have the velocity or trajectory to injure the eye, whereas when firing above eye level, the face lies in the path of the wire collation pieces. Even with eye protection, the potential risk is higher because of the downward path of these small projectiles.

We believe that penetrating ocular injuries from nail-gun wire is a previously unreported risk of operating nail guns by improper technique or without protective eyewear. With models being designed for home use by nonprofessionals, the need to educate users about these risks and appropriate safety precautions is greater than ever.

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Avellino Dystrophy in a Patient After Laser-Assisted In Situ Keratomileusis Surgery Manifesting as Granular Dystrophy

Avellino dystrophy is an autosomal dominant corneal stromal disease that shares features of both granular and lattice corneal dystrophies.2 Molecular genetic techniques have shown that granular, lattice, and Avellino dystrophies share the same genetic locus and map to chromosome 5q. Distinct mutations in the BIGH3 gene cause the various 5q31-linked corneal dystrophies.2 These mutations are R555W in granular dystrophy, R124C in lattice type 1, and R124H in Avellino dystrophy.

We report a case of progressive corneal Avellino dystrophy following laser-assisted in situ keratomileusis (LASIK) surgery manifesting clinically as granular dystrophy. This case highlights the importance of combining molecular testing with clinical and histopathological phenotypes.

Report of a Case. The patient, a 53-year-old white man, complained of a bilateral decrease in vision over 7 years. He described the visual loss as affecting his right eye more than his left eye. There were no other symptoms, known diseases, or known allergies. Nine years ago, the patient underwent a LASIK procedure bilaterally at another institution. At the time of surgery, the patient was informed that he had a “corneal disease” that would not affect the surgical procedure. The patient is not aware of any family history of eye disease.

On examination, best-corrected visual acuity was 20/300 OD and 20/200 OS. Slitlamp examination revealed multiple, crumb-like opacities in the corneal stroma and diffuse, central corneal stromal haze in the right eye (Figure 1). The patient demonstrated similar findings in the left eye. The patient did not have corneal epithelial defects or corneal edema bilaterally. Dilated fundus examination results were normal.

The patient underwent penetrating keratoplasty in the right eye indicated by decreased vision secondary to deposits in the midstromal layer of the cornea. The patient’s corneal button was sent for pathological analysis. Postoperative visual acuity at 9 months measured 20/40 OD with Snellen acuity.

Histopathological examination of the corneal button revealed a linear band of eosinophilic deposits along the LASIK flap interface (Figure 2). These deposits stain red with Masson trichrome stain (Figure 3). Congo red staining did not reveal any presence of amyloid (Figure 4). The specimen also demonstrated an area of epithelial tissue peripherally in the corneal stromal along the LASIK flap interface (Figure 5).

Subsequently, the patient’s blood was drawn and sent to Duke University Medical Center for genotype analysis. The patient’s TGFBI (BIGH3) gene was completely sequenced, which revealed an R124H gene mutation consistent with Avellino dystrophy. There is no known family history of corneal dystrophy.

Figure 1. Preoperative slitlamp photograph of the cornea.
Comment. Traditionally, corneal stromal dystrophies were diagnosed and classified on the basis of clinical and histopathologic findings. Avellino dystrophy has a wide variety of phenotypes. The earliest manifestation is usually superficial granular deposits in the stroma. Over time, patients develop latticelike lesions deeper in the stroma. Lattice lesions are often small and difficult to identify histologically and in some cases may be absent. As a result, it is often misdiagnosed as granular dystrophy. The discovery of various mutations in the BIGH3 gene that are associated with different 5q31-linked corneal dystrophies provides a tool for more accurate diagnosis. In this case, had we relied on the slitlamp appearance and histologic staining pattern, we would have misdiagnosed the patient’s condition as granular dystrophy. Identification of the R124H mutation in this patient provided the correct diagnosis of Avellino dystrophy.

The development of granular deposits along the LASIK flap interface in the laser ablation zone in post-LASIK patients with Avellino dystrophy has been previously reported. Herein, we show the histologic features of such an exacerbation. Dense hyaline deposits are identified as a linear band in the stroma anteriorly, corresponding to the LASIK flap interface. The presence of epithelium at the same level as the band of hyaline deposits confirms this region as the flap interface. In this case, amyloid was absent.

Mutations in the BIGH3 gene result in abnormal keratoepithelin, a 68-kD adhesion molecule. In 5q31-linked corneal dystrophies, studies suggest that corneal keratocytes and/or epithelial cells produce the abnormal keratoepithelin, which interacts with proteoglycans, keratin, and other extracellular proteins leading to various stromal deposits. Additionally, keratoepithelin is secreted by keratocytes after trauma. The accumulation of deposits at the LASIK flap interface in the laser ablation zone is thought to be the result of keratocyte stimulation from the microkeratome and laser ablation or the formation of a potential space at the flap-stromal interface as a result of the surgery. The reason that hyaline deposits appear to preferentially accumulate is unclear since the mechanism of formation of hyaline material vs amyloid in Avellino dystrophy from the R124H-mutated BIGH3 gene product is not known.

This case supports the diagnosis of Avellino dystrophy as a contra-
indication for LASIK surgery because of the increased deposition of granular material and a decrease in best-corrected visual acuity. Additionally, this case stresses the importance of a combined molecular/phenotype classification of corneal dystrophies.


Minocycline for the Treatment of Ocular and Ocular Adnexal Sarcoidosis

Sarcoidosis is a chronic systemic inflammatory disorder characterized by noncaseating granulomas. Although almost any organ can be affected, common sites of involvement include the skin, lungs, lymph node, eye, and ocular adnexa. Corticosteroids have remained the first-line therapy since their introduction in the 1950s. However, because of the significant adverse effects associated with chronic corticosteroid use, treatment is typically reserved for those with severe visceral involvement or refractory ocular and ocular adnexal involvement.

A number of steroid-sparing agents have been used with varying success in the treatment of chronic sarcoidosis. Bachelez et al reported clinical response in 10 of 12 patients with cutaneous sarcoidosis treated with minocycline. We describe herein the first reported case of ocular, ocular adnexal, and systemic sarcoidosis treated with minocycline.