneal trauma and infective keratitis[1] from traditional infectious causes, such as trachoma, Onchocerciasis, and leprosy. There is a preliminary report of significance from Madurai that 44% of all central corneal ulcers are caused by fungi.[2]This high prevalence of fungal pathogens in south India is significantly greater than that found in similar studies in Nepal (17%)[2], Bangladesh (36%)[3], and south Florida (35%).[4] It is essential to determine the local aetiology within a given region when planning a corneal ulcer management strategy. The most common cause for bacterial keratitis is known to be *Streptococcus pneumoniae* based on various studies done.[5] With regard to fungal keratitis the strain causing infection varies between the south and north of India. In the south, of the 155 fungal isolates cultured from 154 corneal ulcers 47.1% were *Fusarium* species, and 16.1% were Aspergillus species.[6] In another study from south India, Fusarium (506, 37.2%) and Aspergillus species (417, 30.7%) among the hyaline fungal spectrum and Curvularia species (39, 2.8%) among the dematiaceous isolates, were the predominant organisms seen. [7] Whereas in North India, the spectrum of fungi isolated were *Aspergillus* species in 78 (41%) followed by Curvularia species in 55 (29%).[8] In another study, Aspergillus flavus was the most common fungus isolated in 31.16 per cent cases, followed by A. fumigatus (16.88%) and Fusarium spp. (7.79%). Yeasts were also isolated in 21.62 per cent cases. [9]

Types of Corneal Infections-

Bacterial keratitis progress rapidly with patient's complaining of rapid onset of pain, photophobia, and decreased vision. The most common groups of bacteria responsible for bacterial keratitis

are: Streptococcus Pneumonia, Pseudomonas, Enterobacteriaceae (including Klebsiella, Enterobacter,Serratia and Proteus),and Staphyloco ccus species. Upto 20% of cases of fungal keratitis (particularly candidiasis) are complicated by bacterial co infection. Acute pain with watering and rapidly spreading corneal ulcer is likely to be due to Pseudomonas aeruginosa and Streptococcus pneumoniae. The ulcer usually present with severe conjunctival congestion, epithelial defect, distinct margins, wet slimy surface, mucopurulent discharge, stromal inflammation with infiltrates and suppuration, DM folds, severe AC reaction, synechiae, Hypopyon (occurs as a reaction to the released exotoxins which is usually sterile with fluid like consistency and is mobile). Hypopyon is also an important sign of Pneumococcal or Pseudomonas ulcer. Hemorrhagic hypopyon is associated with pneumococcal or Herpes simplex keratitis.[10] Gram Positive Cocci are white discrete stromal infiltrates with minimal haze forming small abscess like lesions. Staphylococcus is seen in compromised eyes with Bullous keratopathy and in chronic herpetic keratitis. Pneumococci have a progressive edge with other edge healing. Gram Negative Bacilli are rapid and corneal destruction may be complete in 24-48 hours. Pseudomonas causes severe inflammation, greenish discharge, marked corneal melt, ring infiltrates, surrounding stroma has ground glass appearance, diffuse greying of epithelium.

The characteristics of atypical bacteria are:

Mycobacteria presents with a cracked wind shield appearance with spoke like margins. Its often seen in post Lasik eyes and heals slowly. Nocardia occurs from minor trauma or exposure to soil. It has multiple, raised, superficial, white small pin head, wreathe like infiltrates with small filaments and bush fire like borders. Bacillus is a gram positive Bacilli occurring post trauma commonly in broom stick injury, with rapid aggressive peripheral stromal abscess. The following ring ulcer is distinct stromal infiltrate remote from area of trauma. Moraxella which is a gram negative diplo bacilli, occurs in malnourished, diabetics and alcoholics. They are superficial with moderate oedema and minimal infiltrates.

Fungal keratitis is characterised by insidious onset with an indolent course. Mainly caused by yeast (mainly candida) and filamentous (septate and nonseptate) fungi. Fungal organisms usually extend from mid periphery to the centre of cornea or can even extend from the cornea into the sclera and intraocular structures.[11] Filamentary Fungi present with an epithelial defect, infiltrate with hyphate feathery ill defined edges, elevated and dry surface with rough texture, greyish white appearance. Presence of brown pigmentation, satellite lesions and ring like infiltrates are other characteristic findings. Hypopyon (contains fungal filaments, thick, immobile), stromal abscess, yellow immune ring and endothelial plaque are also seen. Candida may resemble staphylococcal, Moraxella ulcers, Stromal herpes and other low virulent bacterial ulcers. [12]

Viral keratitis is the most common cause of infectious blindness in the Western world. Patient usually present with photophobia, redness and watering. Mainly caused by Herpes Simplex Virus type 1, Herpes Zoster Virus and Adenovirus. Herpes Simplex Virus -Primary herpes infection of the eye usually unilateral blepharo conjunctivitis with characteristic vesicles on the skin of the lids, follicular conjunctivitis, preauricular lymphadenopathy, and occasional punctate keratitis. After primary infection, recurrence rates of Herpes simplex keratitis (HSK) are about 25 percent within 1 year and 33 percent within 2 years and 63% in 20 yrs.[13] Earliest signs are raised SPK, Macro Dendritic ulcers (commonest presentation, linear branching pattern with terminal bulbs, swollen epithelial borders that contain live viruses,, and central ulceration through the basement membrane taking double stain), sensation reduced. Geographic ulcer – broad epithelial defect wherein borders are scalloped, irregular and angulated Marginal ulcer - dendrite develops close to the limbus, its anterior stroma gets infiltrated by leukocytes from the limbal blood vessels Metaherpetic /Persistent Epithelial Defect-occurs

due to damaged basement membrane, antiviral toxicity or neurotrophic component. Its characterised by heaped up edges, smooth ovoid contour, irregular corneal surface, located in the central or inferior paracentral area, and usually lies within the interpalpebral fissures. Stromal Keratitis can be primary or secondary (to infectious epithelial keratitis, neurotrophic keratopathy, or endotheliitis).Two forms are Necrotizing Stromal Keratitis and Immune Stromal Keratitis. Necrotizing Stromal Keratitis presents with dense stromal infiltrate, ulceration and necrosis (viral replication occurs within stromal keratocytes resulting in severe host inflammatory response) which usually results in thinning and perforation within a short period. It is usually a diagnosis of exclusion from fungal/bacterial/Acanthamoeba keratitis/retained FB/drug toxicity. Immune Stromal Keratitis (ISK), also known as Non necrotizing Stromal Keratitis and Stromal Interstitial or Disciform Keratitis, is a manifestation of chronic, recurrent ocular HSV disease. ISK may clinically present with focal, multifocal, or diffuse cellular infiltrates which may obscure the underlying endothelium, wessley immune rings appear as granular greyish ring formed by antibody complement cascade. Recurrent viral infection are characterised by neovascularisation, ghost vessels or scaring.[14] Endothelitis present with keratic precipitates (KP), minimal iritis, endothelial guttate, oedema of overlying stroma and epithelium without stromal infiltrate or neovascularisation. It can be classified as Disciform Endotheliitis and Diffuse Endotheliitis. Disciform Endotheliitis present with corneal oedema in a central or paracentral region with a clear demarcation between involved and uninvolved cornea. Always assess the iris for segmental ischemic necrosis which may occurwith or without trabeculitis that may cause a secondary IOP rise.[15] Varicella Zoster - Herpes zoster ophthalmicus (HZO) occurs when reactivation of the latent virus in the trigeminal ganglia involves the ophthalmic division of the nerve. 20% of primary infection reactivates. The virus damages the eye and surrounding structures by secondary perineural and intraneural inflammation of sensory nerves. The prodromal phase of herpes zoster ophthalmicus includes an influenza-like illness with fatigue, malaise, and low-grade fever that lasts for a week, before the rash over the forehead appears, along with varying degrees of dermatomal pain. Subsequently, erythematous macular lesion rapidly progress to papules and vesicles containing clear serous fluid and, later, pustules. These lesions rupture and typically crust over, requiring several weeks to heal completely. They strictly occur on one side of the midline. They present with follicular conjunctivitis, blepharitis. Corneal complications occur in 40 percent of cases o f HZO. Corneal presentation include subepithelial punctate keratitis, Pseudo Dendrites (which are multiple, smaller with tapered ends, without bulbs and central ulceration occurring mainly at periphery), Multiple superficial stromal nummular fine infiltrates, deep stromal infiltrates with lipid and vascular infiltration. Severe cases may also present with uveitis, ARN, PORN, optic neuritis, EOM palsies.

Adenovirus: In the first week usually present with conjunctivitis, pseudomembrane, pre auricular lymphadenopathy and dryness due to lachrymal gland inflammation. Second week corneal involvement may occur with diffuse, fine punctuate epithelial keratitis (PEK) that stains with fluorescein and rose bengal. Third week, immune reaction takes over with subepithelial keratopathy and the viral penetration and inflammation is only up to the bowmans layer. It usually persists for 2-3 weeks. Corneal sensations are normal.

Parasitic Infections

Acanthamoeba Keratitis is now often reported with much frequency due to increased use of contact lens wearers. Exposure to muddy or brackish water is also important predisposing factors. It's more significant among the infectious category because of the delay in its diagnosis. Has a cyst form (dormant) and a trophozoite form (active).30 Bacteria and fungi sometimes coat the surface of the trophozoite that may result in a mixed

infection. Acanthamoeba binds through mannose glycoprotein of corneal epithelium and secretes proteins which are cytolytic to the epithelium and proteases that help in further penetration. Its characterised by pain disproportionate to signs, radial keratoneuritis, mid peripheral stromal ring infiltrate with intact epithelium, satellite lesions and pseudodendrites. May also be associated with scleritis or limbitis. High degree of suspicion should occur, especially in a young contact lens wearer with a recent diagnosis of keratitis, who is not responding to therapy. Steroids should never be instilled even on resolution, as it may convert trophozoite form to cystic form which may activate later.

Microsporidia are obligate intracellular eukaryotic pathogens known to cause superficial punctate keratitis and stromal keratitis in both immune compromised and immune competent individuals. They have been recently reclassified as fungi. Microsporidial keratitis is suspected when an SPK responds but later recurs on tapering steroids. Microscopic examination of a diagnostic epithelial scraping reveals aggregates of intracellular organisms in the cytoplasm of epithelial cells consistent with microsporidia. Steroid use should be discontinued, and resume treatment with topical voriconazole, 1%, every 2 hours.

Assessment of Healing

Healing ulcer is characterised by re epithelisation, blunting of the hyphate edges, decrease in stromal infiltrates, decrease in stromal oedema and endothelial plaque, cessation of corneal thinning, decrease in the anterior chamber reaction, height and surface of hypopyon. If the ulcer is not healing then advice for recapture to confirm initial diagnosis, recheck the anneal structures, repeat blood sugar levels. We as well initiate alternative treatment modalities like Intrastromal, intracameral or subconjunctival antimicrobial therapy.[16] Surgery is warranted in case of perforated corneal ulcer, impending perforation, if ulcer is not healing in spite of appropriate and adequate therapy for 1 week, fulminant ulcer that's not showing any response in three days, a slow healing ulcer for 1 month

Clinical Features-

Infectious: Usually has a traumatic onset, slow to fast progressing, minimal to severely symptomatic with sharp aching pain. Conjunctival and Ciliary hyperaemia with lid oedema and discharge are present. Ulcers are central or superior, more than 1 mm and superficial to deep with epithelial defect, swollen edges, dry/wet surface. May also present with pseudo guttate, DM folds and endothelial plaque. They are usually associated minimal to marked AC reaction, KP's and Hypopyon[33]

Inflammatory: Usually due to Contact lens, dryness, OSD, immune systemic diseases with a slow progress, symptomatic with FB sensation. There is minimal conjunctival congestion, with Ciliaryhyperaemia. Infiltrates are peripheral, less than 1 mm, single/multiple, well defined, subepithelial or in superficial stromal. Lucid interval is present between limbus and infiltrates. There is minimal AC reaction with or without hypopyon.

Importance of detailed drawings-

Standardised schematic representation of corneal disorders is essential for proper follow up of patients, and also for clinical research. It improves clinician's focus and treatment skills. It may come in handy during medico legal issues. Following colour coding is generally used to document the findings of anterior segment

Black: Contact lens, Limbus, Sutures, Foreign bodies, Scars, Degenerations, Band keratopathy

Blue: Shades for Stromal Oedema, Small circles for epithelial oedema, Wavy lines to document folds in Descemets membrane

Brown: Pupil, Pigmentation, iron, melanin and iris defects

Red: Blood vessels, Rose Bengal staining, Haemorrhages

Orange: Hypopyon, Keratic precipitates

Green: Fluorescein staining of cornea, Punctuate epithelial keratopathy (dots), Filaments (small lines), Epithelial defects, Lens and vitreous haze

Diagnosis

Table 1: Laboratory diagnosis

Stain	Organism	Comments
Grams	Bacteria, Fungi , Acanthamoeba, Microsporidia	-
Giemsa	Bacteria, Fungi , Acanthamoeba, Microspordia, Chlamydial Inclusions	-
10% KOH	Fungi	Corneal tissue digested by KOH
Periodic Add Schiff	Fungi	Fungal elements Take up Magenta colur
Gomori Methenamine Silver	Fungi	Fungal flaments Seen in black colour
Calco Flour White	Fungi, Acanthamoeba	Fluorescent Microscope
ZiehlNeelsen	Microbacteria, Nocardia	-
Immunoflorescent	Virus, chiamydia	-
Acridine Orange	Bacteria, Fungi, Acanthamoeba,	-
Cold Carbol Fuchsin	Nocardia	-

Table-2: Laboratory diagnosis

Agar	Organism	Comments
Blood Agar	Aerobic Bacteria, Saprophtic Fungi	37^0 for Bacteria, Room temp. for Fungi
Chocolate Agar	Haemophiilus, Neisseria, Moraxella,	5-10% CO_2
Brain Heart Infusion	Bacteria, Fungi	-
Sabourauds Dextrose	Fungi	Room temperature
Thioglycolate Broth	Anaarobic And Aerobic Bacteria	-
Lowenstein Jensen Agar	Atypical mycobacteria	-
E. Coli Plated Non Nutrient Agar	Acanthamoeba	-

Treatment specific medications

Table-3: Anti microbial medications and dosages

Organism	Strain	Topical Drug
Bacteria	Gram Positive Cocci	Moxifloxacin/Gatifloxacin
		Cefazolin (50 mg/ml)
	Gram Negative Bacilli	Amikacin (20 mg in 0.5ml)
		Cefotaxim (50 mg/ml)
	Atypical	Amikacin (20 mg in 0.5 ml)
		Moxifloxacin/ Gatifloxacin

Table-4: Anti microbial medications and dosages

Organism	Strain	Topical Drug
Fungal	Filamentary-South-Fusarium	Amphotericin B (0.15%) eye drops
		Natamycin 5% eye drops
		Tab. Voriconazole 100 mg twice daily Tab Itraconazole 200 mg twice daily
	Filamentary-North -Aspergillus	Amphotericin B(0.15%) eye drops
		Voriconozole/Natamycin eye drops
		Tab. Voriconazole 100 mg twice daily Tab Itraconazole 200 mg twice daily
	Candida	Amphotericin B (0.15%) eye drops
		Fluconazole eye drops
		Tab Fluconazole 150 gm

Table-5: Anti microbial medications and dosages

Organism /Strain	Topical Drug
Adenovirus	Tear substitutes every one hour
	Vigamox 2 times a day
	Flouromethalone eye drops 3 times a day tapered in 5 days
	Oral diclofenac twice daily x3 days.

Table-6: Anti microbial medications and dosages

Organism /Strain	Topical Drug	
Adenovirus	Tear substitutes every one hour	
	Vigamox 2 times a day	
	Flouromethalone e/d 3 times a day tapered in 5 days	
	Oral diclofenac twice daily x3 days.	
Herper Simplex Virus	Punctate Epithelial keratitis	Tear substitutes
	Metaherpetic/Persistent epithelial defect	Vigamox 2 times a day
	Stop all medications	Preservative free tear substitutes
	Punctal Plugs/ Banadage Contact Lens	Flouramethalone 3 times a day tapered weekly
	Tab Doxy 100 mg od × 10 days	Tab Vitamin C 2 tab qid
	Lateral tarsorrhaphy	Glue Banadage Contact Lens/Amniotic Membrane Transplantation inlay with overlay
	Dendritic Ulcer	Acivir eye ointment 5 times a day
		Atropine eye drops 2 times a day
	Stromal Disciform	$1/8^{th}$ Betnesol eye drops 4 times a day
		Atropine eye drops 2

		times a day
	Stromal Necrotizing	Tab Acivir 400 mg 1-0-1× 2weeks
		1/8th Betnesol eye drops 2 times a day
		Atropine eye drops 2 times a day
	Disciform Endothelitis	Tab Acivir 400 gm 1-0-1 × 2 weeks
		1/8 th Betnesol eye drops 4 times a day
		Atropine eye drops 2 times a day

Other medications

1.Cycloplegics- atropine e/d b.d – relieves pain due to ciliary spasm, prevents synechiae, brings more antibodies to aqueous humor and reduces exudation.

2.Topical steroids: prescribed after resolution of infection is ensured, helps to avoid scarring.

3.Systemic analgesics and anti inflammatory: relieves pain and edema

4.Vitamin C helps in collagen synthesis.

5.Anti-glaucoma-if pressures are high-tab acetazolamide ½ tab t.i.d

Surgical management-

Glue BCL – Ulcer should be located away from limbus. Cyanoacrylate glue is bacteriostatic and longer lasting than fibrin glue. It is also believed to inhibit polymorphonuclear lymphocytes and the production of collagenases, which may halt the corneal melting process.

Intracameral / Intrastromal injections

1. Intra stromal: one or more Intrastromal injection of antimicrobials at the junction of clear cornea and infiltrates, using a 30-gauge needle in five quadrants to form a barrage around the ulcer.
   a. Voriconazole 5%(50 µg in 0.1 ml)
   b. Fluconazole 0.2% (2 µg in 0.1 ml)
   c. Ampho B 0.7% (7.5 µg in 0.1 ml)
2. Intra cameral : injections of antimicrobial through a paracentesis
   a. Voriconazole 5%(50 µg in 0.1 ml)
   b. Fluconazole 0.2% (2 µg in 0.1 ml)
   c. Ampho B 1% (10 µg in 0.1 ml)

Patch grafts

Corneal patch grafts, either lamellar or full-thickness, can be used temporarily for central corneal perforations (> 3 mm) or permanently for peripheral corneal perforations or descemetoceles. Ideally, all necrotic tissues and epithelium are removed from the bed of the ulcer or margins of the corneal perforation until a viable tissue is reached before a lamellar disc of donor cornea or patch graft is sutured in place with interrupted 10-0 nylon sutures.

Lamellar Grafts

Lamellar keratoplasty virtually eliminates the risk of graft rejection. But the disadvantages of lamellar keratoplasty are intralamellar neovascularisation and incomplete removal of pathogens.[17] Lamellar keratoplasty has been performed for Acanthamoeba, fungal, herpetic, post-laser in-situ keratomileusis (LASIK) Mycobacterial keratitis, post Gonococcal keratitis, descemetoceles and graft infection.[18]

Therapeutic Penetrating Keratoplasty

The primary aim of the procedure is to eliminate the infectious disease process and establish the integrity of the globe. This procedure offers a microbiological cure rate of up to 100% in bacterial keratitis; recurrence of infection remains a concern following fungal, Viral and Acanthamoebic keratitis. Indicated when ulcer does not respond despite the maximum therapy applied.[19]

1. During the TPK

a. Place the appropriate trephine over the cornea and create an indentation in the epithelium.

b. Use lid speculum

c. Suture a Fleringa ring in place to provide scleral support,

d. In cases of large ulcers that reach up to the limbus, peritomy is required and homeostasis is achieved by the use of wet-field cautery.

e. If possible, a 1 mm rim of healthy corneal tissue should also be removed to leave a stable, no infected recipient bed.

f. Careful partial-thickness trephination with a Sharp trephine is done in the absence of any perforation

g. In eyes with a perforation,

i. Support is obtained with cyanocrylate and viscoelastic protection and anterior chamber can be reformed, care should be taken to avoid exerting excessive pressure on the globe to prevent extrusion of the ocular contents.
ii. A freehand dissection of the host bed may be done.
h. Clearing the anterior chamber of exudates
(i) Irrigation of the anterior chamber is done using medicated a balanced salt solution.
(ii)Removal of cataracts should be deferred because the lens forms an effective barrier that prevents the spread of infection into the vitreous.
i.Donor button should be trephined after the size of the recipient opening is measured and preparation of the host bed, because necrotic tissue may require additional trimming which may alter the size of the graft.
Suturing: by 10-0 monofilament Nylon interrupted sutures passing though at least 70% depth of the host cornea is the preferred technique. Full thickness bites are not taken as they may form a conduit for passage of infection from the cornea into the anterior chamber. It is not uncommon to use greater number of sutures than conventional technique of keratoplasty (16 Sutures) [20, 21] See table 8 for guidelines for post operative management of therapeutic keratoplasty.
Evisceration / Enucleation: Corneal ulcers that result in the loss of eye in elderly population[22]
a.Are frequently associated with glaucoma and persistent epithelial defects.
b.The majority of these cases have non-healing microbial keratitis caused by pseudomonas aeruginosa.
c.Present late to clinic
d.Suffer from long standing severe pre-existing ocular disease due to systemic associations like rheumatoid arthritis.

CONCLUSIONS

Corneal ulcers from an important part of the disease spectrum seen by the Ophthalmologist in India, not just because of its prevalence, but also because of the severe ocular morbidity that occurs when managed inappropriately. This arrticle addresses some important aspects of thos problem. Following the principls described can result in good outcomes in the majority of patient with infectious keratitis.

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