and iris root was 1309 mm. Ciliary processes were absent in the normal region of ciliary body (Figure 1).

Megalophthalmos anterior with ciliary body dysplasia was diagnosed.

Comment

Our case of megalophthalmos anterior demonstrates that the enlargement of the iris-lens diaphragm and ciliary ring during eye development (keratodysgenesis and irido*ciliary*goniodysgenesis) can result in an insertion of ciliary processes on the posterior surface of peripheral iris.

Arcus lipoides, mosaic corneal dystrophy, pigment dispersion, cataract and lens dislocation are associated ocular anomalies.[6] To our knowledge, ciliary body dysplasia in megalophthalmos anterior has not yet been described.

In a case of buphthalmos due to infantile glaucoma, normal insertion with a prolongation of ciliary processes has been seen by ultrasound biomicroscopy.[7] However, ultrasound biomicroscopy examinations of ciliary body in megalophthalmos anterior have not yet been performed.

We emphasize that in cases with megalocornea, ultrasound biomicroscopy is a helpful, additional tool for examination of the ciliary body. Perhaps ciliary body dysplasia may be a further feature for distinguishing megalophthalmos anterior from buphthalmos.

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Sir,

Multilayered amniotic membrane transplantation for partial thickness scleral thinning following pterygium surgery

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Scleral thinning and necrosis is a serious complication of pterygium surgery. It is commonly seen after chemotherapy or irradiation to prevent recurrence.[1] Scleral patch graft[2] or lamellar patch graft with preserved corneosclera[3] is usually performed in cases of severe thinning to restore the normal ocular surface integrity. We report two cases of partial thickness scleral thinning (scleral dellen) with thinning of adjacent cornea in one case (corneoscleral dellen), both of which were treated with multilayered amniotic membrane transplantation, suggesting that this surgical procedure can be an alternative treatment in this clinical situation.

Case 1

A 30-year-old female presented to us on 16 October 1999, with complaints of pain and discomfort in the right eye of 1 month duration. She had undergone pterygium excision in that eye 1 month back and the referring physician had noted scleral thinning postoperatively. On examination, she had a visual acuity of 20/20 in the right eye and 20/30 in the left eye. Slit-lamp examination revealed superficial scarring involving the nasal cornea in the right eye. Adjacent sclera showed thinning (Figure 1a) and was avascular. Gentamicin sulphate 0.3% four times a day along with artificial tears were prescribed. The patient underwent multilayered amniotic membrane transplantation over the area of scleral thinning.

The procedure was performed under peribulbar anaesthesia. Four millilitres of 2% xylocaine with adrenaline and 3 ml of 0.5% bupivacaine with hyalase were injected. Preserved human amniotic membrane was used in the procedure. Human amniotic membrane was prepared and preserved by the

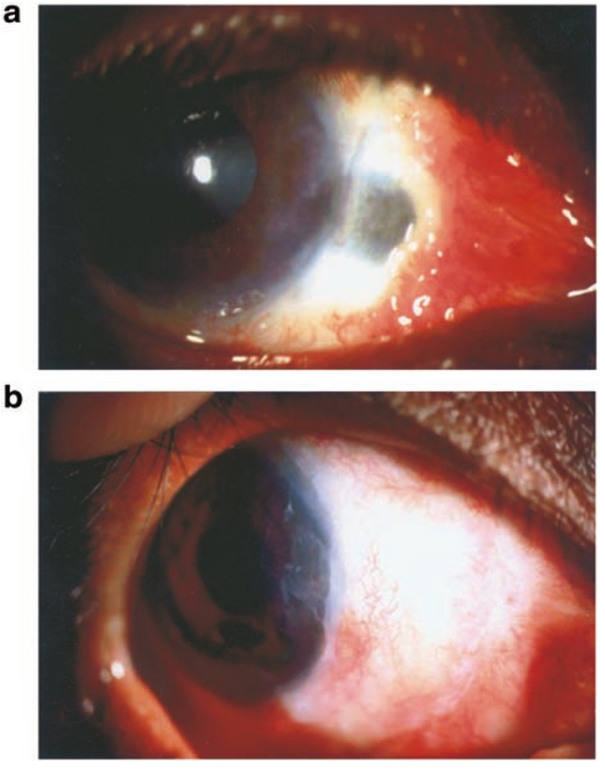

Figure 1 (a) Diffuse slit-lamp view of Case 1 showing the area of scleral thinning. (b) Diffuse slit-lamp view of the same following amniotic membrane transplantation, 6 weeks after surgery showing amniotic membrane covering the defect.

standard method.[4] The human placenta was obtained shortly after elective caesarian delivery. Serological tests were performed to exclude human immunodeficiency virus, hepatitis virus type B, hepatitis virus type C and syphilis. Under laminar air hood, the placenta was clear of blood clots with sterile Earle's balanced salt solution containing 50 μg/ml of penicillin, 50 μg/ml of streptomycin, 100 μg/ml of neomycin and 2.5 μg/ml of amphotericin B. The amnion was separated from the rest of the chorion by blunt dissection through the potential spaces between these two tissues and flattened onto a nitrocellulose paper with epithelial/basement membrane surface up. The paper with the adherent amniotic membrane was then cut into different sizes and was stored at -80°C in a 1:1 combination of Dulbecco modified Eagle's medium and glycerol.

The area of scleral thinning was defined. Three layers of preserved human amniotic membrane were applied over the area of thinning, and anchored to the episcleral tissue with interrupted 10–0 monofilament nylon sutures. Pieces of amniotic membrane were separated from the nitrocellulose paper and placed in the defect with no special care to put either the epithelial or stromal side up. Over this a layer of amniotic membrane with stromal side down was sutured to the adjacent conjunctiva with interrupted 10–0 monofilament nylon sutures. Postoperatively the patient was on topical 1% prednisolone acetate tapered over 3 weeks. This was initiated at three times a day along with 0.3% gentamicin sulphate eye drops 4 times a day for a week and artificial tears. Sutures were removed 2 weeks after surgery. At the last follow-up 6 weeks after surgery, the defect was well covered by the amniotic membrane which had epithelialised (Figure 1b).

Case 2

A 35-year-old female patient reported to us with a diagnosis of scleral and corneal thinning (corneoscleral dellen) following combined phacoemulsification and pterygium surgery in the left eye which was performed 5 days earlier. Antimitotic drugs were not used during surgery. There was a partial-thickness scleral thinning accompanied by thinning of the adjacent cornea. The surrounding sclera was pale (Figure 2a). Ciprofloxacin 0.3% eye drops six times a day along with artificial tears were prescribed. A

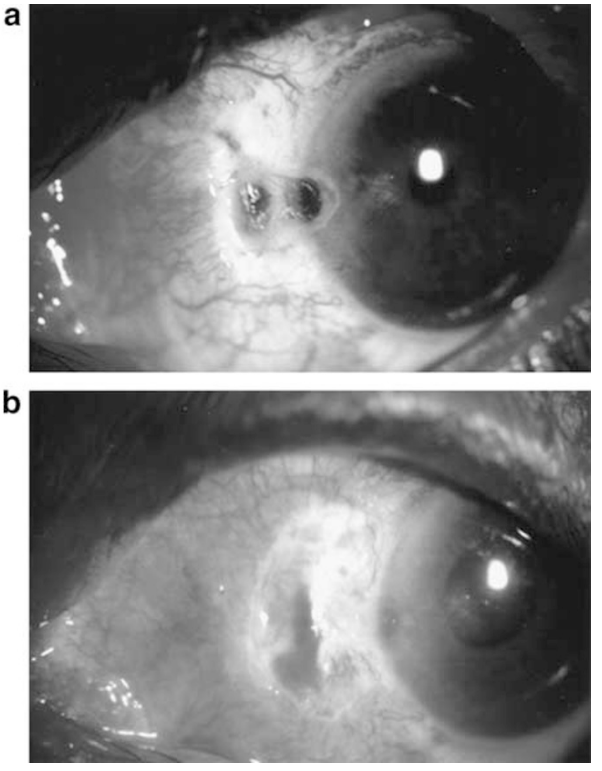

Figure 2 (a) Diffuse slit-lamp view (of Case 2) showing partial thickness scleral thinning with thinning of adjacent cornea. (b) Diffuse slit-lamp view of the same patient, 3 weeks after surgery showing amniotic membrane covering the area of scleral or corneal thinning.

multilayered amniotic membrane transplant was performed under peribulbar block. The residual tenon's tissue was excised from the scleral bed. The raw area of the sclera was covered with two layers of amniotic membrane filling the defect; another layer of amniotic membrane was applied with stromal side down and was sutured to the adjacent conjunctiva with interrupted 10–0 monofilament nylon sutures (Figure 2b). Postoperatively the patient was on 0.1% betamethasone sodium phosphate eye drops four times a day, 0.3% gentamicin sulphate eye drops four times a day and artificial tears six times a day. The sutures were removed after one week. Topical steroid medication was tapered and the patient was continued on artificial tears. At last follow-up, one month after surgery the eye was quiet and the surface had epithelialised.

Comments

Amniotic membrane, the innermost layer of placental or fetal membrane, consists of a thick basement membrane and an avascular stroma. The various observed effects of amniotic membrane after transplantation include rapid epithelialisation, return of normal epithelial phenotype,[4] reduced inflammation,[5] reduced vascularisation and reduced scarring. The amniotic membrane acts as a substrate for surface epithelialisation.

Kruse *et al*[5] reported the use of multilayered amniotic membrane transplantation for reconstruction of deep corneal ulcers.[5] They found this procedure useful in treating deep corneal ulcers and even descemetoceles. The stromal thickness was maintained even when the transplanted layer of amniotic membrane had gradually been dissolved. This study led us to surmise that multilayered amniotic membrane could allow surface reconstruction in partial-thickness scleral necrosis and corneal thinning as it is said that in a less severe case of corneoscleral thinning, even a conjunctival graft alone without lamellar corneal or corneoscleral or scleral reinforcement is sufficient to fulfil the requirement of normal healing.[6,7]

Chen *et al* reported amniotic membrane transplantation for severe neurotrophic corneal ulcers in 15 eyes.[8] More than one layer of amniotic membrane was applied in six of these eyes. Handa *et al* recently reported use of multilayered amniotic membrane transplantation for the treatment of deep ulceration of the cornea and sclera.[9] The authors performed multilayered amniotic membrane transplantation in severe ulcers to achieve the goals of collagen layer supplementation, basement membrane reconstruction, promotion of epithelialisation and wound healing. Two of these patients had scleral ulcers because of pterygium and foreign body. In these cases, the ulcer was filled with autotenon's capsule tissue. The second amniotic membrane was transplanted as a basement membrane (amniotic membrane graft). Amniotic membrane was placed in the ulcer with epithelial side up and secured with 10–0 nylon sutures. The third amniotic membrane was transplanted as a cover (amniotic membrane patch) with 10–0 nylon or 8–0 vicryl sutures. These cases epithelialised with conjunctiva, one case after 10 days and the other after 27 days. The sclera regained its original thickness.

In the two cases reported, the surgical details available did not suggest the use of antimetabolites during surgery or irradiation. The eyes were quiet and scleral defect was filled well with the amniotic membrane after surgery. The scleral thickness was regained. The surface had epithelialised well. We feel that in these cases of partial scleral thinning we achieved our objectives of filling up the scleral defect and adequate surface reconstruction. We propose this alternative technique as it is a simple procedure compared to scleral or corneoscleral patch grafts. Unfortunately, the surgical follow-up of the cases is too short to comment on the long-term usefulness of the procedure in this situation.

To conclude, multilayered amniotic membrane transplantation is a useful alternative choice to scleral or corneoscleral lamellar patch graft for scleral thinning following pterygium surgery. Further studies with long follow-up are warranted to evaluate the efficacy of the procedure in this clinical situation.

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Sir,

Acute posterior multifocal placoid pigment epitheliopathy with retinal vasculitis and papillitis

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Acute posterior multifocal placoid pigment epitheliopathy (APMPPE), defined by Gass in 1968,[1] is characterized by a rapid, but transient, loss of visual acuity associated with acute onset of multifocal discrete yellow-white placoid lesions at the level of the retinal pigment epithelium (RPE) in the posterior pole. Spontaneous resolution over several weeks to months occurs with pigment epithelial alterations but little change in the adjacent retina or choroids. In the acute phase, the fluorescein angiogram characteristically shows early hypofluorescence of the ophthalmoscopically visible lesions followed by late hyperfluorescence. The disorder was presumed to involve primarily the RPE,[1] whereas other investigators proposed that the underlying process in APMPPE is an inflammatory vasculitis affecting the choroidal vessels and that the RPE was secondarily affected.[2–6] This report describes a patient in whom the clinical and fluorescein angiography findings were typical of APMPPE but in whom retinal vasculitis and papillitis were additional features.

Case report

A previously healthy 35-year-old man was referred for ophthalmological examination because of a 7-day history of decreased vision in the right eye. The patient had no personal or family history of eye disease. His medical history was unremarkable and he took no medications. Results of systemic examination were unremarkable. His best-corrected visual acuity at initial examination was counting fingers at 2 feet in the right eye and 20/20 in the left eye. He could not discern any color test plates using the right eye and correctly identified 15 of 15 Ishihara color plates with the left eye. There was a 2+ relative afferent pupillary defect in the right eye. The ocular movements were normal. The anterior segment examination results were within normal limits. There were trace vitreous cells present in the right eye. Ophthalmoscopy disclosed a pigmentary lesion involving the fovea and multiple yellow-white placoid lesions in the right posterior pole, typical of APMPPE. The retinal arteries close to the optic nerve head were surrounded with yellowish infiltration. The optic disc was swollen and hyperemic with blurred margin and peripapillary flame-shaped retinal hemorrhages (Figure 1). No overlying subretinal fluid was seen. The left eye had one chorioretinal scar in the posterior pole and another one in the periphery. Fluorescein angiography of the right eye showed that the placoid lesions were hypofluorescent in the early phases (Figure 2) and hyperfluorescent in the late

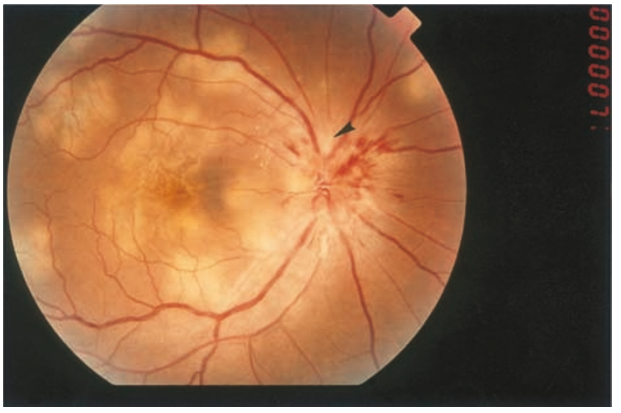

Figure 1 Acute fundus photograph of the right eye shows placoid lesions at the posterior pole, a central pigmentary lesion, vasculitis (arrowhead) and papillitis with peripapillary flame-shaped hemorrhages.

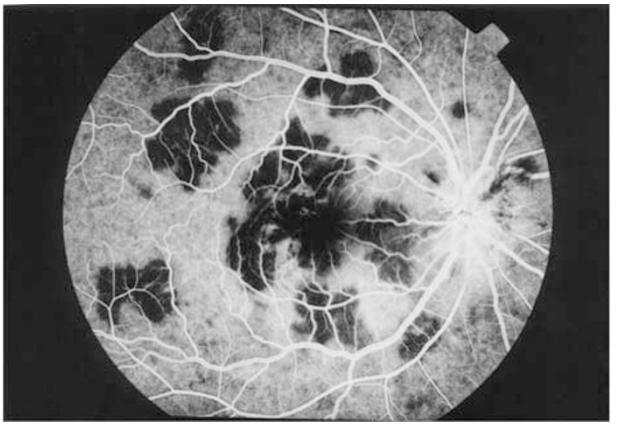

Figure 2 Early-phase fluorescein angiogram shows areas of hypofluorescence corresponding to the placoid lesions, and optic disc leakage.