

epithelial defects are strongly associated with the risk of developing DLK and are considered to be accountable for a considerable increase in its incidence.^{1,3,5,6} It is hypothesized that an epithelial defect in late DLK facilitates epithelial–stromal interaction, resulting in activation of keratocytes within the cornea, specifically in the interface.⁵ In this case, an epithelial defect was observed at initial presentation and was thus considered the main contributing factor for the development of DLK. Early aggressive treatment of DLK with topical steroids often results in good visual outcomes and minimal sequelae.^{1,4} DLK can also result in regular or irregular astigmatism and hyperopic shift,⁴ and changes in refraction in this case could have probably resulted from emmetropization. The importance of long-term follow-up of these patients should be taken into consideration, as late postoperative LASIK complications can be resolved adequately if diagnosed in a timely fashion.

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REFERENCES

1. Jin GJC, Lyle WA. Late-onset idiopathic diffuse lamellar keratitis after laser in situ keratomileusis. *J Cataract Refract Surg.* 2005;31:435-7.
2. Iovieno A, Amiran MD. Diffuse lamellar keratitis 8 years after LASIK caused by corneal epithelial defect. *J Cataract Refract Surg.* 2011;37:418-9.
3. Kamiya K, Ikeda T, Aizawa D. A case of late-onset diffuse lamellar keratitis 12 years after laser in situ keratomileusis. *Jpn J Ophthalmol.* 2010;54:163-75.
4. Randleman JB, Shah RD. LASIK interface complications: etiology, management, and outcomes. *J Refract Surg.* 2012;28:575-86.
5. Moilanen JAO. Keratocyte activation and inflammation in diffuse lamellar keratitis after formation of an epithelial defect. *J Cataract Refract Surg.* 2004;30:341-9.
6. Toda I. LASIK and the ocular surface. *Cornea.* 2008;27:S70-6.
7. Gritz DC. LASIK interface keratitis: epidemiology, diagnosis and care. *Curr Opin Ophthalmol.* 2011;22:251-5.

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Corneal keloid with cystoid cicatrix: post–small-incision cataract surgery



Keloids, although rarely seen in common clinical practice, may cause significant ocular morbidity. A 68-year-old female presented with a whitish mass in her left eye, associated with diminution of vision, foreign body sensation, and watering. This was preceded by a history of small incision cataract surgery (SICS). She underwent excision of the lesion, with a scleral patch graft and conjunctival autograft. The mass was confirmed to be a keloid on histopathologic examination. Here, we report this unique case of post-SICS corneal keloid, which, to our knowledge, has not been reported previously.

Keloids are benign proliferations of fibrovascular tissue. Keloids involving the cornea, although rare,¹ have been reported in a wide range of age groups, especially in the first 2 decades of life. These are bulky lesions commonly seen secondary to trauma and surgery.² However, no cases of corneal keloids following SICS have been reported so far. Here, we discuss a case of corneal keloid occurring after SICS in an adult patient.

CASE REPORT

A 68-year-old female presented to the outpatient department of our centre with a history of a whitish lesion in her left eye, initially noticed 1 year previously and

which had been gradually increasing in size over the past 3 to 4 months. It was associated with diminution of vision, foreign body sensation, and watery discharge. There was no blood-stained discharge. The patient had a history of SICS 4 years previously in the left eye and 2 years previously in the right eye. There was no history of ocular trauma.

On evaluation, the patient had a best corrected visual acuity of 6/6 OD and 1/60 OS, which improved to 6/12 after pupillary dilatation. There was no limitation of extraocular movement, and eyelid closure was complete. Slit-lamp biomicroscopy OS revealed a white-coloured elevated lesion saddling the superior limbus and measuring 7 mm vertically and 11 mm horizontally in its greatest dimensions. Inspection of the lesion revealed a gelatinous appearance with an irregular surface. There were no overlying vessels. On palpation of the lesion, it was soft to firm in consistency, with no tenderness. Along the superior border of the lesion, prolapsed uveal tissue could be seen through thin translucent cicatricial tissue that was encapsulating it. Examination findings suggested the presence of a superior SICS wound. The underlying cornea appeared normal, and the anterior chamber was regular with an updrawn pupil. The patient had pseudophakia with a posterior chamber intraocular lens in the sulcus. Intraocular pressure was 14 mm Hg OS. Indirect ophthalmoscopy revealed a normal fundus. The right eye was unremarkable on examination.

Keratometry values could not be captured OS. An anterior segment optical coherence tomography revealed corneal thickness of 520 microns OD and 610 microns OS, with the maximum height of the overlying lesion being 1.83 mm. The lesion did not appear to involve the corneal stroma, and there was no angle involvement, which was confirmed on ultrasonographic biomicroscopy (Fig. 1). Superiorly, an ectatic cicatrix with uveal tissue incarceration was noted. Based on clinical features and examination, we made a provisional diagnosis of OD pseudophakia, OS pseudophakia with corneal keloid, and ectatic cicatrix.

Surgical intervention was planned in light of the increasing size of the lesion and disturbance of visual functions and for confirmation of the diagnosis. The patient underwent excision of the lesion with a scleral patch graft and fibrin glue–assisted conjunctival autograft. Intraoperatively, the whitish lesion could be easily peeled off from the surface of the cornea, and the underlying cornea was found to be clear and intact. The ectatic cicatrix was then excised. Postoperatively, the patient was maintained on topical antibiotic drops, steroids, and tear supplements.

On detailed histopathologic examination, the first specimen features were suggestive of a keloid (see Fig. 1).

The second specimen revealed focally thinned out epithelium with vascularized stroma, fibrosis, and focal lymph mononuclear cell infiltrate. The posterior part showed the presence of uveal pigment laden cells, confirmed as an associated ectatic cicatrix.

RESULTS

On postoperative day 1, the patient had a best-corrected visual acuity of 6/36 OS with a clear cornea and the scleral patch graft well apposed (Fig. 2). The patient's vision subsequently improved to 6/12, with disappearance of the anterior chamber air bubble at day 7. The patient was kept on regular follow-up, which was noted to be uneventful.

Discussion

Corneal keloid, which is a rare condition, was first reported in the year 1865.¹ Keloids have been broadly categorized as fibrous tumours of the cornea. Initially described to be undifferentiated hyperplasia of corneoscleral tissue, they were thought to have a developmental origin.¹ Smith³ attributed these lesions to inflammation, trauma, or surgery. Corneal keloids were also thought to arise from the stromal cells of the iris as a result of their common association in cases with post-trauma uveal tissue prolapse. O'Grady et al.⁴ suggested

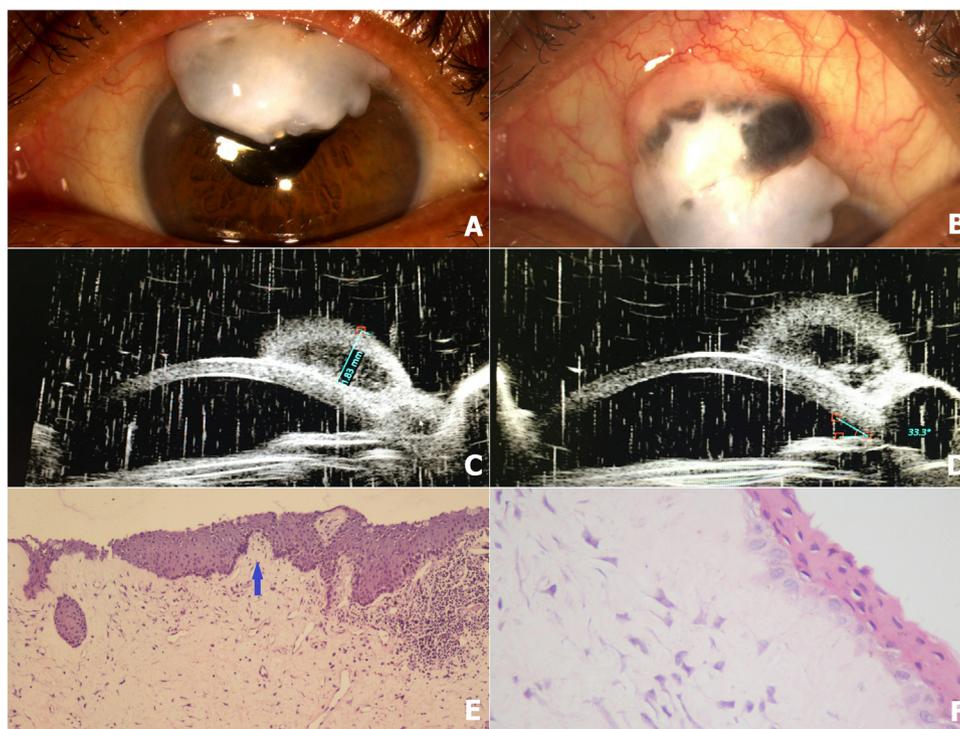


Fig. 1—A and B, (top right and left) Slit-lamp biomicroscopy reveals a whitish lesion straddling the limbus and having a glistening surface with a superior pigmented lesion, suggestive of an ectatic cicatrix. C and D, (middle right and left) Ultrasonographic biomicroscopy image suggesting spared underlying corneal stroma with a superior ectatic tissue along with uveal incarceration. E, Histopathologic examination (right bottom) low-power view, with subconjunctival tissue showing fibrosis and vascularization. The overlying epithelium shows focal hypoplasia and edema (arrow). The stroma showed prominent fibroblasts oriented in all directions anteriorly and posteriorly the stroma was less cellular (hematoxylin and eosin [H&E]; magnification $\times 100$). F, (left bottom) Higher magnification shows plump fibroblasts within the stroma. (H&E; magnification $\times 400$).

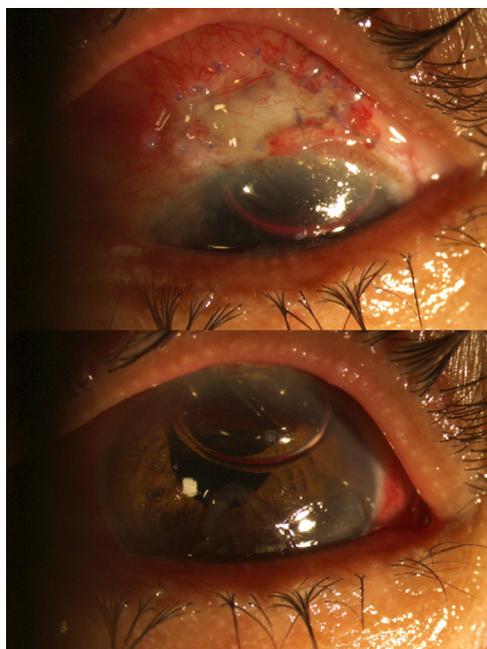


Fig. 2—A and B, Postoperative day 1, clinical image with slit-lamp biomicroscopy under diffuse illumination showing a well-attached scleral patch graft with overlying conjunctival autograft. The cornea appears clear, with an air bubble in anterior chamber.

overgrowth of stromal collagen during the reparative phase as a cause of keloid formation.

Keloids can be congenital, primary, or secondary, depending on the etiology. Secondary keloids have been seen following a number of surgical procedures, such as dermoid excision,⁵ cataract surgery in post-trauma cases,⁶ removal of pterygium,⁷ and so on. Keloids are mostly solitary or multiple nodular lesions, seen usually adjacent to scarred areas. In some cases, underlying cornea is clear, and in some others, deep stromal involvement, with or without destruction of the angle, is seen. In this case, the patient had a history of cataract surgery, and examination findings revealed a smooth, glistening white mass over the cornea; all of this suggested corneal keloid as the most likely clinical diagnosis. Histopathologic examination helped confirm the diagnosis. The histopathologic features of a keloid also depend on the stage.⁶ In the early stages, fibroblasts predominate, with formation of collagen type III and new blood vessels. In the late hyaline stages, collagen fibres become compact, with paucity of fibroblasts and involution of vessels.

Treatment should be considered in cases in which the lesion is increasing in size and the patient is symptomatic. Surgical options include superficial keratectomy, lamellar or penetrating keratoplasty, and sclerokeratoplasty.^{2,8} Recurrences of corneal keloids, although rare, have been reported. Recurrences can be prevented by inhibiting fibroblast-mediated collagen synthesis.⁸ The roles of

steroids, mast cell stabilizers,^{9,10} cyclosporine,¹¹ and physical therapy, such as cryotherapy and laser therapy, have been evaluated.¹⁰

CONCLUSIONS

Corneal keloids, although not commonly seen in routine clinical practice, are important differentials for nodular lesions of the ocular surface; when managed in a timely manner, they have acceptable anatomical and functional outcomes.

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REFERENCES

1. Duke-Elder. *System of Ophthalmology VIII Part II 1986-88*. London, UK: Henry Kimpton; 1995.
2. Vanathi M, Panda A, Kai S, Sen S. Corneal keloid. *Ocul Surf*. 2008;6:186-97.
3. Smith H. Keloid of the cornea. *Transact Am Ophthalmol Soc*. 1940;38:519-38.
4. O'Grady RB, Kirk HQ. Corneal keloids. *Am J Ophthalmol*. 1972;73:206-13.
5. Gaviria JG, Johnson DA, Scribbick F. 3rd. Corneal keloid mimicking a recurrent limbal dermoid. *J Pediatr Ophthalmol Strabismus*. 2005;42:189-90.
6. Bourcier T, Baudrimont M, Boutboul S, Thomas F, Borderie V, Laroche L. Corneal keloid: clinical, ultrasonographic, and ultrastructural characteristics. *J Cataract Refract Surg*. 2004;30:921-4.
7. Park CY, Ji YH, Chung ES. Bilateral pterygium and corneal keloid in a 9-month-old child. *J Korean Ophthalmol Soc*. 2003;44:2171-7.
8. Chawla B, Agarwal A, Kashyap S, et al. Diagnosis and management of corneal keloid. *Clin Exper Ophthalmol*. 2007;35:855-7.
9. Cibis GW, Tripathi RC, Tripathi BJ, et al. Corneal keloid in Lowe's syndrome. *Arch Ophthalmol*. 1982;100:1795-9.
10. McElvanney AM, Adhikary HP. Corneal keloid: aetiology and management in Lowe's syndrome. *Eye*. 1995;9:375-6.
11. Esquenazi S, Eustis HS, Bazan HE, et al. Corneal keloid in Lowe syndrome. *J Pediatr Ophthalmol Strabismus*. 2005;42:308-10.

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