Necrotizing Scleritis Following Diode Laser Transscleral Cyclophotocoagulation

Sunny Y. Shen, MRCS
Jimmy S. M. Lai, FRCS, FRCOphth
Dennis S. C. Lam, FRCS, FRCOphth

Abstract. A 23-year-old Chinese woman presented with necrotizing scleritis 10 months after diode laser cyclophotocoagulation for the treatment of her neovascular glaucoma. There were areas of scleral thinning with uveal prolapse in the superior quadrant. The inflammation and progression of the staphyloma were controlled with systemic steroids. Diode laser cyclophotocoagulation may not be safe in patients suspected to have underlying systemic vasculitic diseases. [Ophthamnc Surg Lasers Imaging 2004;35:251-253.]

INTRODUCTION

Necrotizing scleritis is a serious ocular disease that can be associated with systemic autoimmune vasculitis.\(^1\) It may also occur after uneventful cataract surgery and pterygium excision,\(^2,3\) in which case it is called surgically induced necrotizing scleritis. It is recognized clinically as a focus of intense scleral inflammation. As the disease progresses, the sclera becomes thinner with staphyloma formation because of collagen destruction. The underlying uveal tissue will become visible. In the presence of increased intraocular pressure (IOP), the staphyloma formation is intensified and the globe may perforate. Diode laser transscleral cyclophotocoagulation has been used to treat neovascular glaucoma.\(^4\) We describe a case of necrotizing scleritis following diode laser transscleral cyclophotocoagulation for the treatment of neovascular glaucoma. We are unaware of previous reports of diode laser transscleral cyclophotocoagulation induced by necrotizing scleritis and could find no reference to it in a computer search using the MEDLINE database.

CASE REPORT

A 23-year-old Chinese woman presented with neovascular glaucoma of the right eye secondary to central retinal vein occlusion. She also had a history of optic neuritis in the same eye 2 months previously. On presentation, her right eye had no light perception and the IOP was 52 mm Hg. The IOP failed to be controlled with maximal antiglaucomatous medications and there was severe ocular pain. Diode laser (IRIS Medical Instruments, Inc., Mountain View, CA) transscleral cyclophotocoagulation using the G-probe was performed twice for IOP and pain control. The inferior 180° and the whole 360° were treated in two separate sessions. The laser energy used was 2,000 mW and adjusted to an audible "pop" subthreshold level with a burn duration of 2 seconds. A short course of topical steroid (prednisolone acetate 1%) was prescribed after each treatment. The IOP was maintained at approximately 30 mm Hg with three antiglaucomatous eye drops and the eye was pain free.

Ten months after the second treatment, the eye became inflamed and developed areas of scleral thinning with uveal prolapse in the superior quadrant (Fig. 1). Ultrasound biomicroscopy showed various degrees of scleral thinning, mainly in the superior quadrant (Fig. 2). The site corresponding to the lesion shown in Figure 1 had a scleral thickness of 0.3 mm. There was massive inflammatory cellular reaction in the anterior chamber. The patient had no symptoms of arthralgia or arthritis. Results of chest x-ray and routine blood tests were normal. Results of specific hematologic...
investigations for connective diseases including rheumatoid factor, anti-extractable nuclear antigen, cytoplasmic-antineutrophil cytoplasmic antibody (C-ANCA), anticardiolipin antibody (IgG and IgM), antinuclear antibody, lupus anticoagulant, C-reactive protein, protein C, and protein S were negative. However, the anti-DNA titre was mildly raised and the C-ANCA level was positive. The levels of complement C3 and C4 were decreased. A swab for bacterial culture yielded no organism. A course of oral prednisolone (60 mg daily) was prescribed and the ocular inflammation responded. The scleral thinning was covered by intact conjunctival epithelium and remained static.

DISCUSSION

Surgically induced necrotizing scleritis has been reported following cataract extraction, trabeculectomy, squint surgery, and pterygium excision.\(^2\,^3\) Autoimmunity or hypersensitivity is the likely etiologic factor for its development.\(^3\)

Our case had certain similarities with surgically induced necrotizing scleritis. The scleral thinning occurred 10 months after laser cyclophotocoagulation. The area of thinning corresponded to the site of laser application. There was associated inflammation and the disease responded to systemic steroids. In surgically induced necrotizing scleritis, there is immune complex-mediated vasculitis, a Type III hypersensitivity, causing local tissue ischemia.\(^5\) Diode laser cyclophotocoagulation has been shown to cause extensive destruction of the pars plicata and damage to the sclera.\(^6\) The tissue damage may have activated the complement pathway with release of chemotactic factors for inflammatory cells. On the other hand, the scleral thinning in our patient may also be a direct damaging effect of the laser. However, the eye had no previous surgery and had normal scleral thickness before the laser treatment. Second, the staphyloma occurred in the superior quadrant where laser was applied only once and not in the inferior quadrant where laser was applied twice. Moreover, the delayed onset of the staphyloma and the response to steroid treatment suggested that surgically induced necrotizing scleritis was the cause of the scleral thinning rather than direct laser damage.

Although hematologic test results were suggestive but not confirmative of the presence of systemic autoimmune disease, the possibility could not be absolutely excluded. It has been reported that serological markers for autoimmune disorders are present in only 62% of surgically induced necrotizing scleritis.\(^3\) Moreover, the occurrence of optic neuritis and central retinal vein occlusion in this patient may signify an underlying systemic vasculitic disease.\(^7\)

In young patients presenting with neovascular glaucoma secondary to central retinal vein occlusion, a detailed systemic evaluation and laboratory tests should be performed to identify underlying autoimmune disease. Our case has shown that necrotizing scleritis can occur after diode laser transscleral cyclophotocoagulation. In view of the risk of surgically induced necrotizing scleritis, management of neovascular glaucoma secondary to central retinal vein occlusion in patients suspected of having systemic vasculitic disease must be cautious. It is recommended
that medical treatment be adopted as much as possible. If laser cyclophotocoagulation is used for medically uncontrolled cases, we suggest using a lower power without inducing "pop" and increasing the number of treatment sessions with fewer burns each session. Intensive topical steroid treatment after the procedure may minimize the risk of developing surgically induced necrotizing scleritis. A long-term close follow-up of patients is recommended because surgically induced necrotizing scleritis can occur many months after treatment.

REFERENCES