Use of orbital floor steroids in the management of patients with uniocular non-necrotising scleritis

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Abstract

Most cases of non-necrotising scleritis can be successfully treated with non-steroidal anti-inflammatory drugs. If these are ineffective, then high-dose systemic corticosteroids, with all their attendant side-effects, are usually required. We have used orbital floor injections of depot steroid in the management of nine patients with non-necrotising scleritis in an attempt to avoid the use of systemic steroids, or to allow the dose of steroids to be reduced while maintaining disease control. A temporary reduction in inflammation was achieved in all cases, which allowed the use of systemic steroids to be avoided altogether in two patients and delayed in the others. Non-steroidal anti-inflammatory drugs and systemic corticosteroids remain the mainstay of treatment for non-necrotising scleritis, but orbital floor injections may be a useful adjunct in certain cases.

Scleritis is a potentially serious disorder, which can occur in both young and old patients. Although it can be associated with an underlying collagen disease, many patients are healthy. Pain is a predominant feature of active scleral inflammation, and is the usual presenting complaint.

Patients with anterior diffuse (Fig 1) or nodular, non-necrotising scleritis, and posterior scleritis, can often be treated with non-steroidal anti-inflammatory drugs (NSAIDs), such as flurbiprofen or indomethacin. Necrotising scleritis, however, always requires systemic steroid therapy, sometimes in combination with other systemic immunosuppressive drugs.¹ Topical steroid therapy can control episcleral inflammation but has no effect on scleral inflammation. Subconjunctival steroid injections are contraindicated because of the risk of scleral thinning and perforation.²

Some patients with non-necrotising disease do not respond to non-steroidal therapy and require systemic corticosteroids for disease control. As

with intraocular inflammation, high doses (prednisolone 40–80 mg per day) may be required. Many patients with uniocular posterior uveitis, however, are spared the problems associated with systemic steroid therapy by the use of orbital floor sub-Tenon's injection of depot steroid preparations when there is active inflammation.³ This route of treatment is particularly useful in patients with unilateral disease who are otherwise healthy. It is also useful in maintaining control of a unilateral exacerbation of disease while on a reducing regimen of systemic steroids.

In this study we aimed to see if patients with uniocular non-necrotising scleral inflammation in whom NSAIDs were ineffective, or could not be tolerated, could be treated with orbital floor injections of long-acting depot steroid preparations. We also treated three patients in whom the disease process had relapsed while on a reducing regimen of systemic steroids, in order to avoid returning to a high steroid dosage.

Patients and methods

Patients were recruited from the Scleritis Clinic at Moorfields Eye Hospital. The scleritis was classified as either anterior or posterior, and diffuse, nodular, or necrotising.¹ Investigations were performed to exclude an underlying systemic disease process.

All patients with necrotising disease were treated with high-dose systemic corticosteroids (prednisolone 60–80 mg per day), together with other immunosuppressive therapy if required. All patients with anterior diffuse or nodular, or posterior, scleritis were initially treated with flurbiprofen 100 mg three times a day, unless this previously could not be tolerated or had been found to be ineffective, in which case systemic steroids were started. Oral H₂ receptor antagonists were given concurrently to patients with a previous history of peptic ulceration or indigestion.

After one or two weeks of treatment the patients were reassessed. If the pain was controlled, and the degree of inflammation reduced, the NSAIDs were continued, and eventually tapered off when the disease process was quiescent. If there was no change, or if the inflammation had increased, then systemic steroids were started in cases of bilateral disease. Patients with unilateral disease, however, received, with informed consent, an orbital floor injection of 40 mg methylprednisolone acetate (Depomedrone). The steroid was mixed with 0.2 ml lignocaine 2%, and administered through the lower lid, at the junction of the inner two-thirds and outer one-third.⁴ The patients were examined again after one or two weeks, and

Figure 1 Anterior diffuse scleritis.

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Accepted for publication
14 November 1990

Table 1 Results

Patient no	Age/sex	Diagnosis	Indication for depot injection	Duration of effect	Delay achieved before use of high-dose steroids
1	66/F	Nodular	NSAID hypersensitivity	4 days	2 weeks
2	50/M	Nodular	NSAID intolerance	Symptom relief; signs unchanged	Steroids not required
3	24/F	Posterior	NSAID ineffective	1 week	2 weeks
4	65/M	Nodular	Relapse on flurbiprofen, prednisolone 10 mg	5 days	2 weeks
5	48/F	Posterior	Relapse when prednisolone <20 mg	Allowed steroid reduction for 5 weeks	6 weeks
6	63/F	Diffuse	Severe steroid side effects	2 weeks	5 months (3 injections)
7	37/F	Nodular	Relapse when prednisolone <20 mg	3 months	9 months
8	89/F	Diffuse	Relapse on prednisolone 5 mg	3 months	No increase required
9	64/F	Posterior	Relapse on prednisolone 5 mg	4 weeks	4 weeks (injection repeated for further relapse at 8 months to allow blood pressure control)

subsequently at intervals determined by the disease activity. Additional treatment, including repeat injections, was administered as required. If an injection was repeated within several weeks, shorter acting methylprednisolone succinate (Solumedrone) was used instead of Depomedrone to avoid build-up of high levels of depot steroid beneath the globe.

Results

All patients included in this trial had non-necrotising, anterior diffuse or nodular, or posterior scleritis, active in one eye only. No underlying disease process was detected in any patient.

The results are displayed in Table 1. Nine patients received an orbital floor steroid injection. They could be divided into two groups: group A (patients 1–3) in whom NSAIDs were ineffective or could not be tolerated, and in whom systemic steroids had not yet been used; group B (patients 4–9) in whom systemic steroids had been previously used with good effect, but recourse to a high dose was now to be avoided. Seven patients received one injection, one received two, and another three.

In group A the treatment achieved pain control for only four days in one patient and seven days in another. Both required subsequent high-dose systemic steroid therapy to control the scleral inflammation, instituted two weeks after injection. In the third, although pain was controlled, a large scleral nodule remained unaffected. No steroids were required in this patient, however, as there was now no active inflammation.

In group B only one patient (no. 8) did not require an increase of systemic steroids to achieve disease control eventually. The use of high-dose steroids, however, was delayed for five months in one patient and nine months in another. In patient nine the use of orbital floor steroids allowed a dangerously high blood pressure to be satisfactorily controlled before oral steroids were started.

There were no complications from the procedure, which was generally well tolerated by the patient.

Discussion

This study has demonstrated a limited role for orbital floor steroid in the management of non-

necrotising scleritis. Although the local steroid does exert some anti-inflammatory action, this tends to be short-lived. Systemic NSAIDs, particularly flurbiprofen and indomethacin, are well established as the first-line treatment of non-necrotising scleritis.¹ Necrotising scleritis, however, always requires high-dose systemic corticosteroids, sometimes with additional immunosuppressives such as cyclophosphamide and azathioprine.⁵ Systemic steroids are also used in cases of non-necrotising disease which do not respond to NSAIDs. Although they are generally very effective at controlling scleral inflammation, their use is associated with many side effects which can preclude their use. 'Pulse' steroid therapy has been used to try to reduce these side effects^{6,7} and more recently cyclosporin A.⁸

Patients with uniocular intraocular inflammation have long been spared treatment with systemic steroids by the administration of orbital floor depot steroid preparations. Watson and Hayreh⁹ have stated that intraorbital steroid injections have only a transient effect with scleritis, and that frequent injections are required for disease control. Benson,¹⁰ however, states that in posterior scleritis retrobulbar steroids can be effective and without complications.

It is always desirable to minimise, or avoid altogether, the use of systemic steroids, in the management of non-necrotising scleritis. Mondino and Phinney,¹¹ reporting on six patients with non-necrotising scleritis who were not satisfactorily controlled on either NSAIDs or steroid alone, managed to reduce the dose of controlling systemic corticosteroid by combining prednisolone with oral indomethacin. We have reported on five patients with non-necrotising disease in whom the concurrent use of cyclosporin A allowed the reduction of systemic steroids without relapse.¹² In this study the use of orbital floor steroids allowed systemic steroids to be avoided altogether in one patient, to be maintained at a low dose of 5 mg in another, and delayed the progress of disease in two others. Perhaps the most important function was in tiding over a patient, whose high blood pressure presented a positive contraindication to the administration of high-dose steroids, until the blood pressure could be reduced.

NSAIDs remain the initial therapy for non-necrotising scleritis, followed by systemic steroids should the NSAIDs prove to be ineffec-

tive. If the use of steroids is temporarily contraindicated, however, or should it prove to be difficult to reduce the minimum controlling steroid dose, then orbital floor steroid should be considered as a useful adjunct.

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