variable postoperative onset of symptoms, ranging from days to years.

Conservative treatment like artificial tears and antibiotic ointments may be instituted at the clinician's discretion for a discrete period of time while monitoring for signs of regression or progression. Implementation of autologous serum tears may also be considered, especially for cases demonstrating recalcitrance of conjunctival epithelial defect resolution. Removal of the calcific plaque also facilitates healing. In more severe cases, additional treatment options available for utilization include placement of amniotic membrane grafts, autologous conjunctival flaps, or simultaneous placement of both.[5] Forms of tissue media available for scleral surface repair include sclera, cornea, pericardium, fascia lata, dermis, and cartilage.[9–14]

This case of scleral thinning illustrates a complication that can arise after the I-BRITE procedure. Comprehensive ophthalmologists should be aware of the potential risks and complications with this surgery, the surgery known as regional conjunctivectomy with MMC, and the alert issued by the ASCRS regarding the procedure.[6,15]

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Can J Ophthalmol 2016;51:e115–e116
0008-4182/16/$-see front matter
http://dx.doi.org/10.1016/j.jcjo.2016.03.004

Necrotizing scleritis after pterygium excision with fibrin glue–fixated conjunctival autograft in Behcet's disease

Surgically induced necrotizing scleritis (SINS) is an uncommon autoimmune reaction occurring at surgical wounds; only 4 cases after pterygium surgery have been reported.[1–4] Pterygium excision with conjunctival autografting has become the surgical technique of choice in the treatment of primary and recurrent pterygia because of the decreased recurrence rates.[5–8]

Scleritis occurring in patients with Behcet's disease has rarely been reported in the literature.[9] We present the first case of SINS after primary pterygium excision with fibrin glue–fixed conjunctival autograft in a patient with Behcet's disease.

A 48-year-old Caucasian female patient was referred to the ophthalmology clinic for treatment of nasal primary pterygium in her left eye. Pterygium excision and conjunctival autograft transplantation fixed with fibrin glue (Tisseel; Baxter, Vienna, Austria) were performed uneventfully. No adjunctive agents were used, and gentle wet field cautery was performed. On postoperative day 1, the slit-lamp examination revealed an avascular, de-epithelialized white pale graft and dehiscence of the graft edges with corneal epithelial defect at the site of the pterygium excision. There was no visible inflammation at the surgery region or on the graft. A wipe sample from the graft surface revealed no organisms in the smears, and the culture revealed no growth. Topical treatment with corticosteroid and antibiotics was initiated. The conjunctival graft was stitched from the 4 edges with 10.0 nylon (Alcon Laboratories, Fort Worth, Tex.) sutures after verification of

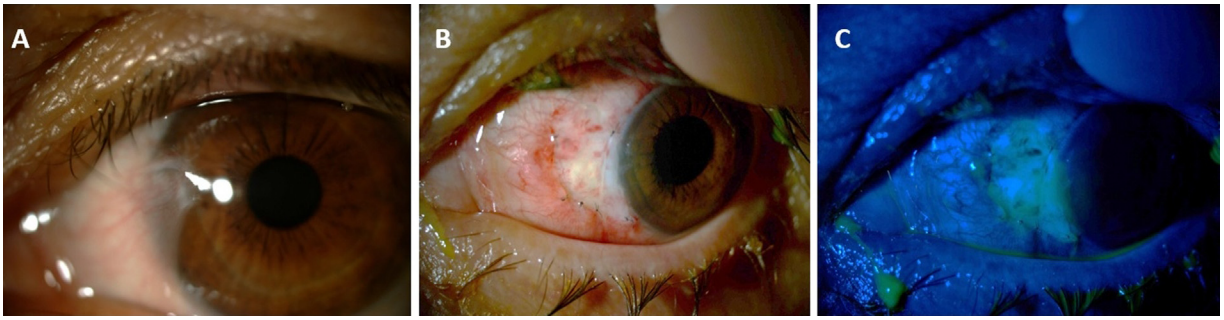

Fig. 1—Photograph of the anterior segment of the left eye. (A) Preoperative view of the nasal pterygium. (B) (Postoperative second day) The subconjunctival hemorrhage areas are seen under the avascular conjunctival autograft. The 10.0 nylon stitches are visible at the dehiscent regions of the graft. (C) (Postoperative second day) The fluorescein staining of de-epithelialized autograft is shown.

the correct side placement on postoperative day 2 (Fig. 1). Despite the adopted treatments, the graft and the sclera started thinning without any accompanying ocular symptoms. The conjunctival graft was removed and sent for a detailed pathology and microbiology work-up, and a new autograft was implanted using fibrin glue and 10.0 nylon (Alcon Laboratories) sutures for fixation on postoperative day 14, which resulted in similar avascularization within a week (Fig. 2). Smears and culture of the removed graft did not show any microorganisms. Pathological evaluation revealed ischemic necrosis in the graft. A detailed investigation of the patient's medical history revealed a previous suspicion and investigation for Behcet's disease. A diagnosis of SINS was suspected, and systemic steroid (oral prednisolone 32 mg/day, with a weekly taper) and topical autologous serum therapy were initiated on postoperative day 22. Despite the systemic and topical treatments, the new graft showed gradual thinning. Blood studies and venereal disease research tests showed normal results. Systemic examination was performed by a rheumatologist, and the presence of oral aphthae and erythema nodosum helped to confirm the diagnosis of Behcet's disease.

The nasal teno-conjunctiva was advanced through the limbus, and a cryopreserved amniotic membrane, obtained from the Istanbul University Lions Eye Bank, was first cut (oversized, approximately 4–6 mm larger than the size of the scleral bed) and then transplanted over the flap on postoperative day 32 (Fig. 3). Because of the nasal retraction, the nasal conjunctiva and Tenon's capsule were advanced to the limbus again and were refixed to the corneolimbal junction with 10.0 Vicryl sutures on postoperative day 42. Ten days after the last procedure, the amniotic membrane had degraded, and the conjunctiva had healed without any scar (Fig. 4).

Over the next 10 days, the conjunctival inflammation decreased. On follow-up, at postoperative day 80, the patient presented with anterior uveitis that was managed with topical prednisolone acetate and cyclopentolate drops. Three months later, the patient was comfortable, with best-corrected visual acuity of 20/20 in the operated eye. She was continued on artificial tear substitutes.

SINS induction after pterygium excision with conjunctival autograft has been reported previously in only 4 case reports, and all grafts were secured with sutures.[1–4] In our case the conjunctival autograft was fixated with fibrin glue, which was previously implicated to improve postoperative comfort, reduce surgery time, avoid suture-related complications, and result in lower recurrence rates compared to suturing.[5] SINS and scleromalacia perforans related to Behcet's disease have been reported in only 1 case report with no history of surgery. To the best of our knowledge, SINS has not been reported in a patient who had

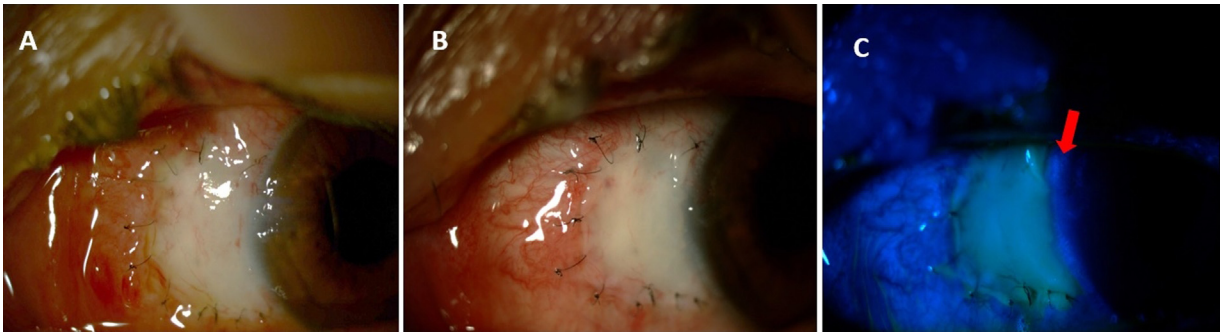

Fig. 2—Photograph of the anterior segment of the left eye. (A) (Postoperative 21st day) Similar avascular and pale graft is seen in a week after a new autograft was fixed with fibrin glue and 10.0 nylon stitches. (B) (Postoperative 30th day) The retraction and gradual thinning of the graft at the limbal area is shown. (C) (Postoperative 30th day) The fluorescein staining of de-epithelialized autograft and limbal graft retraction (red arrow) is visible with the cobalt blue lightening.

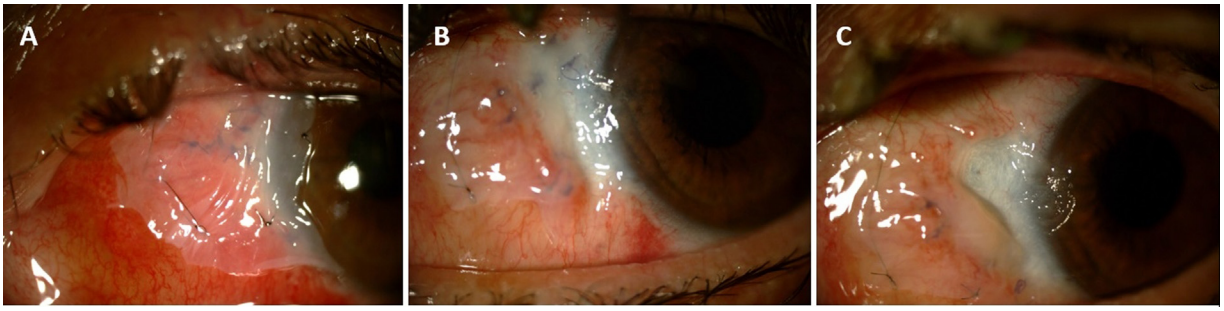

Fig. 3—Photograph of the anterior segment of the left eye. (A) (Postoperative 32nd day) Nasal teno-conjunctival advancement and amniotic membrane transplantation over this advancement flap is shown. (B) (Postoperative 35th day) Nasal flap retraction and gradual thinning of the amniotic membrane graft is shown. (C) (Postoperative 36th day) Gradual thinning of the amniotic membrane graft and melting of the underlying white, ischemic sclera is shown.

undergone autografts fixed with fibrin glue and in a patient who has a diagnosis of Behcet's disease.[10]

The avascular scleral bed, caused by disruption of the episcleral vasculature during ocular surgery and excessive use of cautery, and incorrect placement of the conjunctival graft are the 2 main reasons for graft necrosis reported in the literature.[11,12] These etiologies need to be ruled out before making a diagnosis of SINS.

An abnormal pallor and avascular appearance are the first postoperative findings to suspect inversion of the graft. The graft will shrink and become necrotic within 24–48 hours if it is inverted so that the epithelium is opposed to the sclera, instead of Tenon's capsule.

The latent period of presentation of necrotizing scleritis after surgery may vary widely between 1 day and 51 years.[1–12] In the case reported, during the first postoperative hours, the case seemed to be a graft inversion because of the early shrinkage and necrosis after surgery.[13] We eliminated graft inversion by testing the nonepithelial fluorescein staining of the graft sides, which would be expected to show diffuse staining if the nonepithelial side of the graft was above. The position of the graft was also checked with the operating microscope, and the edges were sutured on the second day to avoid possible graft necrosis because of displacement.

Delayed-type hypersensitivity reaction directed against ischemia or an antigen revealed and altered after the tissue injury has been implicated as the causative factor of SINS.[4,11] The rapid response to immunosuppressive agents and the more frequent occurrence in patients who had multiple surgeries support the view that an immunologic reaction is involved in the pathogenesis.[2] Alternative theories cover generalized immune complex deposition in the episcleral vessel wall from vasculitis that is similar to the pathogenesis of Behcet's disease.[1] This theory may help describe the predisposition to SINS in our patient.

Systemic nonsteroidal anti-inflammatory drugs, systemic corticosteroids, and immunosuppressive drugs have been reported to be effective in the management of SINS. Suspecting SINS, we initiated systemic steroid therapy in addition to topical steroid. In our case, SINS did not respond to local and asystemic steroid therapy, which was reported in a previous similar case study.[14]

Amniotic membrane transplantation was previously reported to be successful in reducing inflammation and in preventing globe perforation.[15] An amniotic membrane was transplanted over this conjunctival advancement flap and over the adjacent cornea, with the epithelial side up. The patient responded well to this combined teno-conjunctival advancement with amniotic membrane transplantation. The scleral thinning healed completely within 10 days.

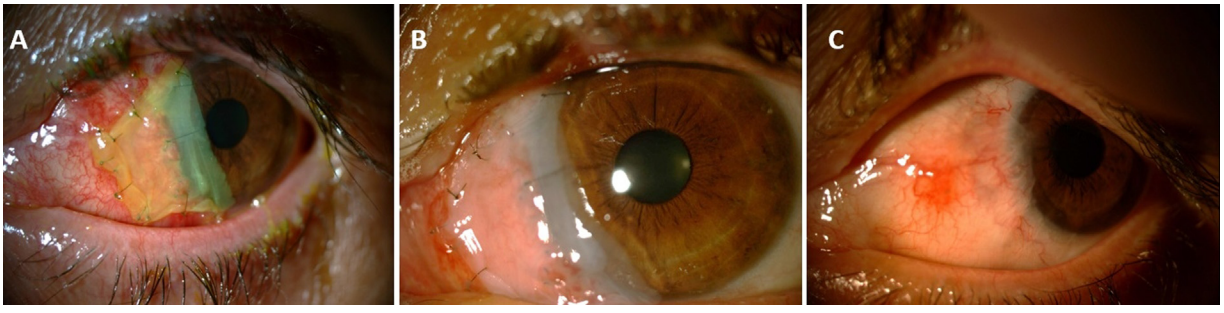

Fig. 4—Photograph of the anterior segment of the left eye. (A) (Postoperative 42nd day) Refixation of nasal teno-conjunctiva and retransplantation of amniotic membrane over this advancement flap is shown. (B) (Postoperative 47th day) Nasal flap and amniotic membrane are in correct positions. (C) (Postoperative 52nd day) The amniotic membrane degraded, and the conjunctiva healed without a trace.

A pale, white avascular graft may occur on the first postoperative day with underlying avascular sclera and should lead one to suspect this complication, especially in a patient with Behcet's disease. SINS in Behcet's disease can be surgically managed successfully using the combined teno-conjunctival advancement with amniotic membrane transplantation technique.

Disclosure: The authors have no proprietary or commercial interest in any materials discussed in this article.

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Can J Ophthalmol 2016;51:e116–e119
0008-4182/16/$-see front matter
http://dx.doi.org/10.1016/j.jcjo.2016.03.007

Unique presentation of anti-GQ1b antibody syndrome

Miller Fisher syndrome (MFS), first described in 1956, includes a triad of ophthalmoplegia, ataxia, and areflexia,[1] and it was thought to be a variant of Guillain–Barre syndrome. The initial discovery of anti-GQ1b antibody raised the possibility of using it as a specific marker for MFS.[2,3] It is now known that elevated anti-GQ1b antibody production can be seen within a spectrum of conditions, including acute ophthalmoparesis, Bickerstaff's brainstem encephalitis, and Guillain–Barre syndrome, in addition to MFS. Here, we report a case of elevated anti-GQ1b antibodies with marked pupillary findings and ophthalmoplegia, in the absence of areflexia, ataxia, or other neurological findings.

An 18-year-old previously healthy, right-handed, Caucasian male patient presented to the emergency room because of 1 day of acute onset bilateral mydriasis, left upper lid ptosis, and binocular diplopia that was worse on right gaze. He was in his usual state of health until 1 week before presentation, when he developed upper respiratory tract infection symptoms with nasal congestion, for which he took over-the-counter decongestants. On the night before presentation, he took 80 mg of dextromethorphan and 800 mg of guaifenesin (the daily maximum recommended dose) and became symptomatic with the previously described presentation upon awakening. Otherwise, he denied blurry vision, pain with eye movement, headache, fever, or chills, and further review of systems was entirely negative. He denied smoking, alcohol, or use of illicit drugs. His family history is significant for his father having multiple sclerosis.

On ophthalmologic evaluation, visual acuity was 20/20 OU with normal colour vision (10/10 Ishihara colour plates). Both pupils were 8 mm and did not constrict to direct light or accommodative stimulus. Ocular motility testing revealed a 20% limitation of abduction of the right eye and 10% limitation of elevation of the left eye. Two millimeters of left upper lid ptosis was noted, as well as a positive Cogan's lid twitch; however, no improvement was noted after 2 minutes of ice placed on the left upper lid. Orbicularis strength was normal and symmetric. The remainder of his ophthalmologic examination findings were within normal limits. Findings of detailed neurological examination were otherwise normal. Specifically, reflexes were normal, with no ataxia or dysdiadochokinesia. Magnetic resonance imaging/angiography (MRI/A) of the brain and orbits was unremarkable. Lumbar puncture revealed a normal cerebrospinal fluid (CSF) profile, without albuminocytologic dissociation.