Ocular Trauma From Nail Gun Cartridge Wire

Nail guns are potentially lethal tools1,2 that fire nails in rapid succession at high velocity. The nail cartridges are held together with wire, plastic, glue, tape, or paper. When wire is used, the 2 carrier wires (Figure 1) are called the wire collation. As the nail is fired, these 2 wires are sheared off, leaving 2 barb-like pieces of wire (<6 mm) attached to the nail. The 2 pieces are then sheared off the nail as it penetrates the surface.

Ocular nail-gun injuries reported so far describe nails as the cause of injury.3,4 To our knowledge, there have been no reported ocular injuries from the wire collation. We believe that flying pieces of wire collation from nail guns represent a previously unreported risk of ocular injury. In this article, we report 3 consecutive patients with penetrating ocular injuries from short pieces of wire ejected while using nail guns.

Report of Cases. Case 1. A 45-year-old man was using a nail gun to secure siding boards onto a house when a piece of wire collation penetrated his right eye. Eye protection was not being used. The patient reported immediate pain and decreased vision. Visual acuity was 20/800 OD and 20/20 OS. Slitlamp examination of the right eye showed a metal wire perforating the cornea, with one end embedded in the lens and the other protruding about 2 mm from the cornea. A traumatic cataract was present. The corneal wound was Seidel negative. The fundus could not be visualized. Computed tomography showed a 6-mm, linear, metallic foreign body penetrating the right globe and lodged in the anterior chamber (Figure 2).

The patient underwent removal of the foreign body and repair of the corneal laceration, followed 1 week later by cataract surgery with posterior chamber intraocular lens implantation. One week postoperatively, visual acuity was 6 ft/200 OD, improving with a pinhole to 20/200. Slitlamp examination showed a sealed corneal wound with corneal edema, 2 to 30 cells per high-power field in the anterior chamber, and a well-centered posterior chamber intraocular lens with intact posterior capsule. The patient was subsequently lost to follow-up.

Case 2. A 36-year-old man was nailing plywood above his head with a nail gun when he felt sharp pain in the right eye. He had lowered his protective eyewear because of fogging. The patient saw a piece of wire...
protruding from the cornea and removed it (using a needle-nose pliers) prior to seeking care.

Visual acuity was 20/30 OD and 20/20 OS. Slitlamp examination of the right eye showed an approximately 1.5-mm paracentral full-thickness corneal wound that was Seidel positive. The anterior chamber was formed with 3 to 40 cells per high-power field and flare. There was an iris transillumination defect underlying the corneal wound. The lens was clear and the anterior capsule was intact. Intraocular pressure was 11 mm Hg OD and 15 mm Hg OS. Dilated fundus examination was normal.

The corneal wound was glued and a bandage contact lens placed on the cornea. On the fourth day, the patient was diagnosed with endophthalmitis and underwent treatment with anterior chamber and vitreous tap and intravitreal injection of antibiotics. *Streptococcus pneumoniae* and viridans streptococci were isolated from the vitreous tap. This was followed by a pars plana vitrectomy, lensectomy, membrane peel, and intraocular antibiotic injection. At the 9-month follow-up, the best-corrected visual acuity was 20/25 OD.

**Case 3.** A 26-year-old man was using a nail gun to secure siding boards onto a house when a piece of wire collation penetrated his left eye. His protective eyewear had “slipped down” while he was nailing overhead. The patient experienced pain and decreased vision.

Visual acuity was 20/20 OD and 20/100 OS. Slitlamp examination of the left eye showed a full-thickness paracentral corneal wound with a piece of metal wire piercing the lens with one end in the anterior chamber and the other in the anterior vitreous (**Figure 3**). An orbit radiograph confirmed a 5-mm metal foreign body overlying the globe (**Figure 4**). The patient underwent a pars plana vitrectomy, lensectomy, intraocular foreign body removal, and corneal wound repair. This was followed by a secondary intraocular lens placement. At the 5-month follow-up, best-corrected visual acuity was 20/20 OS.

**Comment.** Three patients with full-thickness corneal wounds and intraocular foreign bodies resulting from nail-gun wire were seen in our department within the last year. None had effective eye protection at the time of injury, and 2 were nailing above eye level.

The size of the wire collation (about 1 mm X 6 mm) and its sharp end, caused by shearing from the main coil, possibly increase the likelihood that it will penetrate the eye and be retained, should it impact with sufficient velocity. Therefore, it is important to rule out an intraocular foreign body. A fine-cut computed tomographic scan may be helpful in evaluating the location of the wire within the globe. On the other hand, because the wire is relatively thin, the intraocular tissue damage at entry is limited, resulting in potentially good postinjury vision.

The position and angle at which the nails were fired may have contributed to the injuries. When firing straight ahead or downward, the sheared-off wire collation may not
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 eye wear. 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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Author Contributions: Dr Bhandari had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Financial Disclosure: None reported.


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. 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. 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. The specimen also demonstrated an area of epithelial tissue peripherally in the corneal stromal along the LASIK flap interface (Figure 4).

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.