possible endogenous sources. He was a farmer by profession, but there was no history of injury with vegetative matter. Although we found an endogenous embolus in the iris vasculature, we were not able to locate the primary source.

Our case highlights the need to consider fungal etiology as a differential diagnosis in an iris granuloma. In our case excision biopsy of the iris granuloma and an AC tap played a vital role in clinching the diagnosis. Institution of steroids without antifungal treatment could be disastrous in case of a misdiagnosis.

Our case is the first report of *Aspergillus* iris granuloma in an immunocompetent individual. A high degree of clinical suspicion, combined with microbiological and histopathological evaluation helped to arrive at an appropriate diagnosis and successful management.

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AB Majji¹, MK Aasuri², GK Vemuganti³ and S Sharma⁴

¹Smt Kanuri Santamma Retina Vitreous Centre
LV Prasad Eye Institute
LV Prasad Marg

Banjara Hills
Hyderabad-500 034, India

²Cornea and Anterior Segment Services
LV Prasad Eye Institute
LV Prasad Marg
Banjara Hills
Hyderabad-500 034, India

³Ophthalmic Pathology Service
LV Prasad Eye Institute
LV Prasad Marg
Banjara Hills
Hyderabad-500 034, India

⁴Jhaveri Microbiology Centre
LV Prasad Eye Institute
LV Prasad Marg
Banjara Hills
Hyderabad-500 034, India

Correspondence:

AB Majji

Tel: 91 40 3608262

Fax: 91 40 3548271

E-mail: ajit@lvpeye.stph.net

Sir,

E-Cadherin distribution in normal and dysplastic conjunctival epithelium

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Conjunctival and other mucosal associated lymphoid tissues share functional and morphological similarities including the presence of intraepithelial lymphocytes (IELs) in the basal epithelial layer.¹ The conjunctiva can induce immune tolerance to encountered antigen, wherein repeated challenge by an antigen at a mucosal surface causes suppression of the local immune response at the same and possibly other tissues, despite systemic immunogenicity to the same antigen.² It is believed that IELs of mucosal epithelia have a suppressor role in the induction of mucosal tolerance.^{2,3} IELs express the integrin $\alpha E\beta 7$ (human mucosal lymphocyte antigen, HML-1), the ligand for which is E-cadherin, an epithelial intercellular adhesion molecule.⁴ Normally E-cadherin is responsible for the induction and maintenance of cell polarity and differentiation in both foetal and adult epithelial

tissues.⁵ Homotypic E-cadherin/E-cadherin bonds mediate intercellular calcium-dependant binding and contribute to the integrity of all epithelial tissue.⁶ Heterotypic E-cadherin/ α E β 7 binding is thought to anchor IELs and retain them within the epithelium.⁴ In previous studies we have demonstrated that conjunctival IELs are distributed along the basal epithelial cells in normal conjunctiva and throughout the entire epithelial thickness in conjunctival dysplasia.^{1,7} We wished to examine whether this correlated with the distribution of E-cadherin.

Report

Paraffin-embedded sections of eight normal conjunctivas and seven specimens of conjunctival intraepithelial neoplasia (CIN) were immunostained for E-cadherin by the sensitive avidin/biotin indirect immunoperoxidase technique, using the monoclonal anti-E-cadherin antibody 5H9 (Europath, UK). The slides were examined under light microscopy and brown positive staining for E-cadherin was noted. On histological grading, five of the CIN samples were severely dysplastic, one was of moderate grade and one was mild grade. The research was performed according to the tenets of the Declaration of Helsinki.

We found that all the normal conjunctival samples stained positively for E-cadherin. The distribution was confined to the conjunctival epithelium and staining was seen through all epithelial layers (Figure 1a). There was intense cell membrane staining and mild cytoplasmic staining of the normal epithelial cells. E-cadherin staining of the cell membrane in the superficial squamous layer was more subdued, when compared to the rest of the epithelium. E-cadherin expression in the dysplastic samples was evenly distributed through the entire epithelium (Figure 1b). There was no relationship between the grade of dysplasia and E-cadherin distribution in these samples. Positive staining for E-cadherin in the dysplastic epithelium was more intense in the cytoplasm than in the cell membrane.

Comment

We have demonstrated that E-cadherin is present in all regions of the normal and dysplastic human conjunctival epithelium. The reduced intensity of staining in the superficial layers of the normal conjunctival epithelium is possibly a prelude to desquamation. In normal conjunctiva, IELs are distributed almost exclusively in the basal layer. It is possible that IELs are bound to the basal epithelial cells by the heterotypic E-cadherin/ α E β 7 bonds as they

Figure 1 (a) Photomicrograph (×100, immunoperoxidase) of normal bulbar conjunctiva demonstrating E-cadherin staining of the epithelium. (b) Photomicrograph (×400, immunoperoxidase) demonstrating E-cadherin distribution in biopsy specimen of conjunctival squamous dysplasia.

migrate into the epithelial compartment through the pores in the basement membrane. As only a minimal number of lamina propria CD8+ T cells express α E β 7, it is likely that α E β 7 is induced in the microenvironment of the epithelial compartment.⁸ The α E β 7 mediated binding of IELs to E-cadherin on the basal epithelial cells may prevent them from migrating any further in the normal conjunctival epithelium.

In dysplastic conjunctival tissue, E-cadherin was evenly distributed throughout the epithelium in all samples. This appears to correlate with the distribution of IELs which are present in all layers of the dysplastic epithelium.⁷ This may be partly due to the relative lack of basement membrane in dysplastic epithelium and also due to the entry of lymphocytes into the epithelium by routes besides basement membrane pores, for example via blood vessels.

It has also been suggested that dividing epithelial cells may influence the distribution of IELs,⁷ which would explain the IEL distribution along the basal (mitotic) layer of normal, and all layers of dysplastic epithelium. Dividing cells produce growth factors like

TGF- β , which are known to induce expression of α E β 7 on IELs.⁹ This may be another reason why IELs are present throughout all the layers of the dysplastic epithelium but only along the basal cells of normal conjunctival epithelium.

We did not notice any definite relationship between the degree of dysplasia and E-cadherin expression. These findings contrast to observations made with cervical intraepithelial neoplasia where reduced E-cadherin expression related to an increasing loss of cell differentiation.¹⁰ The observations made in this study with regard to E-cadherin suggest that the distribution of IELs in normal conjunctiva and CIN cannot be explained by the distribution of E-cadherin alone.

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RAH Scott¹, HS Dua¹, A Joseph¹, R Haynes¹, D Snead² and NM Hand²

¹Department of Ophthalmology
Queen's Medical Centre
Nottingham, UK

²Department of Pathology
Queen's Medical Centre
Nottingham, UK

Correspondence: HS Dua
Tel: +44 (0)115 9249924 Ext. 42530
Fax: +44 (0)115 9709963
E-mail: Harminder.Dua@nottingham.ac.uk

Sir,

Orbital cellulitis following sub-Tenon's anaesthesia
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The injection of local anaesthetic agents into the episcleral, or sub-Tenon's, space is widely used for cataract surgery. Since its introduction 9 years ago, no serious adverse events have been reported.^{1,2} We have recently encountered a case of orbital cellulitis following sub-Tenon's anaesthesia and phacoemulsification with lens implantation. To our knowledge, this is the first report of an infectious complication of sub-Tenon's anaesthesia.

A 63-year-old female patient underwent phacoemulsification with posterior chamber lens implant under sub-Tenon's local anaesthesia. Preoperatively, Benoxinate drops were instilled, and a single injection of 2 ml Lignocaine 2%, 2 ml Bupivacaine 0.75% and 500 IU Hyaluronidase was administered into the inferonasal sub-Tenon's space. The eye was then prepared with an aqueous solution of povidone-iodine 10% (Betadine^R), and a routine phacoemulsification procedure was performed. At the end of the operation, a subconjunctival injection of Gentamycin 20 mg and Betamethasone 2 mg was given.

On the first postoperative day, the patient complained of ocular pain. There were no unusual findings except moderate postoperative anterior uveitis. The patient was commenced on G Maxitrol^R