Infectious necrotizing scleritis and proliferative vitreoretinopathy after scleral buckling in a patient with atopic dermatitis

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ABSTRACT

Purpose: To report a case of necrotizing scleritis and proliferative vitreoretinopathy due to an acute infection following scleral buckling for the management of rhegmatogenous retinal detachment in a patient with untreated atopic dermatitis.

Observations: A 40-year-old man with untreated atopic dermatitis presented with rhegmatogenous retinal detachment in his right eye. He underwent uncomplicated scleral buckling surgery with an encircling silicon sponge band, then noticed severe hyperemia and purulent discharge in the eye at 3 weeks after surgery. The silicon sponge was exposed and had migrated anteriorly. The patient was then diagnosed with post-scleral-buckling infection and underwent scleral buckle removal. Marked scleral thinning due to necrotizing scleritis was evident beneath the scleral buckle. Eye discharge culture findings were positive for Staphylococcus aureus. After removal of the scleral buckle, the patient’s necrotizing scleritis improved, but he developed proliferative vitreoretinopathy. The patient then underwent pars plana vitrectomy, which resulted in reattachment of his retina.

Conclusions and Importance: Although postoperative infection within 1 month after scleral buckling is rare, surgeons should note that patients with atopic dermatitis are at high risk of postoperative infection after scleral buckling, leading to the development of sight-threatening complications such as necrotizing scleritis, which could lead to incomplete retinal attachment and proliferative vitreoretinopathy.

1. Introduction

Rhegmatogenous retinal detachment (RRD) is a sight-threatening condition, with an annual incidence of 1 in 10,000 people. Major surgical procedures for RRD include scleral buckling (SB), pars plana vitrectomy (PPV), or a combination of the two methods. 1–6 While there has been a shift over the past two decades toward PPVs due to improved surgical technology, 7–9 primary SB remains the treatment of choice for certain patients with RRD, such as those without posterior vitreous detachment, round or atrophic holes, or inferior breaks, as well as those on dialysis. 10 The incidence of SB infection was 3%–5% among patients with RRD in the 1970s. 11–13 Various interventions (e.g., soaking of the silicon sponge in antibiotics, washing the surgical field, careful draping, and improving the silicon sponge material) have reduced the incidence of SB-related infections to <1%. 14 However, some patients, such as those with atopic dermatitis, are reported to have a high risk of SB infection. 15–17

Here we report a case of necrotizing scleritis and proliferative vitreoretinopathy after SB infection in a patient with untreated atopic dermatitis.

2. Case report

A 40-year-old man presented with RRD in his right eye and was referred to Hiroshima University Hospital. He had undergone cataract surgery in both eyes 7 years prior; he had also undergone PPV with silicon oil for RRD in his left eye 5 years prior. Furthermore, he had untreated atopic dermatitis (Fig. 1). On examination, his best-corrected visual acuity findings were 20/200 in the right eye and hand motion in the left. Intraocular pressure findings were 10 mm Hg in the right eye and 8 mm Hg in the left. Slit lamp examination showed bilateral blepharitis, bilateral eye lid eczema with exudates, and atopic keratoconjunctivitis in both eyes. There was no intraocular inflammation in the right eye, but posterior synechiae (360°) with myosis were observed in the left eye. Fundus examination of the right eye showed macula-off...
RRD with atrophic retinal holes in the nasal and temporal periphery (Fig. 2). The left fundus could not be evaluated because of myosis and silicon oil emulsification. Because the patient had been lost to follow-up after the vitrectomy in the left eye, visual function in that eye had deteriorated due to postsurgical inflammation.

SB was chosen for the right eye, based on the reasoning that it presented a lower risk of postsurgical intraocular inflammation than did PPV. Thus, the patient underwent SB surgery with an encircling silicon sponge band (#506, MIRA Inc, Uxbridge, MA, USA) and cryopexy. Before surgery, the skin was scrubbed with 10% povidone-iodine (Niten, Nagoya, Japan). During surgery, the operative field was repeatedly irrigated with 0.25% povidone-iodine. Mattress sutures with 4–0 Mer-siline (Ethicon, Inc. N.J., USA) were placed over the silicon sponge for scleral fixation. After surgery, the patient was treated with topical 0.5% moxifloxacin (four times daily), betamethasone sodium phosphate (four times daily), tropicamide (twice daily), and dorzolamide/timolol (twice daily). The patient was referred to a dermatologist and was prescribed 0.1% tacrolimus ointment (once daily) and 0.3% heparinoid cream (twice daily) for his face. Gradual improvement in eyelid eczema was observed after this treatment. One week after surgery, best-corrected visual acuity in the patient’s right eye remained 20/200, but reduction of subretinal fluid was evident (Fig. 3). Although the patient exhibited trace conjunctival injection, no obvious intraocular inflammation was observed (i.e., no anterior chamber cells, vitreous haze, or retinal vasculitis). However, the patient noticed severe hyperemia and purulent discharge in the right eye at 3 weeks after surgery. His best-corrected visual acuity had deteriorated to 20/400; he also exhibited marked lid edema and conjunctival injection. Because the conjunctiva and sclera around the sponge were necrotized, the silicon sponge had become exposed and then migrated anteriorly (Fig. 4). The patient had 2+ anterior chamber cells in the right eye on slit lamp examination. Fundus examination of the right eye showed 2+ vitreous haze without obvious retinal detachment. He was therefore diagnosed with post-SB infection and underwent scleral buckle removal. Marked scleral thinning was evident beneath the scleral buckle site at the time of surgery (Fig. 5). Superonasal sclera showed discoloration (necrotic slough and bare choroid). Eye discharge culture results were positive for Staphylococcus aureus, which exhibited sensitivity to cephalosporin, cefmenoxime, gentamicin, and vancomycin; it showed resistance to levofloxacin, gatifloxacin, and ofloxacin. The patient was therefore diagnosed with infectious necrotizing scleritis and received 2 g/day of cefazolin sodium intravenously for 4 days; he also received topical cefmenoxime hydrochloride, gentamicin, and erythromycin. After removal of the scleral buckle, the conjunctival injection improved, such that the bare sclera was covered by conjunctiva (Fig. 6). Although the bare sclera was completely covered by conjunctiva at 2 weeks after buckle removal, proliferative vitreoretinopathy developed. The patient then underwent PPV with sulfur hexafluoride gas tamponade. The sclera was very thin at 3 mm–10 mm from the corneal limbus because of buckle infection. To prevent wound closure failure or scleritis reactivation, the vitrectomy ports were set at 2 mm from the corneal limbus. Although the patient showed vitreous opacity due to vitritis, there were no signs of endophthalmitis (e.g., retinal infiltration, hemorrhage, or vasculitis). He exhibited inferior retinal detachment with a primary nasal retinal hole. A subretinal fibrotic ‘clothesline’ membrane extended horizontally from

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**Fig. 1.** Pictures of atopic dermatitis in this case: (A) face, (B) hands. The patient had not received treatment for his atopic dermatitis for several years. He had bilateral blepharitis and eczema. He rubbed both eyes frequently because of constant itching.

**Fig. 2.** Fundus photograph (A) and optical coherence tomography image (B) of the right eye. Macula-off rhegmatogenous retinal detachment with atrophic retinal holes was present in the nasal and temporal periphery (not shown). Vertical arrow shows the direction of the optical coherence tomography image.

**Fig. 3.** Optical coherence tomography image of the right eye at 1 week after scleral buckling. Subretinal fluid was reduced by scleral buckling.
5 o’clock to 8 o’clock. The subretinal strand was removed through an intentionally created retinal hole, using 25-gauge internal limited membrane forceps (Fig. 7). The surgery was conducted uneventfully and the retina was reattached (Fig. 8). The patient’s right best-corrected visual acuity improved to 20/40. His necrotizing scleritis and ocular inflammation subsided after buckle removal and vitrectomy.

3. Discussion

Atopic dermatitis is associated with various ocular complications such as blepharitis, keratoconjunctivitis, keratoconus, cataract, glaucoma, herpetic ocular disease, and retinal detachment. Here, we reported the case of a patient with untreated atopic dermatitis who had RRD that led to necrotizing scleritis and proliferative vitreoretinopathy following SB infection; the findings in this case demonstrated the difficulty of surgical management in such patients.

The incidence of post-SB infection is low. However, when such infections occur, they may lead to sight-threatening complications. The most common cause of blepharitis in patients with atopic dermatitis is staphylococcal disease. It has been reported that methicillin-resistant S. aureus infection after SB procedures is present in 18.8% of patients with atopic dermatitis, but only 0.4% of those without atopic dermatitis. In the present case, it was difficult to attach the surgical drape to the affected skin, which led to intraoperative exposure of the bare eyelid. The bacteria (originally in infected skin) could presumably access the buckle material easily because of surgical-drape detachment, leading to acute postsurgical infection.

Postoperative necrotizing scleritis occurs most commonly in patients with pterygium, followed in frequency by patients who have undergone cataract surgery and those who have undergone retinal detachment surgery. Whereas >70% of cases of necrotizing scleritis are of noninfectious etiology in patients who have undergone cataract surgery, infection is predominant in patients with pterygium (71.4% of cases) and those who have undergone SB (97.2% of cases). The most common causative organisms are staphylococci. Atopic dermatitis is thought to exert a synergistic effect with inflammation present in other organs, similar to other immune-mediated inflammatory diseases. Additionally, bacterial protein products of S. aureus can act as triggers or accelerate inflammation.

Numerous risk factors have been identified for the development of proliferative vitreoretinopathy; these include pre-/post-surgical prolonged intraocular inflammation, vitreous hemorrhage, choroidal detachment, large number of retinal breaks, excessive cryotherapy, and endolaser usage. In our patient, multiple factors may have driven the abnormal wound healing response, thereby contributing to the development of proliferative vitreoretinopathy. First, the patient was a smoker who had a history of intraocular surgery; second, his retinal detachment involved the macula; third, postoperative SB infection had led to infectious necrotizing scleritis and vitritis; and fourth, he developed incomplete retinal reattachment due to early scleral buckle removal.

The major treatments for RRD are SB and PPV. Uncomplicated situations, such as RRD with good visibility of the fundus and single breaks, are usually treated with SB. In contrast, PPV is indicated in patients with complicated RRD (i.e., those with giant retinal tears, vitreous hemorrhage, breaks at the posterior pole, or proliferative vitreoretinopathy). For patients with RRD who have multiple retinal breaks in different quadrants, without complications of proliferative vitreoretinopathy, the choice of surgical procedure varies among surgeons. This patient’s case was complicated because, although he was young, he exhibited pseudophakia and had diffuse RRD with multiple atrophic holes (nasal and temporal). The patient was first treated with SB in the right eye because
of his young age and the surgical outcome of vitrectomy in his left eye had been poor. While atopic dermatitis is a risk factor for postsurgical infection, there have been no reports of an association between atopic dermatitis and endophthalmitis after PPV. The ideal surgical procedure for patients with concurrent RRD and atopic dermatitis remains unclear. Nevertheless, if a patient presents with RRD and untreated atopic dermatitis, dermatological treatment should be initiated immediately to lower the risk of postoperative infection.

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**Fig. 6.** Composite 9-gaze slit lamp photographs of the right eye after silicon sponge removal. The conjunctival defect was epithelized with reduction in conjunctival injection. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

**Fig. 7.** Intraoperative view of pars plana vitrectomy. Subretinal strand was removed, with 25-gauge forceps, through intentional retinal hole. Yellow arrows shows subretinal strand. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

**Fig. 8.** Fundus photograph of the right eye after pars plana vitrectomy. There were well-healed laser scars around the retinal hole and iatrogenic retinal hole. The subretinal strand was completely removed.
4. Conclusions

Postoperative infection after SB is rare, but surgeons should be aware that patients with atopic dermatitis have a relatively high risk of postoperative infection after SB, which may cause sight-threatening complications such as necrotizing scleritis and proliferative vitreoretinopathy.

Patient consent

Written informed consent was obtained from the patient for publication of this case.

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Authorship

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Declaration of competing interest

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