Dexamethasone implants and neovascular glaucoma in central retinal vein occlusion

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Introduction

Dexamethasone intravitreal implants (Ozurdex) are a NICE-approved therapy for the treatment of macular oedema (MO) in patients with branch retinal vein occlusion (BRVO) and non-ischaemic central retinal vein occlusion (niCRVO), and have been reported to reduce the ischaemic complications of these conditions.1, 2, 3

Interestingly, 2 important studies, such as GENEVA and SCORE study, have shown controversial results regarding the effect of intravitreal steroids on the reduction of retinal neovascularisation.4, 5

We present a case series of five patients who developed neovascular glaucoma (NVG), after being treated with dexamethasone implants for clinically-diagnosed niCRVO.

The purpose of this study was to report that neovascularisation is still a clinical problem in patients with niCRVO treated with Ozurdex implants, indicating that close observation for the development of NVG is still required in these patients.

Methods

This was a retrospective case study of 25 patients with clinically-diagnosed niCRVO, treated with dexamethasone intravitreal implants and followed-up for 12 months.

The diagnosis of niCRVO was based on visual acuity (VA), clinical features and the absence of a relative afferent pupillary defect (RAPD). Patients were treated with dexamethasone implants if their VA was worse than 6/12 with central macular oedema.

NVG was diagnosed as neovascularisation of the angle (NVA) visible on gonioscopy and associated with raised intracocular pressure.

Results

5/25 patients developed NVG (20%), all within 8-12 weeks of their most recent dexamethasone implant. All five affected patients had initially presented with the clinical features of niCRVO and had visual acuities of 6/24 – 6/36 with no RAPD.

All had an improvement in visual acuity in response to treatment with dexamethasone intravitreal implant of 1-3 Snellen lines.

All patients were successfully managed with a combination of anti-hypertensive agents, intravitreal bevacizumab injections and panretinal argon laser photoagulation. No patients received further dexamethasone intravitreal implants.

Conclusions

All five of our patients developed NVG within 8-12 weeks of their most recent dexamethasone implant, at which time it would be expected to be exerting its full effect. Thus our case series, whilst small, suggests that the dexamethasone implant does not prevent the development of NVG.

In addition, three patients were diagnosed with NVG following an earlier diagnosis of steroid-induced ocular hypertension, suggesting that it may be easy to overlook early NVG, particularly in a situation where 15% of patients are expected to develop steroid-induced ocular hypertension.1

One of the assumptions that has supported the adoption of the dexamethasone implant in the UK is that treated patients may require less frequent follow-up than patients managed with ranibizumab or observation.2 On the contrary, our study suggests that frequent and careful observation is still required to guard against NVG, and that patients with ocular hypertension should be assessed carefully for the presence of subtle iris and angle neovascularisation.

References

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