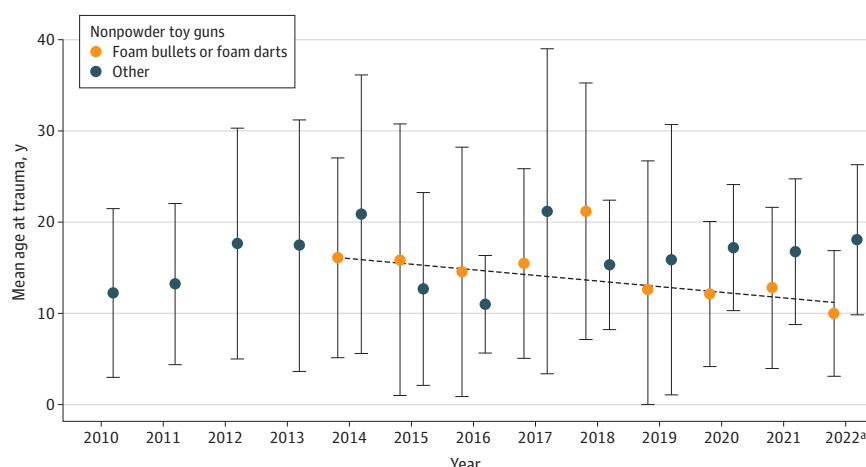


Figure 2. Annual Mean Age at Ocular Trauma Associated With Nonpowder Toy Guns



Simple linear regression of mean age at trauma for toy guns using foam bullets or foam darts is shown (dashed line) (slope, 0.57; 95% CI, -0.95 to 0.18;  $P = .01$  for deviation from 0). Error bars represent SDs.

<sup>a</sup> Reported from January through June.

volving foam bullets or foam darts or whether the use of this term was associated with this particular brand. Nevertheless, there has been no evidence that one brand would be less sight-threatening than others either in the peer-reviewed ophthalmic literature or in our experience. Recommendations might prevent many of these unintentional injuries, such as consumer attention to age labeling and consideration of protective goggles, while manufacturers could promote use of safety glasses to protect children's eyes.

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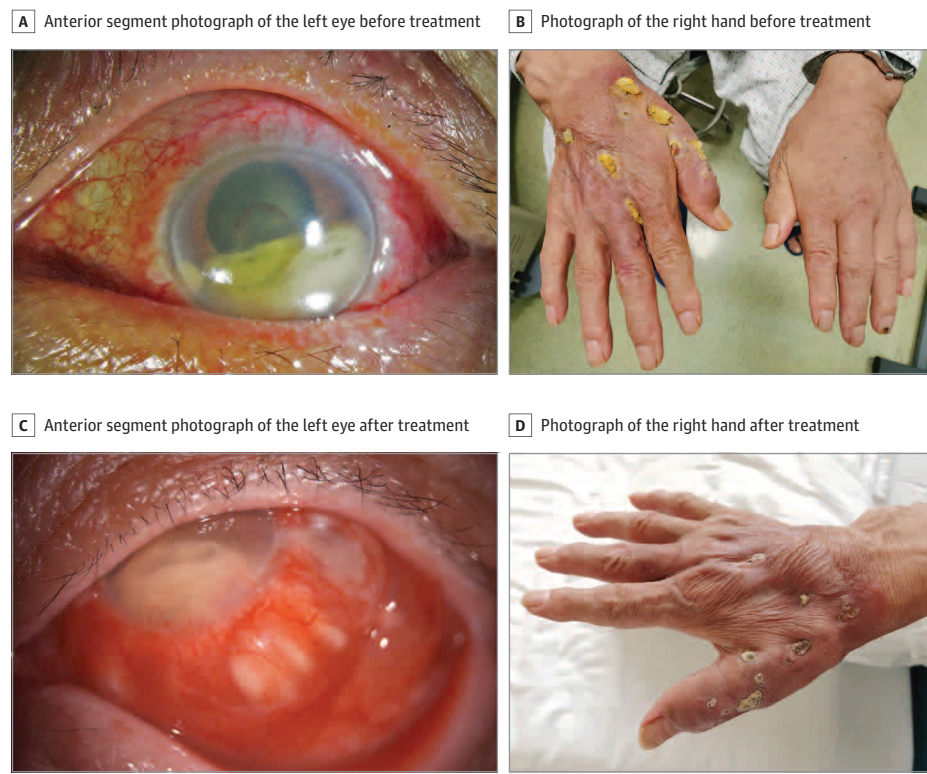
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## OBSERVATION

### Endophthalmitis Secondary to Fish Tank Granuloma

*Mycobacterium marinum* is identified as a slowly growing nontuberculous mycobacteria<sup>1</sup> and is common in atypical mycobacteria that cause opportunistic infection in humans.<sup>2</sup> *M marinum* leads to infection when damaged skin is exposed to contaminated fish, fish tanks, or swimming pools, which is also known as fish tank granuloma or swimming pool granuloma. Infections are usually limited to the skin and soft tissues of body extremities. Herein, we describe an uncommon case of endophthalmitis secondary to fish tank granuloma.

**Report of a Case |** A 68-year-old man presented with progressive painless red eye and blurred vision for 2 months. The patient had a 20-year history of type 2 diabetes and a 10-month history of tenosynovitis in his right hand after injury while handling fish, which was refractory to antibiotic treatment. Visual acuity was 20/20 OD and counting fingers OS. Conjunctival hyperemia, large yellowish keratic precipitates, 2+ cells,

Figure. Endophthalmitis and Nodular Lesions in the Hand Infected With *Mycobacterium marinum*

A, Conjunctival hyperemia and hypopyon in the left eye. B, Nodular lesions in the dorsum of the right hand. C, Subconjunctival purulent nodules, corneal edema, and hypopyon after treatment in the left eye. D, Subsided nodular lesions of the right hand after treatment.

3+ flare, and 5 mm of hypopyon in the anterior chamber were noted in his left eye (Figure, A). Anterior scleritis was seen in the temporal and inferior areas. Through 1+ vitreous haze, no abnormal changes were found in the fundus. His right eye was normal. Multiple crusted and ulcerated nodules, forming a linear distribution, could be seen in his right hand (Figure, B). Results of laboratory tests were unremarkable.

Bacterial endophthalmitis of his left eye was considered. Intravitreal injection of 1 mg of vancomycin was performed. Results of smear and culture of aqueous humor sampled before injection were both negative. After treatment, the anterior chamber reaction continued to worsen. The aqueous humor was resampled for next-generation sequencing analysis. Sixteen sequence readings of *M marinum* were detected, accounting for 0.0151% of the genome coverage. Polymerase chain reaction was not performed.

Clarithromycin, rifampicin, and ethambutol were administered orally. Rifampicin eye drops were added. Corticosteroid was used topically but not systemically. Skin lesions subsided gradually (Figure, C). However, hypopyon increased and vitreous opacity worsened in the left eye. Visual acuity dropped to light perception OS. Vitrectomy was performed. Necrotic scleritis and scleral perforation near the corneal limbus was found during the surgery. No focal lesion or mass was found in the retina and the choroid, except for small retinal hemorrhages. Vitreous culture grew mycobacterium, while findings of acid-fast staining of smear were still negative. Meanwhile, pathology of skin biopsy of the right hand showed caseous necrosis. Approximately 1 month after treatment,

scleral purulent nodules, severe corneal edema, and hypopyon were noted in his left eye (Figure, D), with no light perception OS. Evisceration was finally performed.

**Discussion** | *M marinum* optimally grows at 30 °C and hardly grows at 37 °C,<sup>1</sup> which has made it difficult to be identified in ordinary culture. The vitreous sample was cultured in Lowenstein-Jensen medium at 35 °C, and then mycobacterium grew. Meanwhile, the temperature of the ocular anterior segment is close to 30 °C.<sup>3</sup> This may partially explain the more severe inflammation in the anterior segment. Due to above reasons, the ocular infection was speculated to be exogenous. The scleral nodules might be the portal of entry.

The intraocular infection was refractory to combined antibiotic therapy, which is similar to that caused by other nontuberculous mycobacteria.<sup>4</sup> To our knowledge, only 2 cases of ocular infection caused by *M marinum* have been reported.<sup>5,6</sup> In the first case of sclerokeratitis, combination of rifampin, minocycline, and ethambutol controlled the inflammation after penetrating keratoplasty. In the second case of keratitis, 4-week combined treatment of trimethoprim, sulfamethoxazole, and ciprofloxacin failed to resolve the disease, and a corneoscleral graft was transplanted. The prognosis of intraocular infection caused by nontuberculous mycobacteria is poor. Corticosteroids are not recommended because of their association with a long-term course and vision decline.<sup>4</sup> Loss of eye is inevitable in nearly one-third of cases.<sup>4</sup>

In conclusion, we present a patient with endophthalmitis caused by *M marinum*. *M marinum* should be considered in

cases of nodular scleritis or uveitis. Acid-fast staining and specific culture are recommended. Next-generation sequencing is helpful to identify the organism sensitively and quickly. Considering the detrimental outcomes, timely diagnosis and combined treatment are quite important to prevent the loss of vision and eye.

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## Compound Heterozygous *LTBP2* Mutations Associated With Juvenile-Onset Open-Angle Glaucoma and Marfan-Like Phenotype

The gene encoding latent transforming growth factor  $\beta$  binding protein 2 (*LTBP2*) has been implicated in the development of primary congenital glaucoma (PCG) and other developmental glaucomas.<sup>1</sup> Mutations in *LTBP2* causing PCG have been identified in consanguineous families, suggesting an autosomal recessive inheritance pattern.<sup>2,3</sup> Ocular phenotypes associated with *LTBP2* mutations include goniodysgenesis resulting in elevated intraocular pressure (IOP) and glaucomatous optic nerve damage, lens instability or dislocation, and high myopia.<sup>2,3</sup> We present a young patient carrying compound heterozygous mutations in *LTBP2* leading to juvenile-onset glaucoma and other ocular sequelae.

**Report of a Case** | The patient is a 32-year-old female whose race she self-reported as African American. At age 13 years, she was diagnosed with glaucoma with elevated IOP, which

was managed by bilateral trabeculectomy. Approximately 4 years later, significantly elevated IOP in both eyes was detected accompanied by severe cupping and open iridocorneal angle in each eye (Figure 1A and B). She had no evidence of Haab striae, buphthalmos, or high iris insertion suggestive of PCG. Ultimately, she required bilateral glaucoma tube shunt implantation. Subtle lens dislocation superonasally in the right eye was observed at her initial visit at age 17 years, which slowly progressed over a 16-year period (Figure 1C and D). At age 32 years, she developed lens dislocation in the left eye.

Given the constellation of taller stature than her relatives (180.4 cm) (Figure 2), long fingers, and dislocated lens, a connective tissue disorder was suspected, especially Marfan syndrome, and the patient underwent cardiac and genetic evaluation. Serial echocardiograms failed to reveal any abnormalities, but hypermobility of the shoulders, digits, and knees was observed. A comprehensive connective tissue genetic testing panel including 92 genes (Invitae) revealed 2 heterozygous variants in the *LTBP2* gene (c.709C>T p.Arg237\* and c.3776-1G>C splice acceptor), not previously reported in gnomAD (0.00%) or other publications. A complete family history was negative for similar findings. Cascade testing of the patient's 2 children with normal ocular examination results at age 4 years and 10 years revealed 1 child carrying each variant, consistent with transheterozygous mutations responsible for the patient's predominantly ocular and mild systemic phenotypic manifestations.

**Discussion** | *LTBP2* is expressed in ocular tissues that regulate IOP, including the trabecular meshwork and ciliary body.<sup>4</sup> Homozygous *LTBP2* mutations in a domestic cat population caused elevated IOP, globe enlargement, and elongated ciliary processes, similar to human PCG. Subtle lens dislocation with zonular instability was common, though complete ectopia lentis occurred in less than 10% of the animals.<sup>5</sup> In humans, homozygous *LTBP2* mutations have similarly been associated with megalocornea, ectopia lentis, and myopia.<sup>3</sup> Although glaucoma and ectopia lentis associated with *LTBP2* mutations have been reported, the cause of glaucoma is often attributed to ectopia lentis. Biallelic mutations in the *LTBP2* gene have been associated with microspherophakia and early ectopia lentis in patients with a Marfan-like phenotype (tall stature, high-arched palate, long arm span) and later secondary glaucoma development.<sup>3</sup> This patient presented with severe bilateral optic nerve cupping and subtle dislocated lens only in 1 eye. Over more than a decade, we detected slow progression of lens dislocation in 1 eye and also recently detected subtle dislocated lens in the other eye. Careful longitudinal observation suggests that glaucoma development is independent of dislocated lens. Compound heterozygous mutations in *LTBP2* have previously been reported in a patient with juvenile open-angle glaucoma.<sup>6</sup> However, this patient had isolated ocular pathology with no other systemic findings. Our report highlights the pluripotent functions of *LTBP2* in the eye as well as systemically. Identifying patients who carry these mutations is important