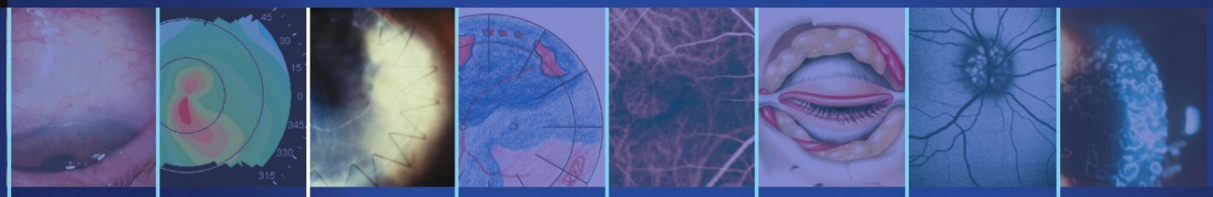
ESSENTIALS IN OPHTHALMOLOGY

G. K. KRIEGLSTEIN · R. N. WEINREB

Series Editors



Glaucoma | Cataract and Refractive Surgery | Uveitis and Immunological Disorders | Vitreo-retinal Surgery | Medical Retina | Oculoplastics and Orbit | Paediatric Ophthalmology, Neuro-ophthalmology, Genetics | Cornea and External Eye Disease

Uveitis and Immunological Disorders

Edited by

U. PLEYER

C. S. FOSTER

Springer

Essentials in Ophthalmology

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Uveitis and Immunological Disorders

With 88 Figures, Mostly in Colour
and 22 Tables

Springer

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Foreword

The series *Essentials in Ophthalmology* was initiated two years ago to expedite the timely transfer of new information in vision science and evidence-based medicine into clinical practice. We thought that this prospicient idea would be moved and guided by a resolute commitment to excellence. It is reasonable to now update our readers with what has been achieved.

The immediate goal was to transfer information through a high quality quarterly publication in which ophthalmology would be represented by eight subspecialties. In this regard, each issue has had a subspecialty theme and has been overseen by two internationally recognized volume editors, who in turn have invited a bevy of experts to discuss clinically relevant and appropriate topics. Summaries of clinically relevant information have been provided throughout each chapter.

Each subspecialty area now has been covered once, and the response to the first eight volumes in the series has been enthusiastically positive. With the start of the second cycle of subspecialty coverage, the dissemination of practical information will be continued as we learn more about the emerging advances in various ophthalmic subspecialties that can be applied to obtain the best possible care of our patients. Moreover, we will continue to highlight clinically relevant information and maintain our commitment to excellence.

G. K. Krieglstein
R. N. Weinreb
Series Editors

Preface

This second volume of *Uveitis and Immunological Disorders* in the *Essentials in Ophthalmology* series provides the reader with up-to-date and relevant information. Our knowledge and understanding of immune-mediated diseases has increased exponentially over the past few years, especially in the areas of immunopathogenesis and immunogenetics. This volume will provide the practitioner with practical information on how to diagnose and treat these difficult, and in some cases, blinding disorders. In addition, there are important discussions of the mechanisms underlying these conditions that incorporate the most recent, up-to-date research material available. The features "Summary for the Clinician" and "Core Messages" enhance the value of the chapters by helping the reader focus on the important messages in each chapter.

The scope of chapters ranges from diseases that are relatively common and usually require only topical therapy, such as ocular allergy and dry eye, to diseases that may result in blindness, such as contact lens-associated infections, autoimmune keratitis and some forms of uveitis. Several topics, for example handling corneal graft rejection and cataract extraction in uveitis patients are of particular interest for the ocular surgeon. Two chapters focus on recurrent ocular infections, herpes keratitis and ocular toxoplasmosis, which still remain sight-threatening disorders. Our better understanding of the underlying immune pathology has resulted in new treatment approaches, which are highlighted by experts on anti-TNF and gene therapeutic strategies.

This volume contains information of interest to a wide range of ophthalmic subspecialists. For example, the anterior segment subspecialist would have an interest in subjects such as contact lens associated infections, autoimmune Keratitis, ocular allergy, dry eye, corneal transplantation and herpes keratitis. Retina and uveitis specialists have a special interest in the chapters dealing with uveitis and its mechanisms and latest aspects in therapy. Lastly, the chapters on optic neuritis and neoplastic masquerade syndromes are important for interdisciplinary handling of these patients.

We are glad that the previous volume of *Uveitis and Immunological Disorders* had a broad readership and positive acceptance, which is underlined by the fact that it has been translated into Chinese and Italian.

We are sure that this edition will also reach its audience and would like to thank all authors who contributed their valuable time to complete this volume.

U. Pleyer
C. S. Foster
Volume Editors

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Chapter 1

Contact Lens-Related Corneal Infection

1

Barry A. Weissman, Anthony J. Aldave, Bartly J. Mondino

Core Messages

- Complications of contact lens wear are numerous and occur in all external ocular tissues. Only microbial keratitis (MK) and neovascularization, however, are common causes of associated loss of vision.
- MK is an ophthalmic emergency because of the potential for loss of vision.
- Contact lens wear has become a major risk factor for MK, joining trauma, dry eye, and preceding corneal surgery (e.g., cataract extraction, penetrating keratoplasty, refractive surgery).
- Extended wear and poor contact lens care remain the major risk factors for contact lens-associated MK.
- Using modern, highly oxygen permeable contact lenses (Dk values of 100 Fatt units or greater) under open-eye conditions should result in corneal oxygenation similar to that found without any contact lenses. Use of high-Dk rigid and soft lenses for extended wear may moderate the risk of MK, but may not reduce it to the levels found with daily wear of contact lenses.
- Bacteria cultured from contact lens-associated MK are commonly *Pseudomonas* sp. and *Staphylococcus sp.* Bacterial MK is more commonly associated with extended contact lens wear as well as poor contact lens care and hygiene.
- Milder, less threatening, presumed bacterial MK is often initially treated with topical antibiotic monotherapy and close professional supervision, but more severe and/or central infections should first undergo laboratory investigations (cultures, smears, stains) and then be treated aggressively with fortified antibiotics. The clinician should always remain suspicious of *Acanthamoeba* in any contact lens-associated MK.
- *Acanthamoeba* MK is more commonly associated with daily wear, poor contact lens care, and lens exposure to fresh water as opposed to proper contact lens care solutions. *Acanthamoeba* infections can masquerade as herpetic or fungal keratitis in particular, and pain is often out of proportion to the clinical signs.
- Steroid treatment of contact lens-associated MK remains controversial.
- Customized rigid gas permeable contact lenses can often improve vision dramatically after MK has healed, decreasing the need for corneal transplantation.

1

1.1 Introduction

The traditional major risk factors for microbial corneal infection (microbial keratitis or MK) include trauma and preceding corneal compromise such as surgery (e.g., cataract extraction, penetrating keratoplasty, refractive surgery) or herpetic corneal disease. Other participating factors include systemic (e.g., HIV infection, diabetes) and local (topical steroid treatment) immunosuppression, acne rosacea/blepharitis, severe dry eye, and corneal exposure. Contact lens wear has emerged as another risk factor for MK during the past 50 years [14, 43].

Contact lens wear has a long list of potential complications including edema of the various corneal layers, corneal abrasions and neovascularization, and lens soilage and its sequelae (i.e., giant papillary conjunctivitis; Table 1.1), but most patients rarely experience problems that result in permanent vision loss.

Summary for the Clinician

- Complications of contact lens wear can affect all ocular tissues, but are usually benign if patients refrain from sleeping or napping with contact lenses in their eyes, and when patients are compliant with good contact lens care and appropriate hygiene.
- Most complications are self-limiting, reversing even without medical treatment when contact lenses are removed.
- Both MK and neovascularization, however, may result in more serious vision compromise.

Microbial keratitis is unfortunately also a complication of contact lens wear, and, while rare, contact lens-related MK is a sight-threatening disease. For this reason, MK is considered an ophthalmic emergency. Even when "successfully" treated, MK can result in corneal scarring and neovascularization leading to the loss of central corneal clarity, necessitating a corneal transplant in an effort to restore vision. Unsuccessful management may result in the permanent loss of visual function and perhaps even the loss of an eye. Because it can result in a substantial loss of vision, MK is the contact lens wear-associated complication of most concern to both patients and practitioners alike.

Microbial keratitis is identified by the symptoms of sudden-onset ocular pain or foreign body sensation, decreased vision, photophobia, conjunctival vascular injection, discharge and/or lid crusting, blepharospasm, and by the observation of clinical signs of a corneal epithelial/stromal defect with associated inflammatory response (corneal infiltration). MK is often accompanied by an anterior chamber reaction (including a hypopyon in some cases) and lid swelling. Stein et al. [55] found that culture-proven contact lens-associated bacterial corneal infections were more likely:

1. When lesions were single and large rather than multiple, arcuate or small;
2. With epithelial defects, conjunctival discharge, and anterior chamber reactions;
3. When patients were more rather than less symptomatic (pain and photophobia) (Table 1.2).

When suspicious signs and symptoms are found in a contact lens wearer, lesions should be assumed to be infectious in nature and treated accordingly (see below for treatment protocols) until proven otherwise. Whenever any of the signs or symptoms of corneal infection occur, contact lens wear should also be immediately discontinued in both eyes to decrease the potential for bilateral disease.

To add to clinical confusion, however, both corneal infiltrates and epithelial erosions (varying from mild staining to frank abrasion) can occur as nonconcomitant lesions and as such are often noninfectious. Causes include hypoxia, toxic or hypersensitivity reactions, mechanical lens defects and poor fits, lens over-wear, and foreign bodies. Treatment of either of these complications may be similar or differ from that of corneal infection, but is beyond the scope of this chapter.

Other causes of red, painful eyes not specifically associated with contact lens wear that must also be considered in the differential diagnosis include conjunctivitis (allergic as well as infectious), glaucoma (especially acute angle closure), and both iritis and uveitis.

Table 1.1 Physiological complications of contact lens wear. (From [19], with permission.) *GPC* giant papillary conjunctivitis, *SEAL* superior epithelial arcuate lesion, *MK* microbial keratitis

Tissue	Complication type: probable cause(s)
Lids	Toxicity: solution sensitivity
	Allergy: papillary conjunctivitis; GPC due to lens soilage
	Ptosis: GPC; lens insertion and removal
	Blepharitis: bacterial; meibomian gland dysfunction
Bulbar conjunctiva	Injection: mechanical irritation, dry eye; solution sensitivity; hypoxia
	Edema: mechanical irritation; solution sensitivity
	Staining: mechanical irritation; solution sensitivity
Corneal epithelium	3–9 stain: desiccation; contact lens edge chafing
	Pancorneal stain: solution sensitivity; toxicity; blepharitis
	SEAL: mechanical lens problem; lens soilage
	Inferior arcuate stain: desiccation through soft lens
	Foreign body tracks: mechanical foreign body or lens defect
	Cluster stain: contact lens over-wear; hypoxia
	Inferior band (exposure) stain: dry eye (exposure keratopathy); blepharitis
	Abrasion: mechanical foreign body or lens defect; hypoxia; flat contact lens fit, keratoconus, anterior basement membrane dystrophy
	Dimple veil: air bubbles trapped in the tears between the lens and the anterior corneal surface
	Infiltration: infection (viral, bacterial, etc.); solution sensitivity; hypoxia
	Edema (microcysts): hypoxia, endothelial cell dysfunction
Corneal stroma	Edema (central corneal clouding or stromal striae); hypoxia; endothelial cell dysfunction
	Infiltrates: infection (viral, bacterial, etc.); solution sensitivity; hypoxia
Neovascularization	3–9: pseudopterygium: chronic desiccation; chronic lens edge defects, chafing
	Pannus: hypoxia; noncontact lens cause
	Deep stromal vessels: hypoxia; noncontact lens cause (e.g., MK, lues, keratoconus)
Corneal endothelium	Blebs: acute hypoxia, Fuch's dystrophy
	Polymegathism: chronic hypoxia; ageing; anterior segment surgery; Fuch's dystrophy
Microbial corneal infection	Bacterial, protozoal (amoebic), fungal. Viral

Table 1.2 Clinical comparison between bacterial and noninfectious keratitis. (Reprinted from [58], with permission from Elsevier.)

Feature	Bacterial keratitis	Noninfectious keratitis
Onset	Usually acute	Subacute or acute
Predisposing factors	Various: trauma, contact lens wear, prior ocular surface disease, and surgery	Various, including toxic and allergic insults, contact lens wear, blepharoconjunctivitis, herpetic eye disease.
Symptoms	Moderate to severe, increasing pain and light sensitivity	Variable, usually initially mild discomfort or foreign body sensation
Eyelids	Lid edema	Pseudoptosis possible
Conjunctiva	Marked hyperemia with episcleral injection and mucopurulent discharge	Mild hyperemia with mucoid or watery discharge
Corneal epithelium	Usually ulcerated; single larger lesions more common	Usually intact, possibly with punctate staining; can be multiple or arcuate lesions
Corneal stroma	White-yellow suppurative infiltrate with blurred margins and surrounding inflammatory cells and edema, > 1.5 mm, increasing over 24 to 36 hours	White-gray superficial infiltrates usually <1–1.5 mm (tend to remain small)
Corneal endothelium	Pseudoguttata with occasional inflammatory plaque or ring under stromal infiltrate	Minimal changes
Anterior chamber	Variable: cells/flare/hypopyon common	Mild; cells and flare, hypopyon uncommon

Summary for the Clinician

- Microbial keratitis is distinguished from noninfectious kerato-conjunctivitis by its increased severity of symptoms (pain and photophobia) and signs of corneal epithelial defects with associated inflammation (corneal infiltrates, conjunctival injection, and both anterior chamber cell/flare/hypopyon and lid swelling).

1.2 Risk Factors

1.2.1 Extended Wear

Patients can use contact lenses for wear solely during their normal daily activities ("open" eye) or also for use over one or several sleep cycles (extended wear or "closed" eye conditions). "Continuous" wear, alternatively, has been defined as contact lens wear uninterrupted by any intentional occasional lens removal.

The extended and continuous wear of hydrogel contact lenses, in particular, has been shown in several studies to increase the risk of MK [23, 40, 57]. MK has been shown to have an incidence of about 20 per 10,000 people using hydrogel contact lenses for extended wear and about 4 per 10,000 people using hydrogel contact lenses for daily wear per year [9, 48, 52]. Slightly higher rates were recently reported as well [23]. Daily wear of rigid gas permeable (GP) contact lenses is associated with a much reduced risk of MK [9, 36, 44]. The rate of MK with either high Dk silicone hydrogel or GP contact lenses used for extended wear is still in question, but is expected to be less than that found with hydrogel lenses – although it may remain higher than that encountered with daily wear of the same lenses [43, 45].

1.2.2 Contact Lens Care

It seems intuitive that poor contact lens care and hygiene might lead to increased microbial contamination of contact lenses, solutions, and cases. It also seems intuitive that an increased load of micro-organisms in the local environment, available for transfer from the environment to the eye during contact lens "cleaning" and handling, might increase the risks of MK. This particular paradigm of infection may indeed be supported somewhat by data in the case of acanthamoebic infection [16], but – while theoretically attractive – may not be totally supported in the case of bacterial MK [15, 40]. Nonetheless, most clinicians believe that, in general, both extended wear and poor contact lens care increase the risk of MK.

1.2.3 Role of Hypoxia

All rigid contact lenses were made of nonoxygen-permeable polymethyl methacrylate in the mid-1970s, and early hydrogel lenses all had modest oxygen transmissibilities (known as Dk/t). Hypoxia was a very common complication of contact lens use [21, 22, 28, 53].

It is now clear that maintaining oxygen tension in the tear layer (over the metabolizing anterior corneal surface) of about 100 mmHg will preclude physiological hypoxia, although various studies have placed this value between about 20 and 125 mmHg [7, 22, 49].

Most of the modern modest Dk GP and hydrogel contact lenses now available, and particularly those very high Dk silicone hydrogel and GP manufactured from materials with oxygen transmissibility of about 100 Fatt Dk units or greater, generally do not cause clinically observable corneal hypoxia under daily wear conditions [6, 7, 21]. Lenses made from these very high Dk GP and silicone hydrogel materials also appear to provide adequate corneal oxygenation when used on an extended wear basis, even though the precise level of contact lens oxygen permeability necessary to preclude hypoxia under such conditions has yet to be established [7].

When there is clear clinical evidence of hypoxic corneal changes (e.g., epithelial or stromal edema [28], corneal pannus greater than approximately 2 mm unrelated to 3/9 stain [8]), conjunctival and limbal hyperemia (e.g., injection) [47], myopic "creep" [17], or suspected "corneal exhaustion syndrome" [56], the clinician should adjust the contact lens wear schedule or change the contact lens material or design to enhance the availability of oxygen to the anterior corneal surface. Because of all these complications as well as the suspicion that hypoxia increases the risk of MK, increasing contact lens Dk/t is believed to be advantageous.

1.2.4 Role of Immunology

Immunology has been defined as the collection of integrated systems by which an organism defends itself from the assault of micro-organisms. There are both active and passive defenses, including leukocytes, antibodies, skin, and tears. There is a balance at work in that any infection, for example MK, only occurs when the pathogenicity of the microbe overwhelms the immunological defenses of the host.

A major question is whether addressing hypoxia alone is sufficient to reduce the incidence and prevalence of MK during contact lens extended wear to the rate found with daily wear. Several potential paths by which contact lens-driven hypoxia may suppress the immunological defenses of the anterior eye have been proposed. Contact lens wear and hypoxia may cause epithelial defects directly or indirectly (secondary to purely mechanical problems, e.g., abrasions, microtrauma, decreased mitosis and/or adhesion) [4, 20, 35], and any break in the integrity of the ocular surface is known to enhance bacterial infection. Another, more recent, hypothesis is that hypoxia causes changes in the corneal epithelial cell membrane, increasing the potential for bacterial binding [50].

Others believe that there are changes in the closed-eye state, particularly in the constituents of the tears [51], and/or in the ability of the corneal epithelium to resist bacterial invasion [18], beyond hypoxia alone, that makes closed-eye contact lens wear more likely to interrupt the normal immunological defenses of the anterior eye than open-eye contact lens wear. Tears usually

contain multiple antibacterial factors, including lysozyme, lactoferrin, lipocalin, vitronectin, betalysin, phospholipase A2, complement, immunoglobulins, mucins (which may entrap microorganisms for mechanical removal) [24, 51, 59] and occasional leukocytes, all potential targets for disruption. Both local and systemic disease (like Sjögren's syndrome and diabetes) and local or systemic immunosuppression (topical steroid use or HIV infection) are known to disturb one or more aspects of the protective nature of normal tears and/or the ocular surface to increase the risk of corneal infection. Closed-eye contact lens wear, with or without hypoxia, may act similarly. Investigators are actively studying the basic interactions between host and bacteria [11, 18], hoping to unravel the mechanism(s) that allow bacterial invasion of corneal epithelial cells with the goal of discovering ways in which to interrupt these processes.

This is a rapidly evolving research area directed toward enhancing safe contact lens daily and extended wear by assisting the normal immunological defenses of the anterior eye and/or by decreasing the ability of the micro-organisms to attack ocular tissues.

1.2.5 Role of Orthokeratology

Orthokeratology (OK) is the planned use of rigid contact lenses to deliberately modify the anterior corneal surface to neutralize refractive error. OK has been practiced for about half a century, and while efficacy has been questioned by some clinicians, safety has always appeared acceptable.

Recent innovations in rigid GP contact lens manufacture has led to the development of so-called "reverse geometry" contact lenses (with secondary curves steeper rather than flatter than the lens base curve) and the use of these lenses has clearly demonstrated increased efficacy in the OK treatment protocol. At the same time, however, some advocates of this procedure have suggested that OK lenses should be used during sleep (extended wear) as so-called "retainer" lenses and removed during open-eye experience.

This change in lens wear paradigm has unfortunately been accompanied by a number of case reports of subsequent MK in patients treated with unknown OK rigid lenses outside North America and Europe, initially dismissed as "unusual." Recently, reports of MK with OK using known GP lenses of modern designs inside the USA [31, 34] have reached the literature. Is this possible increase in risk more associated just with increased numbers of wearers due to increased popularity, or increased epithelial damage by mechanical pressure on the corneal apex due to OK treatment – or perhaps just closed-eye use as discussed above rather than any specific mechanical or lens design feature of OK? Evolving research will undoubtedly address these questions.

1.3 Microbes

1.3.1 Bacterial Infections

Bacterial corneal infections associated with contact lens (particularly extended) wear are usually attributable to Gram-negative *Pseudomonas aeruginosa*, and less commonly to both Gram-positive *Staphylococcus aureus* and *Staphylococcus epidermidis* [40, 57]. Other bacteria, both Gram-positive and Gram-negative (such as *Proteus*, *Serratia*, *Bacillus* sp., etc.), are also occasionally cultured from such lesions. For contrast, noncontact lens-associated corneal infections are usually more commonly Gram-positive (*Staphylococcus aureus* or *Streptococcus pneumonia*), Gram-negative *Moraxella* sp., or viral (Herpes). Climate and other environmental factors clearly play a role in the epidemiology of noncontact lens-related corneal infection as well, with more fungal keratitis reported from both the south-eastern United States as well as following direct (e.g., traumatic) exposure to plant matter.

Contact lens-related bacterial corneal infection has been primarily associated with wearing rigid or hydrogel contact lenses of limited oxygen transmissibility through one or more sleep cycles (extended or continuous wear) [9, 12, 29, 40, 48, 52, 57, 61]. Some have suggested that hypoxia alone is necessary and sufficient to account for all or most bacterial corneal infections that occur during contact lens wear, but this has not been proven.

Gram-negative bacterial infections tend to be more aggressive, leading to stromal necrosis with substantial discharge (Fig. 1.1), and Gram-posi-

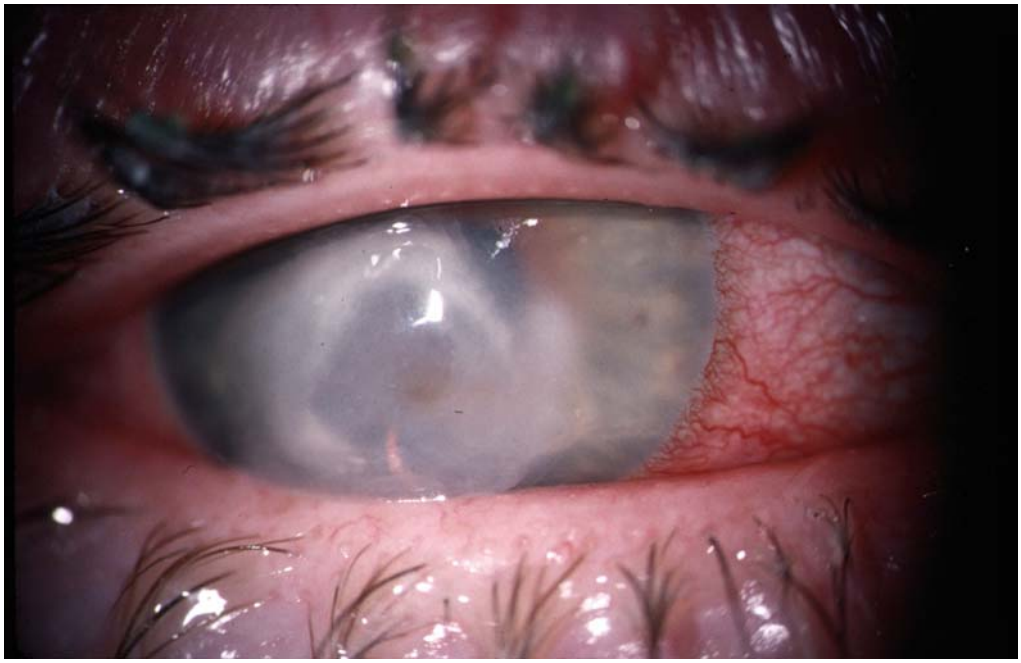

Fig. 1.1 Pseudomonas keratitis following contact lens wear: note both mucopurulent discharge and corneal ring abscess

tive bacterial lesions tend to be less aggressive leading to less discharge and stromal melting, but history and clinical appearance alone may be misleading. Annular corneal infiltrates are seen not only late in the course of acanthamoebic keratitis and early in severe pseudomonas-related corneal infections, but also in the form of an immune ring in herpetic and fungal corneal disease, and sterile anesthetic abuse as well. Results of smears and cultures, and clinical course, are often needed to develop a specific microbiologic diagnosis and hence an appropriate treatment protocol.

Poor compliance with contact lens care procedures leading to enhanced microbiological contamination of lens care solutions, cases, etc., also appears to be a major risk factor for microbial infection, possibly bacterial, but especially due to *Acanthamoeba* [16, 40].

1.3.2 Protozoal Infections

The clinician should always consider the possibility of *Acanthamoeba* species infections in any contact lens-related MK, especially in cases of chronic disease with initially negative culture results that fail to respond to antibiotic therapy. Clinical suspicion should be increased when the patient reports extreme ocular pain and/or a history of exposing his or her contact lenses to nonsterile water, or when an unusual dendritic epitheliopathy (reminiscent of herpetic epithelial disease; Fig. 1.2) or peripheral corneal radial neuropathy (Fig. 1.3) is observed [27, 41, 42, 54].

Acanthamoeba infections can be particularly challenging to confirm by laboratory investigations. Special culture techniques are available, such as culturing on nonnutrient agar coated with an *E-coli* overlay, but corneal biopsy is often necessary. Amoeba cell walls stained with calcofluor white will be seen when examined with fluorescent microscopy. Confocal microscopy can be useful for the diagnosis of corneal infections with *Acanthamoeba*; unfortunately, the limited availability of such instruments in the USA makes cultures and biopsies the more commonly employed diagnostic tests.

Misdiagnosis and medical failures in the treatment of *Acanthamoeba* infections are common.

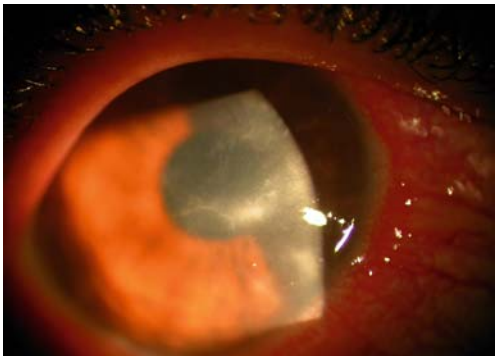

Fig. 1.2 Acanthamoeba keratitis: dendritiform lesion that often leads to misdiagnosis

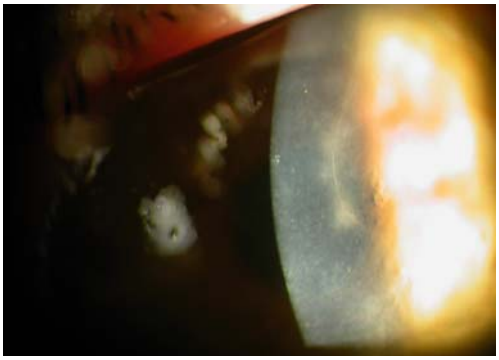

Fig. 1.3 Acanthamoeba keratitis: radial perineuritis

1.3.3 Fungal Infections

Fungal corneal infections (keratomycosis) have been extremely rare among cosmetic contact lens wearers, with the exception of an unusual worldwide collection of Fusarium keratitis possibly related to one brand of soft lens solution in 2006 (under investigation at the time of writing). Most previous cases reported in the literature have involved the use of contact lenses for treatment of aphakia, bandage use of contact lenses, or concomitant chronic treatment with topical steroids in patients suffering from concurrent ocular disease (e.g., neurotrophic epithelial defects, diabetes, trauma) [26, 60]. Fungal corneal infections are often distinguished as "fluffy"-appearing infiltrates with feathered borders, associated with separate satellite lesions. It is important to note that atypical mycobacterium and *Acanthamoeba* infections often mimic fungal corneal ulcers and vice versa.

1.3.4 Viral Infections

Adenoviral and herpetic viral corneal infections can occur during contact lens wear. No causative association has been uncovered for such viral infections. Round subepithelial corneal infiltrates and follicular conjunctivitis can occur with both infections, and discharge tends to be more watery than mucopurulent as in bacterial infections. Both epithelial dendrites and decreased corneal sensitivity are common signs of herpetic infection in particular. Contact lens wear should be discontinued during viral infections unless the contact lens is being used in a treatment protocol. Adenovirus infection is usually successfully managed by supportive therapy such as tear supplements and topical decongestants. Effective topical (Viroptic) and oral antiviral agents are available for the treatment of herpetic eye disease. The clinician who observes apparent herpetic keratitis in association with the use of contact lenses, however, should always consider the possibility of an *Acanthamoeba* infection masquerading as herpes.

It is prudent to consider discarding contact lenses, especially inexpensive disposable soft lenses of any type, that have been worn during an active viral infection and then dispense new contact lenses once the infection has resolved. More expensive customized (primarily rigid GP but also occasionally soft) lenses should be disinfected using the appropriate techniques prior to advising the patient that contact lens wear can be resumed.

Although both the human immunodeficiency virus (HIV) and the prions that cause Creutzfeldt-Jakob disease have been isolated from human ocular tissues (e.g., cornea, conjunctiva, and tears), no reports of disease transmission have been reported from ocular contact. Nonetheless, it is prudent to minimize risks to both patients and clinicians by appropriate disinfection of diagnostic instrumentation, and particularly disinfection (or discarding) of diagnostic contact lenses (whether disease is known, suspected, or unsuspected).