Conjunctivitis

8Ca Binocular Papillary Conjunctivitis
8Cb Monocular Follicular Conjunctivitis

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INTRODUCTION

A personal observation is that many optometrists, while keen to be involved in community ‘red eye’ shared care schemes, articulate fear in the direct prescribing of drugs. As a profession, our primary hurdle is not at the point of prescribing, but is developing diagnostic confidence; without this the prescribing of therapeutics is extremely risky.

While conjunctivitis is usually self-limiting, aetiologies include infective, toxic and allergic mechanisms (Silverman 2008) and misdiagnosis can lead to sight threatening sequelae. Since recourse to lab tests is not usual (Silverman 2008); the diagnosis must be considered a working hypothesis.

A thorough case history, symptomatology and chronicity (Scott 2008, Ostler 1993) are significant aids to diagnosis. However, this paper proposes that to make a safe working diagnosis, emphasis must be placed on whether the inflammatory response is follicular or papillary and whether one or both eyes are involved.

Follicular/Papillary Reactions

Conjunctival Associated Lymphoid Tissue (CALT) (Knop and Knop 2000, Knop and Knop 2007) is the ocular equivalent of Mucosal Associated Lymphoid Tissue (MALT). Distributed in anatomical regions of exposure, MALT expedites the specific immune response to invading pathogens. The lymphoid associated follicles contain germinal centres where plasma and memory B cells are generated (Wood 2001). A follicular response therefore indicates a specific immune response to pathogen and is highly indicative of infection. If the ocular involvement is associated with a systemic infection, such as an upper respiratory tract infection, then lymph nodes, particularly the pre-auricular lymph nodes, may also be involved.

It is interesting then, that bacterial conjunctivitis does not typically induce a follicular response (Onofrey, Skorin and Holdeman 1998, Lowery 2006), while allergic responses to topical medications do (Thygeson 1957, Majmudar 2008).

Thygeson (1957) suggests that a follicle stimulating substances must be small enough to pass through the epithelium; these would include intracellular organisms such as viruses and chlamydia, but also chemicals. Chemicals, while too small to induce an immune response can act as haptens (Wood 2001) inducing a Type IV cell-mediated hypersensitivity (Majmudar 2008).
The reactions may also be a specific adaptation involving ocular immune privilege. Immune privilege (Catania 1995, Knop and Knop 2007) down regulates CD8 Cytotoxic T Cell, and Delayed Type Hypersensitivity reactions because of the potential structural damage of these mechanisms (Wood 2001). Pathogens must necessarily be neutralised with less damaging anti-body responses. Once intracellular more destructive mechanisms are required; the chronic nature of chlamydia could reflect immune privilege down regulation of CD8 T Cell activity.

Papillae develop only where the conjunctiva is firmly attached to underlying tissue, tarsus and limbus; looser tissue will demonstrate less structured chemosis (Ostler 1993). Primarily considered a Type I allergic reaction it also manifests with mechanical irritation from ocular foreign bodies (Weissman 2007) and reflects a non-specific immediate inflammatory response. Large bacteria tend to produce a papillary reaction suggesting they are contained by the innate immune response, reflected by the presence of neutrophils in the primary cellular response (Marlin 2007). Only in more protracted or significant bacterial infections does lymphatic involvement become evident (Marlin 2007).

**Monocularity/Binocularity**

Silverman (2008) indicates that bilateral disease is typically infectious or allergic, while unilateral involvement suggests toxic, chemical, mechanical or lacrimal origin. Certainly, Type I allergies will be bilateral, however, infections from chlamydial (Bashour 2007), molluscum (Scott 2007), simplex and zoster (Scott 2008) are often unilateral. Conversely, while Scott (2007) suggests adenovirus may present monocularly, its’ highly contagious nature make rapid progression to both eyes expected (Scott 2008).

Case 8a was self-evident. The binocularity, papillae, symptoms, chronicity and associated atopy and keratoconus indicated a chronic allergic conjunctivitis. The need to stop ocular rubbing as much as relieve the symptoms, necessitated safe, long term, medication. A mast cell stabiliser was prescribed.

However, especially when the condition is unilateral, other aetiologies must be pro-actively considered; uveitis, keratitis, foreign bodies, lacerations, episcleritis, scleritis. While symptoms and case history should give strong clues, it is easy to overlook a possibility, particularly if
more obvious co-existing observations dominate the clinical picture; particularly relevant with contact lens wearers.

A systematic policy must include examination of the anterior chamber, all levels of the cornea and with stains for the epithelium, bulbar and palpebral conjunctiva, lids and examination of the fellow eye for less obvious signs of binocular involvement.

A monocular, follicular conjunctivitis, as in Case 8b, a young lady 16 years old, must elicit concern.

The complete lack of signs in the fellow eye, and confirmation that co-existing pathologies were not present made chlamydia or a toxic reaction the most likely possibilities. The former requires antibiotics and more in-depth medical management, the latter anti-inflammatories. These dichotomously opposed treatment modalities necessitate diagnostic confidence and an exhaustive case history. Scott (2008) does stress that diagnosis of toxic conjunctivitis must be one of exclusion. No history of monocular use of ocular drops or inadvertent contact with noxious substances could be elicited and the problem was not acute in nature. The preliminary diagnosis of chlamydia was made, a conjunctival scrape organised, azithromycin prescribed and review set post lab results.

No follow-up for this patient was possible. At the initial consultation no comment on the possibilities was made. This seems prudent due to the delicate nature of the problem. At review, with a definitive lab result, specific recommendations, ensuring patient confidentiality, could be made.

Diagnosis of Adult Inclusion Conjunctivitis would necessitate referral for STD consultation; to ensure general health and prevent re-inoculation.

Access to labs and the confidence in conjunctival scrapes would put the management of Adult Inclusion Conjunctivitis within the realms of community care. Referral to ophthalmology within Northumberland Central currently is simply to ensure accurate lab results prior to diagnosis and patient education.
REFERENCES


