Scleroretinal necrosis after a subconjunctival injection of gentamicin in a patient with a surgically repaired episcleral retinal detachment

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PURPOSE. To describe a case of scleroretinal necrosis after a subconjunctival injection of gentamicin in a patient who had an episcleral retinal detachment that was surgically repaired.

METHODS. Case presentation.

RESULTS. Thinning of the sclera due to cryosurgery and the induced localized inflammatory response resulting from the surgical procedure, in addition to the effect of the sponge buckle itself, could have played an important role in accumulation and storage of gentamicin under and adjacent to the buckle after injection. The increasingly higher concentration of the drug under the buckle could have induced a greater penetration of gentamicin through the sclera, which could have been the cause of the scleral-chorio-retinal necrosis observed in this patient.

CONCLUSIONS. Attention must be given to avoid side effects from subconjunctival injection of gentamicin. (Eur J Ophthalmol 2004; 14: 575-7)

KEY WORDS. Subconjunctival injection of gentamicin, Scleral buckle, Scleroretinal necrosis

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INTRODUCTION

Aminoglycoside antibiotics have been widely used in ophthalmology in the treatment and prevention of bacterial infections. Various microscopic studies have reported the retinal toxicity of these drugs, observed especially after intravitreal injections (1, 2). The "safe" dosage of gentamicin still remains a question, even in light of the vast number of studies performed. Loewenstein et al (2) reported in their histopathologic study that retinal toxicity was observed when a dose of at least 400 µg of gentamicin sulphate was injected into the vitreous of rabbit eyes.

The role of aminoglycoside use in the prophylaxis of ocular infections has recently been re-evaluated, especially after the several reported cases involving retinal damage after subconjunctival injection of gentamicin alone (3). In-depth studies have yet to be carried out in order to determine whether it is better to administer the drugs injected subconjunctivally or in the sub-Tenon space, and to determine if inadvertent intraocular injections which penetrate the sclera are the cause of the observed toxicity.

Case report

A 63-year-old man underwent routine surgery to correct an episcleral retinal detachment. The refraction of the eye was +0.50 sphere and there was no evidence of a thin blue sclera. Superior temporal and inferior nasal detachment was caused by a primary horseshoe tear localized at 10 o'clock.

The surgery entailed a subretinal fluid drainage localized to the bullous detachment at 9 o'clock and a small amount of cryosurgery freezing was performed around the tear. A cerclage and a silicone sponge buckle was then applied, localized between 10 and 11 o'clock. Subretinal drainage was permitted through a perforation made with a 5.0 spatulated needle in the subretinal space which did not require suturing. This technique is often used in our ward in retinal detachment surgery because it is safe and self-sealing. At the end of the surgery, a washout using 1 to 2 ml of gentamicin (40 µg/ml) was subconjunctivally administered under the fornix-based conjunctival flap positioned at a fair distance from the sponge buckle.

A retinal reattachment was observed after the first postoperative day, and the visual acuity was 20/40. The patient was discharged and returned for a checkup 1 week later. The patient was again examined after 20 days, and the retina appeared attached. The patient came for another examination 55 days after the operation complaining of decreased visual acuity in the eye that had been operated. Fundus examination showed a retinal detachment with a D2 proliferative retinopathy, and a vitrectomy was scheduled. Scleral ectasia with a uvea prolapse and retinal necrosis located under and adjacent to the sponge buckle was observed during the peritomy.

DISCUSSION

Aminoglycoside antibiotics are normally subconjunctivally injected at the end of many surgical procedures. Several histopathologic studies have reported aminoglycoside toxicity after intravitreal doses of gentamicin, which causes retinal pigment epithelial photoreceptor-outer segment complex toxicity. Focal patches of retinal pigment epithelial necrosis and hyperplasia have been noted 1 week following an intravitreal injection of gentamicin (2). Light and electron microscopic observation have shown an accumulation of macrophages in the subretinal space and disorganization present in both the photoreceptor and the outer segments.

A case of retinal toxicity after inadvertent intraocular injection of 20 mg of tobramycin following a cataract extraction has been reported (4). Furthermore, retinal toxicity has also been seen in doses as low as 30 μ g when the injection was given intravitreally, with the needle bevel down, indicating that the method of injection could also play a role in causing the toxicity. A greater toxicity has been seen when the needle was directed toward the posterior pole, in comparison to when it was directed toward the anterior chamber (1). Some authors emphasize the importance of the needle tip location, direction, and the rate of injection during intraocular injection. When the tip was too close to the retina and when the injection was given too rapidly during the intravitreal injection, a focal defined area of retinal edema developed within 24 to 48 hours, which eventually gave rise to extensive changes in the pigment epithelium and in the retina (1).

The mechanism behind aminoglycoside toxicity after cataract extraction without inadvertent injection has been proposed to be possibly due to the drug entering via the corneal sclera wound by seepage, once the ballooned fornix-based flap came into contact with it (1). When no wound is present, drug diffusion across a biological tissue normally depends on many variables, such as molecular weight, solubility, lipophilicity, and passive binding to tissue.

Some authors have already emphasized the possible drug penetration and its toxicity through a thinned sclera, which is normally present in myopic eyes with scleral dehiscence, especially in the temporal posterior polar region and around the optic nerve (3). Thinned sclera can also be seen in eyes that have undergone multiple surgeries or in areas under the scleral buckle (5). The patient in our case study did not have any wounds where the aminoglycoside could have seeped through during the surgery. The injection of aminoglycoside was in fact administered with a blunt needle, without intravitreal penetration. The intrascleral mattress suture of the buckle and the cerclage were both performed without scleral perforation.

The scleral ectasia flap with the uvea prolapse and the retinal necrosis located both under and adjacent to the sponge buckle which was observed after the subconjunctival injection could have been caused by a combination of many factors.

The cryosurgery could have also caused a thinning of the sclera, inducing a localized inflammatory response as a result of the surgical procedure. The inflammatory response aimed at the sponge buckle itself could have induced an accumulation and storage of gentamicin located under and adjacent to the buckle, inducing a greater penetration of the gentamicin through the sclera, thus causing the scleral-chorioretinal necrosis.

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