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BRIEF REPORTS

Surgically induced necrotizing scleritis after a routine extracapsular cataract extraction

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ABSTRACT

Objective

To report a case of necrotizing scleritis in an elderly diabetic patient who underwent uneventful extracapsular cataract extraction.

Methods

Observational case report.

Results

A 79-year-old diabetic female who had undergone extracapsular lens extraction consulted for unilateral severe eye pain and redness associated with scleral thinning, tenderness, blurred vision, and moderate vitritis. The patient was diagnosed to have surgically induced necrotizing scleritis based on the location, clinical findings, localized hypofluorescence with anterior segment fluorescein angiography, and posterior scleral thickening demonstrated by B-scan. There were no associated systemic findings. The patient responded well to oral steroids; symptoms resolved a few days after treatment was started.

Conclusion

Necrotizing scleritis affecting both anterior and posterior sclera may occur months after cataract extraction. Early diagnosis and management may result in good outcomes.

SCLERITIS is a severe inflammatory disease characterized by inflammatory cell infiltrates of the sclera. It is frequently associated with systemic diseases particularly connective tissue or vasculitic diseases.[1] Necrotizing scleritis is the most severe type of scleritis and may lead to severe pain, globe perforation, and loss of the eye.[2] We are reporting a case of necrotizing scleritis occurring after an uneventful extracapsular lens extraction.

A 79-year-old female consulted at the Ospital ng Maynila Medical Center because of a painful red right eye of 2 weeks duration. Eight months prior, the patient underwent routine extracapsular cataract extraction on the same eye with a postoperative visual acuity (VA) of 6/15 (20/40).

On presentation, patient complained of severe boring pain in the right eye that affected sleep. Visual acuity was 6/30 (20/70). The right eye was tender and violaceous with distinct scleral transparency over the superior limbus through which the underlying uveal tissue could be discerned. The conjunctival, episcleral, and deep episcleral vessels were congested (Figure 1). Cornea was clear with 2+ anterior-chamber cells and flare, absent keratic precipitates and 2+ vitreous cells. The polymethylmethacrylate posterior chamber intraocular lens was well-centered in the capsular bag. Indirect ophthalmoscopy revealed a hazy media with inflammatory cells visible at the area of the superior pars plana. Ocular movements were full and intraocular pressures normal.

The left eye had a visual acuity of hand movement with good light projection due to a brunescent cataract but was otherwise normal.

Medical history was unremarkable and review of systems negative. Physical examination was completely normal.

Workup showed a normal hematological profile, a mildly elevated erythrocyte sedimentation rate of 20 mm/hr, and elevated fasting blood sugar of 7.13 mmol/L. Antinuclear antibodies, rheumatoid factor tests, and chest X-rays were normal. The rheumatologist ruled out the presence of a systemic connective tissue disease based on patient's lack of symptomatology and normal laboratory exams.

A diagnosis of surgically induced necrotizing scleritis (SINS) was made. Patient was initially treated with indomethacin 50 mg/day and ketorolac (Acular, Allergan, CA, USA) eye drops but was shifted to oral prednisone 1mg/kg/day after several days of no response. After two days of oral corticosteroids, the eye pain and redness dramatically lessened. B-scan revealed a thickened posterior sclera (Figure 2). Anterior-segment fluorescein angiography revealed hypofluorescence of the affected areas corresponding to the thinned-out sclera. The patient completed 2 weeks of high-dose oral prednisone after which the dosage was tapered slowly over the next 6 weeks. The involved eye was quiet but manifested residual scleral thinning (Figure 3).

Surgically induced necrotizing scleritis has been reported to occur after various types of surgery, including cataract extraction through a limbal incision, glaucoma, strabismus, detachment, and pterygium.[4,5] The scleral inflammation is adjacent to or at the site of a surgical wound. Initially, the sclera will appear swollen followed by a notable

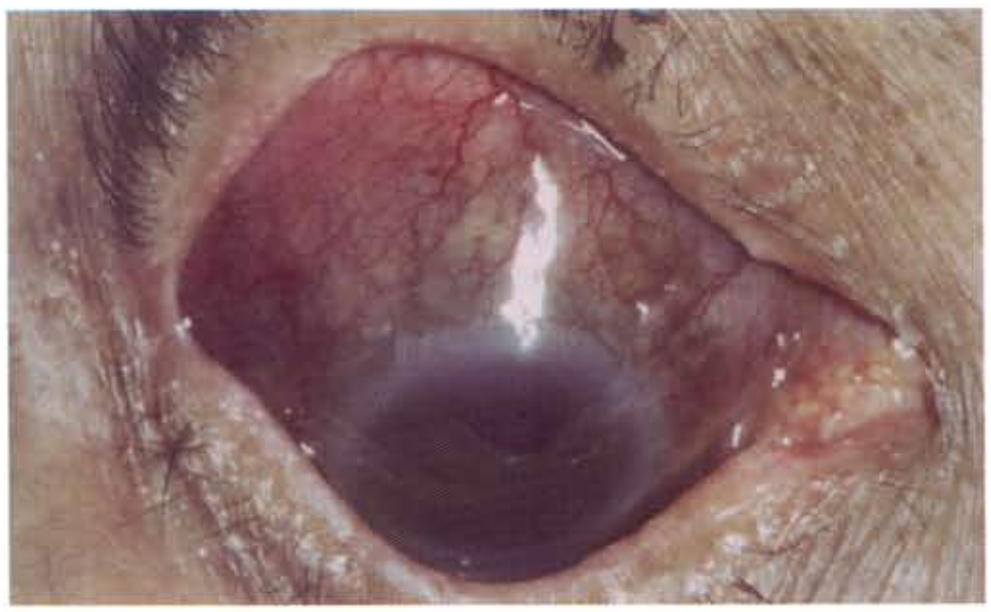

Figure 1. Acutely inflamed sclera with diffuse redness and dilated deep vessels.

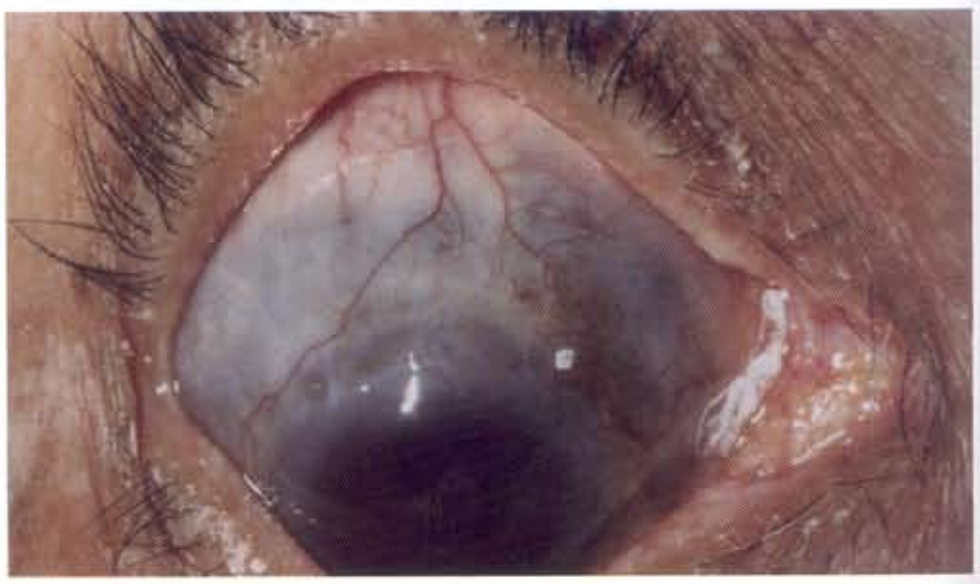

Figure 3. A white eye with residual scleral thinning through which uvea is visible after 1.5 months of corticosteroid treatment.

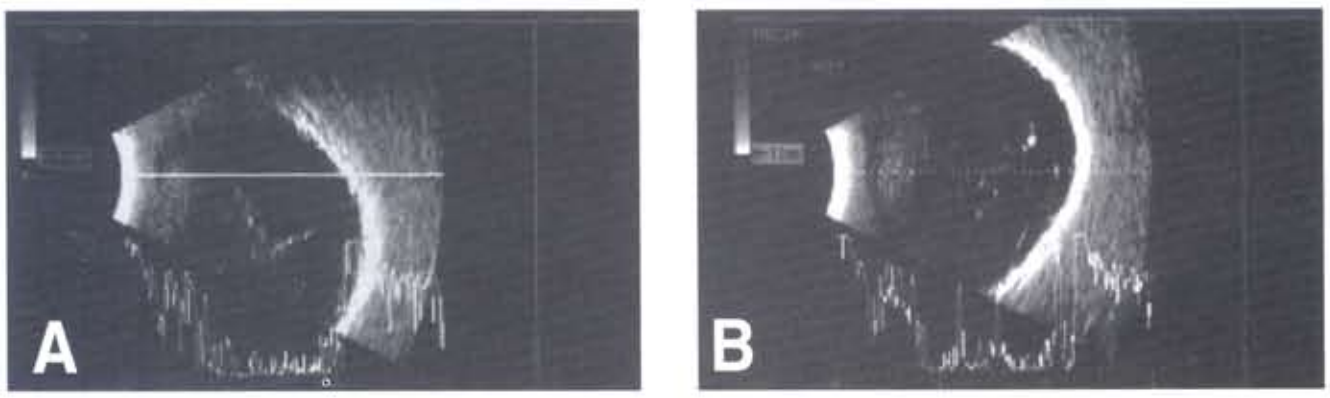

Figure 2. B-scan shows thickened posterior sclera of the right eye (A) compared to the left eye (B).

scleral transparency and thinning as the inflammation subsides or with recurrence of the disease.[6,7] A retrospective study by O' Donoghue[4] on 52 eyes affected with SINS described the following epidemiologic features: females predominate (30:13), mean age 68.2 years (range 32-87), mean onset within 5-7 months of the surgery reported, onset of disease 1 day to 40 years after surgery. Sixty-three percent had an underlying medical disorder, most commonly connective tissue disease and organ-specific autoimmune disease such as thyroid disorder or diabetes.[4] Our patient developed SINS 8 months after and had a high fasting blood sugar undetected until the occurence of the scleritis.

Causes reported were vasculitis precipitated by an induced localized ischemia in the area of the surgical incision (which does not explain the delayed onset of the disease) and other immunologic mechanisms.

The rapid response of patients with SINS to immunosuppressives is evidence that the immune system is actively involved at least in the continuation and maybe the initiation of the inflammatory response.[4] Sevel showed that the histologic appearance at the edge of a lesion of necrotizing scleritis is that of a delayed type of hypersensitivity response.[8] Surgical trauma or ischemia alters or exposes tissue antigens to the immune system, which recognizes them as foreign and starts the immune reaction.

Scleritis after a surgical procedure must always alert the surgeon to a probable infectious etiology when it presents as a suppurative scleral abscess. In such cases, laboratory work-up is essential to identify the causative organism so proper antibiotics can be given. The diagnostic challenge arises when scleritis is not associated with suppuration. Before starting treatment with corticosteroids, routine hematologic and immunologic profiles, as well as laboratory examinations, should be done to rule out syphilis, toxoplasmosis, and tuberculosis as possible causes that need systemic antimicrobial therapy.[3]

Treatment of SINS involves the use of potent immunosupressives. Nonsteroidal antiinflammatory drugs (NSAIDs) are usually ineffective for the condition. Systemic corticosteroids at a high dose (1 mg/kg/day) will usually relieve pain, which is the main improvement indicator. Maintenance low-dose steroid is necessary to keep the patient disease-free. In O'Donoghue's series, the maintenance period ranged from 2 months to 6 years. Adjunctive treatments include topical cyclosporine 2%, oral or intravenous cyclophosphamide, or oral azathioprine. Scleral and corneal patch grafting is indicated for perforation.

Treatment of SINS should be started early to improve visual outcomes. Clear corneal incision should be tried for the contralateral cataractous eye to prevent SINS.

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