

OP-4

EARLY INTERVENTION MAY REDUCE ULTRASTRUCTURAL CHANGES IN DESCMET'S MEMBRANE IN FUCHS ENDOTHELIAL CORNEAL DYSTROPHY

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Background and Purpose Studies in patients with Fuchs endothelial dystrophy (FED) have noted associations between localised endothelial loss, increases in corneal densitometry and deposition of a collagen-rich fibrillar layer. We defined a specific imaging marker on anterior segment OCT (AS-OCT) that arises in the presence of deep stromal folds, known as posterior stromal ripples (PSR). We have shown that pre-operative PSR affects visual recovery after DMEK and are more likely to be present in more advanced disease. Our aim was to determine if PSR reflects ultrastructural fibrotic changes in Descemet's membrane-endothelium (DME).

Methods DME complexes from patients with FED who had preoperative AS-OCT were identified and stained with hematoxylin-eosin, Periodic acid-Schiff as well as anti-CD34 and anti-alpha-smooth muscle actin (α -SMA) antibodies. Preoperative PSR were qualitatively graded and quantified using an algorithm trained to detect posterior corneal surface alterations.

Results 52 patients have been included and to date 9 DME (including negative controls) have been analysed. H&E and PAS showed alterations of the DME complexes in all samples; however, the presence of anti-CD34 and anti- α -SMA was significantly more frequently present in samples from eyes showing pre-operative PSR (both $p < 0.05$). Eyes with preoperative PSR had a lower final visual acuity compared to eyes without pre-PSR (mean [SD] 0.25 [0.1] vs. 0.05 [0.07] LogMAR).

Conclusions Preoperative PSR reflects ultrastructural fibrotic changes along Descemet's membrane and is associated with a lower final visual acuity. This suggests that earlier intervention in patients with FED prior to the development of PSR is indicated.

Oral Abstract Presentation – Problem Case Presentation

OP-5

ENDOART UPDATE

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Case A 64-year-old patient with a history of failed penetrating keratoplasties who underwent endoart artificial endothelial layer surgery will be presented, with a summary of endoart results and current indications.

Oral Abstract Presentation

OP-6

VACCINATION AGAINST SHINGLES AND PREVENTION OF LONG-TERM OCULAR MORBIDITY – A RETROSPECTIVE STUDY OF COST IMPLICATIONS TO THE NHS

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Introduction Shingles is caused by reactivation of latent Herpes varicella-zoster virus (VZV). Herpes zoster ophthalmicus (HZO) is defined as shingles involving the ophthalmic division of the trigeminal nerve. A national public sector shingles vaccination program was introduced in the UK in 2013 offering vaccination to those over 70 years. From 1st September 2023, the age for vaccination has been reduced to 65 years. Vaccination has been available to those aged 50y and over in the private health sector in the UK.

Methods We undertook a retrospective study determining the number of patients aged 50-69y who had attended our hospital with HZO from 1st April 2022 to 30th June 2024. We determined how many were affected with long term ocular sequelae of this infection and the subsequent costs to the NHS.

Results Our study shows that during this 27-month period, we had a total of 48 cases of HZO, with 9 patients (18.8%/1 in 5) going on to develop long-term ocular complications requiring multiple hospital attendances; of these, 44.4% of patients had long-term corneal associated pathology. The total cost of treatment to the NHS of these cases was £41,671.

Conclusions We concluded that for the cost of treatment of these 48 patients by hospital eye services, a total of 340 patients could have been vaccinated against shingles (seven times the number of affected patients). Although further study is required, it would seem advisable in terms of the cost benefit ratio, to reduce the age of vaccination against shingles of patients down to 50 years.

Oral Abstract Presentation – Problem Case Presentation

OP-7

MITOMYCIN INTRAVASCULAR CHEMOEMBOLISATION (MICE) – TREATMENT FOR CORNEAL NEOVASCULARISATION

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Background Corneal neovascularisation (NV) is a challenging condition that can often be refractory to standard treatments, leading to lipid deposition, causing opacification, scarring, irregular astigmatism and decreased visual acuity. The aetiology