CORNEAL MELTS
Biologic agent Infliximab proved effective when all else failed
by Roibeard O’hEineachain in Prague

Recurring corneal melts in patients with rheumatoid disease may respond best to aggressive therapy which can include biologic modalities such as infliximab, an inhibitor of tumour necrosis factor-alpha (TNFα), according to a case study presented by Dipak Parmar FRCOphth, FRANZCO, London, UK, at the Cornea Day Session at the 16th ESCRS Winter Meeting.

The patient was a 47-year-old Sri Lankan man who initially presented in October 2006 with a corneal perforation in his right eye with 80 per cent corneal thinning and 20 per cent corneal thinning in his left eye, Dr Parmar noted. His visual acuity was 6/19 in his right eye and 6/9.5 in his left eye. Both eyes had a peripheral inferonasal gutter at 3 to 6 o’clock.

The patient’s medical history included rheumatoid arthritis of five years’ duration which was systemically quiescent and managed under a rheumatologist at another hospital. His current regimen included prednisolone 4mg per day and methotrexate 7.5mg per week, together with calcium carbonate, alendronic acid and diclofenac.

Dr Parmar initially treated him with corneal glue repair and increased the prednisolone dosage to 60mg per day (plus ranitidine) and gradually increased his methotrexate dosage to 20mg per week over the next three months.

In February 2007 the patient had a repeat corneal perforation in his right eye. Dr Parmar again treated the eye with corneal glue repair together with multilayered amniotic membrane transplantation and Parasol punctual plugs (Odyssey) in both eyes. The patient also received a low dosage of methotrexate, 2.5mg per week, and his condition remained stable for a year at which point he was lost to follow-up.

Two years later he returned to the clinic with corneal perforations in both eyes (Figure 1). Dr Parmar performed bilateral simultaneous tectonic penetrating keratoplasties and increased his methotrexate dosage to 10mg per week and prednisolone to 60mg per day. However, just one month after the penetrating keratoplasties there was a repeat corneal perforation in the patient’s right eye. He underwent corneal glue repair on three further occasions and had his methotrexate dosage increased to 25mg per week. He also received topical cyclosporine 0.05 per cent (Restasis) twice a day in both eyes.

In July 2009 the patient underwent repeat penetrating keratoplasty with amniotic membrane graft overlay in his right eye. However, the graft was not successful and he required yet another penetrating keratoplasty within a week. In addition, the graft in the patient’s other eye developed paracentral thinning, which continued unabated despite the application of a multilayered amniotic membrane graft, high-dose methotrexate and corneal glue repair.

Unfortunately the patient had developed adverse reactions to azathioprine and cyclophosphamide, which meant that there were very few treatment options left. At this point infliximab was suggested as a treatment option to Dr Parmar by his rheumatologist colleague. Infliximab is an anti-TNFα chimaeric monoclonal antibody that prevents TNFα binding to cellular receptors and induces apoptosis in TNFα-expressing activated T-cells.

Immediately after undergoing a repeat tectonic graft in his left eye, the patient commenced intravenous therapy with infliximab administered every six weeks. At 30 months post infliximab both corneas were stabilised, with no evidence of disease progression (Figure 2). His best corrected visual acuity was 6/48 in the right eye and 6/36 in the left, limited by pre-existing corneal subepithelial haze.

Dr Parmar concluded: “I would definitely encourage those of you managing patients with corneal melts secondary to rheumatoid or other inflammatory conditions to consider these biological agents.”

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