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Corticosteroid in the Treatment of *Neisseria gonorrhoeae* Keratitis

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Abstract

Aim: To describe the effective use of corticosteroid in the management of keratitis caused by *Neisseria gonorrhoeae*.

Methods: Retrospective review of the case of a nineteen year old gentleman who presented with a purulent ocular discharge and in whom peripheral corneal melt occurred.

Results: *Neisseria gonorrhoeae* was cultured from conjunctival swab. Systemic and topical antibiotics were given. The peripheral corneal melt was unchanged until systemic corticosteroid was prescribed following which the corneal melt resolved promptly.

Conclusions: *Neisseria gonorrhoeae* keratitis may behave like a peripheral ulcerative keratitis. In such cases topical and/or systemic corticosteroid may be therapeutic despite their usual contraindication in cases of acute infective keratitis.

Keywords

Neisseria gonorrhoea, Keratitis, Corticosteroid, Peripheral ulcerative keratitis

Description

The left eye of a nineteen year old gentleman became red and productive of a purulent discharge. A conjunctival swab was reserved. Presumed to have bacterial conjunctivitis he was treated with topical chloramphenicol and oral flucloxacillin 500mg both four times daily for one week. *Neisseria gonorrhoeae* was however cultured. He was referred to our service for further management.

His visual acuity at this time was 6/6 and 6/24 from his right and left eyes respectively. The right eye looked normal. The conjunctiva of the left eye was injected. It was however clear of discharge. A deep gutter extended from the 10 o'clock to the 3 o'clock position in the superior cornea. A smaller para-central area of corneal thinning was also apparent (Figure 1). Treatment with topical exocin and ceftazidime every hour and intravenous ceftriaxone 2g and oral ciprofloxacin 750mg twice daily was commenced. The appearance of this gentleman's left eye remained unchanged however after five days. Prednisolone 40 mg orally daily was commenced and reduced by 10mg daily every week thereafter. The peripheral corneal melt resolved within two weeks.

Comment

Ocular infection with *Neisseria gonorrhoeae* is relatively rare. We may be less suspicious of it as a result. Indeed, all four cases of corneal perforation secondary to gonococcal infection described in one case series were originally misdiagnosed as epidemic keratoconjunctivitis [1].

Cases occur in sexually active individuals. Infection results from contact with infected urine and/or genital secretions. Such contact may be direct or by indirect 'autoinoculation' wherein infective genital secretions are transferred from their source to the eye's surface by hand-eye contact.

Genitourinary symptoms of gonococcal infection, if they occur (up to 80% of males and 20% females affected in this regard are asymptomatic) may precede ocular symptoms by one to several weeks. Patients are howev er unlikely to make a connection between their sexual behaviour, genital and ocular symptoms. Consequently, retrieval of the pertinent patient sexual and genitourinary system history may be invaluable in suggesting a diagnosis of gonococcal keratoconjunctivitis.

Neisseria gonorrhoeae causes a conjunctivitis which typically presents hyper-acutely with usually bilateral but occasionally, as

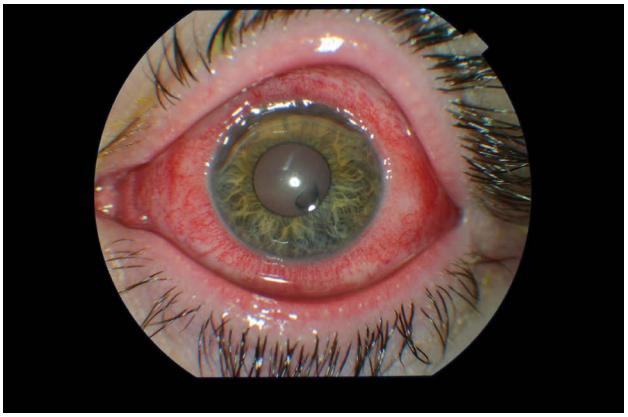

Figure 1: A deep gutter extended from the 10 o'clock to the 3 o'clock position in the superior portion of this gentleman's cornea. A smaller para-central area of corneal thinning was also present.

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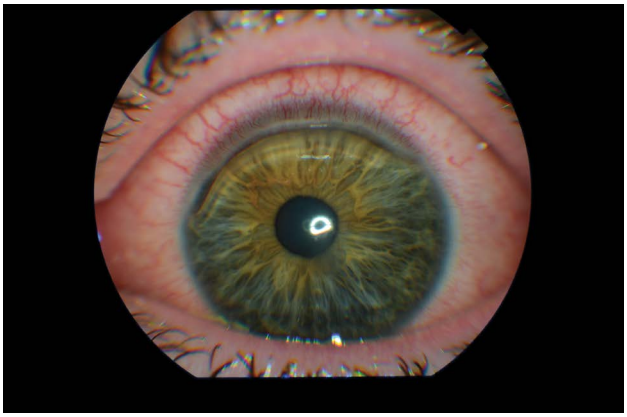

Figure 2: In this gentleman's case the clinical appearance was very similar to an immune mediated peripheral corneal melt. Note the relative lack of stromal disease elsewhere in the cornea beyond the area of ulceration superiorly.

here, unilateral, extreme, conjunctival inflammation and an intensely purulent ocular discharge. The presence of an intensely purulent ocular discharge in a sexually active individual should *always* raise the suspicion of gonococcal eye infection. Tender eyelid oedema can also occur. Ocular motility may be restricted. Indeed unilateral gonococcal conjunctivitis initially thought to be orbital cellulitis has been described [2].

Common corneal findings include a marginal corneal ulcer or corneal melt, sub-epithelial and/or stromal infiltrates and a discrete oedema of the entire cornea [3]. The first of these may be most common and is thought to be due to the formation of pus filled troughs beneath the eyelids.

Currently, the CDC recommends combination therapy with a single intramuscular dose of ceftriaxone 250mg plus either a single dose of azithromycin 1g orally or doxycycline 100mg orally twice daily for 7 days for the treatment of uncomplicated urogenital, anorectal, and pharyngeal gonorrhea. In instances where ceftriaxone is not available, cefixime 400mg orally, plus either azithromycin 1g orally or doxycycline 100mg orally twice daily for 7 days should be used. For patients allergic to the cephalosporins, the CDC recommends use of a single 2g dose of azithromycin orally. In both of these circumstances, a test of cure for these patients should be carried out one week after treatment [4].

In that particular case of gonococcal keratitis described here the clinical appearance was very similar to an immune mediated peripheral corneal melt. Indeed, we were particularly impressed by the relative lack of stromal disease elsewhere in the cornea beyond the area of ulceration (Figure 2).

The influence of inflammatory cells and their enzymatic products in stromal destruction in auto-immune peripheral corneal melt is already well established [5]. The polymorphonuclear (PMN) cell is thought to be most important. Since the migration of PMN cells is from the limbal vasculature into the corneal stroma most immune mediated tissue damage is limbal. *Neisseria gonorrhoea* was not cultured from the cornea of eyes with severe gonococcal keratitis which were enucleated as might be expected were corneal penetration secondary to active bacterial replication [6]. Similarly, in our case, though an aggressive, extensive peripheral corneal melt persisted following the administration of antibiotics, we were unable to demonstrate the presence of organisms in specimens taken at conjunctival swab and corneal scrape. The relative role of the enzymatic products of the PMN cell in the pathogenesis of gonococcal keratitis is unknown but given the appearance of the cornea in the case we describe here we postulate that it is relatively great. Indeed the peripheral corneal ulceration resolved only when systemic corticosteroids were administered.

The successful use of topical corticosteroid in the treatment of a case of *Neisseria gonorrhoeae* keratitis which was associated with diffuse lamellar keratitis three years after laser in situ keratomileusis has already been described [7].

Rapid progression of an ulcerative keratitis to corneal perforation can reportedly occur within 24 h of infection with *Neisseria gonorrhoeae* [8]. Further, the outcome of this infection depends upon its severity when appropriate therapy is commenced [9]. 'Appropriate' therapy in some cases may thus include topical and/or systemic corticosteroid despite their usual contraindication in cases of acute infective keratitis.

Conclusion

We hope this report will serve as a reminder of both the occurrence and indeed the potential severity of gonococcal eye disease. A sexual and genitourinary system history should be obtained from all those with a purulent ocular discharge. Severe gonococcal keratitis may be unilateral. This may behave like a peripheral corneal melt. Consequently topical and/or systemic steroid may be therapeutic.

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