SCLERAL NECROSIS AND INFECTION 15 YEARS FOLLOWING PTERYGIUM EXCISION

K G Au Eong, P S F Tseng, A S M Lim

ABSTRACT

Scleral necrosis and infection are serious late complications of pterygium treatment and are difficult to manage. We describe a 70-year-old Chinese male who presented with scleral necrosis and Pseudomonas aeruginosa infection 15 years after the excision of a pterygium. The infection was treated early and aggressively with intensive topical and intravenous antibiotics and the thin necrotic sclera was reinforced with a donor scleral patch graft when the scleral infection was clinically controlled. The integrity of the globe was maintained by a thin layer of sclera anterior to the graft after the graft gradually shrunk in size and retracted posteriorly. The eye was saved from possible scleral perforation and endophthalmitis. This case is reported to highlight the importance of early aggressive treatment of infection and the value of prophylactic repair of scleral necrosis in the management of these late complications of pterygium treatment.

Keywords: *exudative retinal detachment, pterygium treatment, scleral infection, scleral necrosis, scleral patch graft.*

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INTRODUCTION

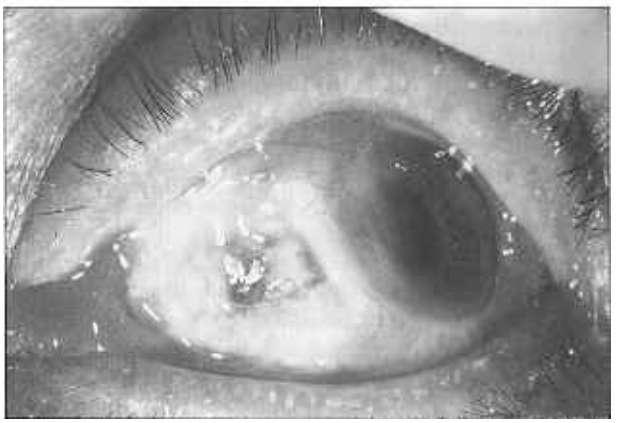
Scleral necrosis is a known late complication of pterygium treatment[1]. In a recent written survey on the treatment of pterygium among all ophthalmologists in Queensland, corneoscleral necrosis was the most commonly quoted complication[2]. Patients with scleral necrosis commonly had received postoperative beta-irradiation[1-3] or cytotoxic eye drops such as mitomycin-C[4]. They may also have associated scleral infection and endophthalmitis[1,3]. *Pseudomonas aeruginosa* is a common organism isolated in these cases although endophthalmitis due to rare pathogens such as *Aspergillus* have also been described[5].

CASE REPORT

A 70-year-old Chinese male was referred to our centre in June 1992 for a painful red left eye associated with purulent discharge and decreased visual acuity for a duration of three weeks. He had a pterygium excised from the nasal aspect of his left eye by an ophthalmologist 15 years previously. He did not receive any postoperative beta-irradiation but he was unsure if he received any cytotoxic eye drops. He was apparently well until six months prior to the referral when he experienced occasional redness and tearing of the left eye which were relieved with topical antibiotics. The right eye was asymptomatic. He did not have a history or any symptom suggestive of rheumatoid arthritis, collagen vascular disease or any other significant medical illness.

His best-corrected distance visual acuity was 6/9 in the right eye and 6/120 in the left eye. Ophthalmic examination revealed a large incomitant left exotropia with loss of adduction in the left eye. A large 12 mm by 12 mm area of scleral ulceration and necrosis was present at the site of the previous pterygium operation (Fig 1). The necrotic area was bare of conjunctiva. Part of the uveal tissue could be seen beneath the thin necrotic sclera indicating the potential of the eye developing endophthalmitis. The cornea was clear. 2 + flare, 3 + cells and a minute hypopyon were present in the anterior chamber. A small fibrin plug on the anterior lens surface was noted. Anterior vitreous cells 2+ were also present. Indirect ophthalmoscopy revealed a fairly clear vitreous and a normal retina. B scan of the left eye was normal. The right eye was normal. The patient was afebrile and systemic examination was unremarkable. Full blood count revealed a mild predominantly polymorphonuclear leucocytosis (Total white cells = 11,500/mm^3 and granulocytes = 77.5%).

Fig 1 – Exotropic left eye with a large area of scleral necrosis and infection at the site of the previous pterygium operation. The uveal tissue could be seen beneath the thin necrotic sclera.



A clinical diagnosis of left scleral necrosis and infection was made and the patient was immediately started on intravenous vancomycin 1g q12H, intravenous ceftazidime 1g q8H and topical cefazolin (50 mg/ml) and fortified gentamicin (14 mg/ml) hourly in the left eye.

The patient remained afebrile and improved clinically with the antibiotic regime. The left visual acuity improved to 6/24 on Day 2 of treatment. The minute hypopyon disappeared and the number of cells in the anterior chamber decreased. The infection was clinically controlled and the patient underwent a tectonic scleral patch grafting on Day 4.

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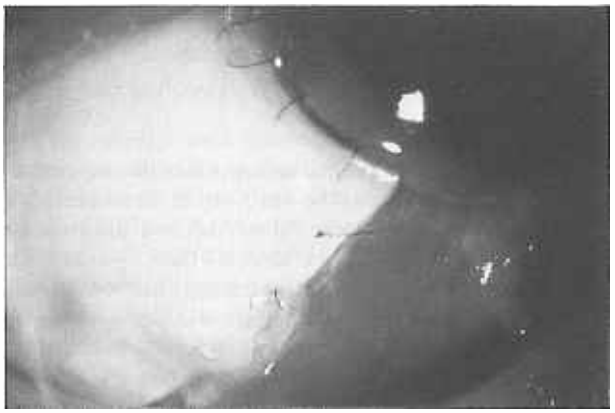
The findings during the operation were consistent with the preoperative assessment. In addition, the insertion of the medial rectus could not be found. A large 12 mm by 12 mm full-thickness donor scleral patch graft was fashioned and sutured with 10/0 nylon over the thin necrotic sclera (Fig 2). As the conjunctival defect was huge, the remaining conjunctiva was only able to cover the periphery of the graft. Subconjunctival injection of gentamicin 20 mg was given at the end of the operation.

Fig 2 – Large 12 by 12 mm donor scleral patch graft covering the necrotic sclera in left eye. Only the periphery of the graft could be covered by conjunctiva because of the large conjunctival defect.



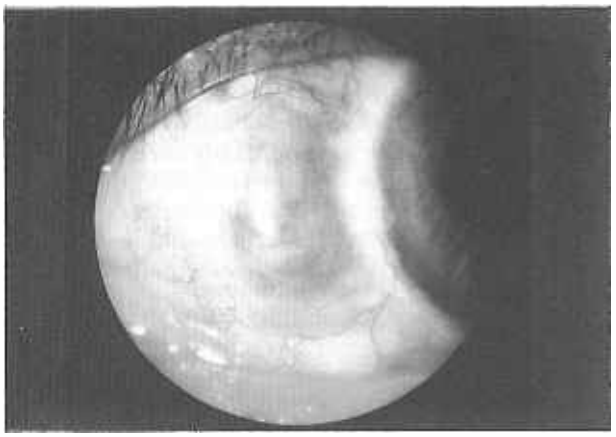
Postoperatively, the graft remained in place and the infection was clinically controlled. Lid closure was normal. A swab taken from the necrotic sclera before commencement of antibiotic therapy grew *Pseudomonas aeruginosa* which was sensitive to ceftazidime and gentamicin. The intravenous vancomycin was terminated on Day 5 when the culture and sensitivity result was known. The intravenous ceftazidime and the topical cefazolin and gentamicin were continued. On Day 8 (fourth postoperative day), the eye was noted to have a large inferior bullous retinal detachment which gradually progressed over the next few days to involve the entire retina. Classical shifting fluid sign suggestive of an exudative retinal detachment was noted. As there was uncertainty regarding whether this could be a rhegmatogenous retinal detachment, the case was reviewed by two consultants in the retinal service. Both agreed that the detachment was exudative. The left visual acuity decreased to only CF (counting figures) at close range. The intravenous ceftazidime was terminated on Day 10. The patient was discharged on Day 13 with topical cefazolin and gentamicin q2H.

During the subsequent follow-up visits, the topical cefazolin and gentamicin were replaced with topical chloramphenicol which was later gradually tapered off over the next four months. The scleral graft gradually shrunk in size and retracted posteriorly. A thin layer of translucent sclera covered by conjunctiva maintained the integrity of the globe anterior to the graft (Fig 3). The exudative retinal detachment resolved gradually as the scleral inflammation decreased and the retina was completely reattached four and a half months later. At the last follow-up visit 14 months after the operation, the globe had remained intact and the visual acuity had stabilised at CF 2 m.

DISCUSSION

We have progressed little in our understanding of the pathogenesis and therapy of pterygium despite having known

Fig 3 – The thin and translucent sclera maintaining the structural integrity of the globe 14 months after scleral patch grafting. The graft has shrunken in size and retracted posteriorly.



the disease for the last 3000 years[6]. Scleral necrosis and endophthalmitis following the treatment of pterygium are not uncommon[1,2] and eyes have been lost following such complications[1,3,5].

The scleral necrosis and infection in our patient is likely to be related to the pterygium excision at the same site 15 years previously although he did not receive any postoperative beta-irradiation and was unsure if he received any cytotoxic eye drops. He did not have any symptoms or signs suggestive of a systemic disorder that might have predisposed him to scleral necrosis and infection.

Scleral necrosis is a difficult clinical problem to manage. This problem is aggravated when superimposed infection with a virulent organism such as *Pseudomonas aeruginosa* is present. Endophthalmitis from direct spread of the organism through the sclera can cause the eye to be lost. Our patient had the potential of developing endophthalmitis and was close to losing his eye when he was first seen in our centre. The importance of aggressive management of infection and the value of prophylactic repair of scleral necrosis have been emphasised by Tarr and Constable[3]. We applied these principles in the management of our patient with good results and saved the eye from possible scleral perforation and endophthalmitis.

We treated our patient early and aggressively with an empirical regime of intravenous vancomycin and ceftazidime and topical cefazolin and gentamicin which covered a broad spectrum of possible bacterial pathogens. It quickly controlled the scleral infection clinically and prevented the eye from developing endophthalmitis. This allowed us to proceed with a tectonic grafting procedure. When the antibiotic sensitivity of the organism cultured was known, the antibiotic regime was fine-tuned by keeping the patient on the minimum "essential" antibiotics.

Interestingly, our patient developed an exudative retinal detachment during the course of his illness. This is not inconsistent with the clinical picture as exudative retinal detachment is known to occur in numerous subretinal disorders which damage the retinal pigment epithelium and thereby allowing fluid derived from the choroid to pass into the subretinal space. It is likely that the inflammation related to the scleral necrosis and infection damaged the retinal pigment epithelium in our patient.

The management of the exudative retinal detachment in our patient proved difficult. Corticosteroid therapy was not given because it alters host defence mechanisms[7] and may cause the

clinically controlled scleral infection to flare up. With successful treatment of the scleral infection and resolution of the inflammation, the exudative retinal detachment resolved completely after four and a half months, presumably because enough retinal pigment epithelial cells had recovered to remove the subretinal fluid and prevent its reaccumulation. Unfortunately, the visual acuity could only improve to CF 2 m after the macula has been detached for four and a half months.

CONCLUSION

Scleral necrosis and infection are serious late complications of pterygium treatment and are difficult to manage. Early aggressive antibiotic therapy may control the infection and prevent the development of endophthalmitis. In some cases, tectonic scleral patch grafting may be necessary to preserve the structural integrity of the globe.

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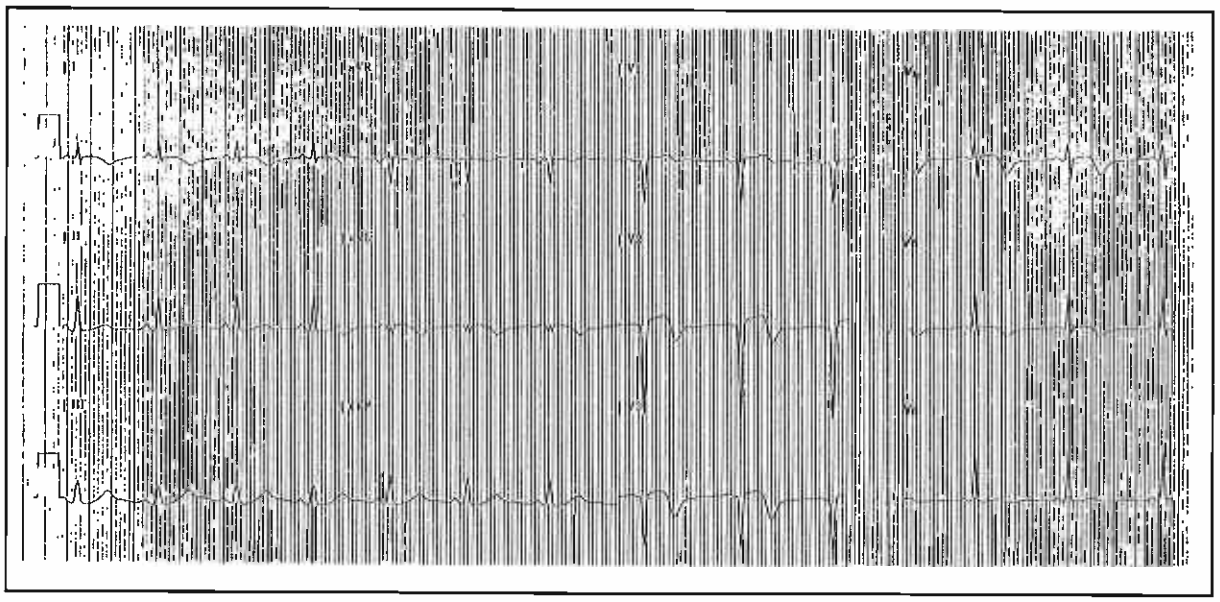
Fig 2 - Subsequent ECG



Answer to electrocardiographic case
Diagnosis: Hyperacute phase of anterior myocardial infarction.

DISCUSSION

Clinical-Electrocardiographic Diagnosis

The initial 12-lead ECG (Fig 1) was interpreted to indicate unstable angina and the patient was treated with intravenous Glycero-Trinitrate (GTN) and heparin infusion. Subsequent ECG (Fig 2) recorded serially over the next few days showed evolved acute anterior myocardial infarct, confirmed by elevated cardiac enzymes.

In retrospect, the history of acute, gripping retrosternal chest pain of 1 hour's duration in a patient with several coronary risk factors (Indian, family history of premature coronary artery disease and smoking) was very suggestive of an acute myocardial infarction (anginal pain should not exceed 30 minutes duration). The initial 12-lead ECG (Fig 1) showed tall and widened T waves in the anterior precordial leads, with depressed ST take-off, as well as ST depression in the inferior leads. The tall and widened T waves with depressed ST take-off has been described by Marriott[1] to indicate an unusual early stage of myocardial infarction[2]. The subsequent ECG (Fig 2) showed evolved anterior infarction. Thrombolytic therapy was not given because the diagnosis was made too late.

Although the combined sensitivity of the patient history and the initial ECG might be only 90% for the early diagnosis of acute myocardial infarction, this combination is still the best technique for early identification[3,4]. Cardiac enzymes become detectable about 6 hours after the onset of acute myocardial infarction, making early diagnosis by enzymes impossible. Echocardiography reliably identifies abnormalities of wall motion due to acute myocardial infarction but fails to distinguish from unstable angina.

Importance of serial ECGs recorded at close intervals

This case illustrates the importance of recording serial ECGs at close intervals. If acute myocardial infarction is suspected and thrombolytic therapy is contemplated (preferably within 6 hours of onset of chest pain), then serial ECGs should be recorded at 15 minutes' intervals so that an early diagnosis can be made. Often, the classic changes of coved ST-segment elevation in the hyperacute phase of myocardial infarction will become apparent. Serial ECGs recorded 8-hourly apart will exceed the ideal time window for initiating thrombolytic therapy.

Differential Diagnosis

The initial ECG could also indicate unstable angina or acute anterior non-Q myocardial infarction. However, the suggestive history and the evolution of the classic coved ST-segment elevation in serial ECGs would have enabled us to make the diagnosis. The tall precordial T waves in the initial ECG is unlikely to be a normal variant or due to hyperkalemia because of the loss of R waves and ST-segment depression in the anterior precordial leads.

Management – thrombolytic therapy?

Administration of intravenous thrombolytic therapy in acute myocardial infarction within 6 to 12 hours of onset is an established therapy that decreases myocardial infarct size and mortality. However, thrombolytic therapy has not been demonstrated to be effective in unstable angina and non-Q myocardial infarction[5-8]. When thrombolytic therapy is used to treat unstable angina with an incompletely obstructed culprit coronary artery, the procoagulant effects of lytic therapy begin to dominate; hence, the potential benefit is diminished while its risks are unabated[9,10].

In this case, the initial ECG could indicate unstable angina or non-Q myocardial infarction and does not satisfy the criteria of at least 1 mm ST-segment elevation in at least 2 contiguous leads for the initiation of thrombolysis. However, the suggestive history and recognition that this pattern can be an unusual early stage of myocardial infarct would lead us to record serial ECGs at 15 minutes' intervals; and if the serial ECGs were to evolve the classic changes of coved ST-segment elevation, we would initiate thrombolysis.

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