Surgically Induced Necrotizing Scleritis Masquerading as Buckle Extrusion

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ABSTRACT

A case of surgically induced necrotizing scleritis (SINS) after 360 degrees circumferential buckling in a 62 years old man. Surgery was uneventful and patient-reported improved vision few days after surgery. However, about a week after surgery he noticed boring pain that became progressively severe. Vision started to decrease but no photophobia. Patient defaulted and did not report for check-up until one month after surgery. Ocular examination revealed a mildly edematous lid, clear cornea and a band-like necrosed sclera at 4 to 8 0'clock quadrant. On a cursory look, the sclera necrosis was thought to be an extruded silicone tire. The anterior chamber was clear with no hypopyon. The retina was attached. High index of clinical suspicion is required to differentiate scleral necrosis from buckle extrusion in some instances. This report highlights a case of sceral necrosis masquerading as buckle extrusion.

Keywords: Scleral Necrosis, Scleral Buckle, Retinal Detachment

INTRODUCTION

Scleral necrosis is a rare complication of ocular surgery with potentially devastating consequences to the eye. Known aetiological factors involved in scleral necrosis includes keratoconjunctivitis sicca, infections and autoimmune vasculitis. Surgically-induced necrotizing scleritis (SINS) has been described following intracapsular and extracapsular cataract surgery, secondary intraocular lens insertion, strabotomy, vitrectomy, surgical iridectomy, trabeculectomy, excess use of cautery, and repeated scleral trauma with forceps.[1-5] De la Maza and Foster[2] described nine patients with non-infectious necrotizing scleritis after ocular surgery, all of whom had autoimmune vasculitis. It typically occurs postoperatively as intense scleral necrosis adjacent to the site of previous scleral or limbal incision. It may or may not be associated with serious ocular complications such as peripheral ulcerative keratitis (PUK), which is often present adjacent to the SINS site.[6] This may progress to perforation with poor visual prognosis. Therefore, early diagnosis and prompt management with steroids, immunosuppressant and/or surgical treatment with amniotic membrane transplantation (AMT) are required for successful management.[6]

In this case report, we present an interesting case of post-scleral buckle SINS at the infero-temporal quadrant of the sclera. It was managed with buckle removal and steroids.

HISTORY

A 62-year-old man had uneventful circumferential buckling with type 276 tire. Barrage laser was applied to retina break. The patient was placed on topical dexamethasone 0.1% 2hourly, moxifloxacin 0.3% two-hourly and diclofenac tablets 50mg twice daily. Patient acknowledged progressive visual improvement until one week after surgery when the pain was reported. The pain was throbbing with associated frontal headaches, no eye discharges but the vision began to decline. Patient was reviewed one month after surgery having failed to honor earlier scheduled check-ups.

On examination, vision was Counting Finger, there were mild lid edema and conjunctival congestion. There was a circumferential whitish scleral patch from 4 to 8 0'clock quadrant which on cursory examination mimicked exposed tire (see attached figure). Because of pain, it could not be ascertained on slit-lamp examination if the whitish patch was the circumferential tire. The

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cornea was clear and the anterior chamber was also clear with no hypopyon, making anterior segment ischaemia unlikely. The crystalline lens was clear and retina was attached. Investigations which included Full Blood Count, Electrolytes Urea and Creatinine, Erythrocyte Sedimentation Rate, Fasting Blood Sugar, Urinalysis, Fasting Lipid Profile and Retroviral Serology were within normal limits. Patient was booked for exploration and possible tire removal.

Surgery was done under general anaesthesia. 360-degree conjunctival peritomy was done and tire reviewed and found to be intact and posterior to the whitish scleral patch. The patch was closely examined and observation made was that the scleral whitish patch was a necrosed sclera covered with sloughs immediately anterior to the tire. Conservative debridement was done to remove necrosed tissue while ensuring the globe was not perforated. The tire was removed after all sutures were severed. Debrided tissue was sent for microscopy, culture and sensitivity a[illegible]elded no growth. Patient was placed on t[illegible] which systemic steroid postoperatively. The visual acuity on last review was Hand Movement.

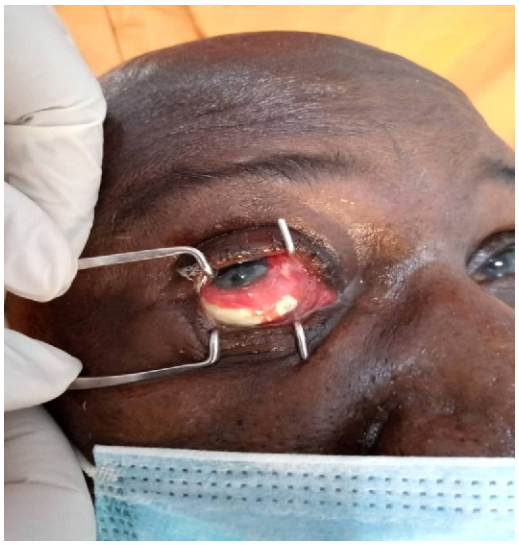

Figure 1: Circumferential whitish scleral patch of necrosed sclera mimicking exposed buckle.

DISCUSSION

Surgically induced necrotizing scleritis has been reported to occur after cataract extraction, trabeculectomy, squint surgery, pterygium surgery, and surgery for retinal detachment.[5,7] In SINS there is a variable latent period between surgery and presentation, which may vary from 1 day to 51 years.[4] The area of scleral melt tends to develop adjacent to the surgical wound and may extend to involve the cornea and whole anterior segment. These cases may or may not be associated with inflammation and PUK adjacent to SINS.[4]

Causative factors such as local ischemia due to disruption of episcleral vasculature during ocular surgery and excessive use of cautery have been implicated in the pathogenesis of SINS. However, the rapid response to immunosuppressive agents also supports the view of an immunologic reaction involved in the pathogenesis.[8] The pathogenesis of PUK and SINS has not been fully elucidated, but both T cell and antibody-mediated pathways have been implicated in the disease process.[9] This explains the simultaneous presence of SINS and PUK.

There were neither symptoms nor signs of auto-immune disorders in this index case. Cautery was not used and neither was any extraocular muscle severed that could have compromised anterior ciliary artery. The clear cornea and anterior chamber suggested there was no anterior segment ischaemia. The case presented here did not have clinical or laboratory evidence of an active infective process causing the scleral ulceration. There was also no clinical evidence of collagen disease or serological evidence of immunosuppression. There was no use of depot steroids or previous exposure to antimetabolites and irradiation. The likely cause was ischemic necrosis possibly due to occlusion of episcleral vessels. Vascular closure of the episcleral vessels can be documented by fluorescein angiography of the anterior segment, a facility not available in our center. Scleral ulceration after ocular surgery can have diverse etiologies and occlusion of episcleral vessels is one of them. Clinical workup and investigations can guide us to the possible etiology so that it can be

treated correctly. While immunosuppression is the treatment of choice in surgically-induced necrotizing scleritis, it may be disastrous in infectious ulceration and of no benefit in ischemic ulceration. In the current case, removal of the circumferential 360 degrees scleral buckle, topical and systemic steroids curtailed disease progression and led to remission of scleritis. The take home message is that non-infectious scleral necrosis could follow a successful scleral buckle which may need removal if necrosis does not abate and scleral perforation becomes imminent.

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