

Case Report

Traumatic glaucoma with features of unilateral pigment dispersion

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Summary

We report a patient with traumatic glaucoma with features of unilateral pigment dispersion. This rare form of secondary glaucoma has only been reported twice previously, with both patients demonstrating angle recession, indicating associated damage to the trabecular meshwork. To our knowledge, this is the first such case reported in which angle recession was absent.

Case Report

A 68-year-old white man presented at Conquest Hospital, Hastings, with a complaint of redness and discomfort in the left eye 2 days after suffering blunt trauma with the tip of a pool cue. Medical history was remarkable for myopia previously treated with radial keratotomies. On examination, the left eye had diffusely injected conjunctiva with anterior chamber cell, traumatic mydriasis, and an intraocular pressure (IOP) of 20 mm Hg, with no gonioscopic evidence of angle recession. Radial keratotomy scars were visible in both eyes. Neither eye had features of pigment dispersion. Fundus examination revealed cup:disc ratios of 0.3 in both eyes, with no disc features suggestive of glaucoma. He was treated as a case of traumatic iritis and started on a tapering course of topical 0.1% dexamethasone every 2 hours.

At 4 weeks' follow-up, the left eye was found to have an IOP of 70 mm Hg and a quiet anterior chamber. Although the left eye was physiologically shorter than the right eye, it was noted to have a visibly asymmetric, deeper anterior chamber on examination. This finding was confirmed by measurements obtained from the IOL-Master (Carl Zeiss Meditec, Jena, Germany): axial length 26.44 mm in the right eye and 25.84 mm in the left eye, anterior chamber depth 3.21 mm in the right eye and 3.45 mm in the left eye. There were no features

of pigment dispersion. The elevated IOP was believed to be secondary to steroid therapy, which was consequently discontinued. He was started on topical latanoprost once daily and dorzolamide/timolol twice daily. This had the effect of reducing his IOP to 18 mm Hg. At 22 weeks' follow-up, his IOP had risen to 54 mm Hg, and he was noted to have pigmented cells in the anterior chamber for which he was restarted on topical dexamethasone 3 times daily. A Humphrey visual field 24-2 test was performed, revealing a mean deviation of -12.32 dB and an inferior arcuate scotoma. His IOP increased to 67 mm Hg, and he was started on oral acetazolamide 250 mg twice daily and referred to a glaucoma specialist.

On examination of the left eye by a specialist 3 days later, the IOP was 42 mm Hg, the anterior chamber was asymmetrically deep with peripheral iris concavity, and there were midperipheral iris transillumination defects without other signs of iris damage. Gonioscopy showed evidence of Sampaolesi's line and no clinical evidence of angle recession (Figures 1–2). The cup:disc ratio in the left eye was 0.65, with superior neuroretinal rim thinning.

The patient was diagnosed with traumatic pigment dispersion syndrome without angle recession and was started on maximal topical IOP reduction therapy; other medications were discontinued. Three days later, his IOP remained high, at 34 mm Hg, and a left trabeculectomy

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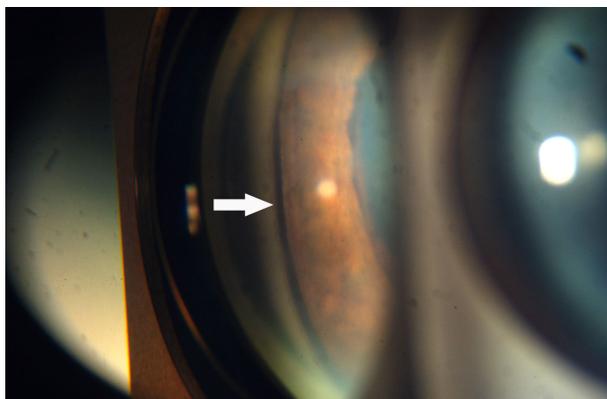


Figure 1. Photograph of the left eye at the time of presentation showing Sampaolesi's line temporally.

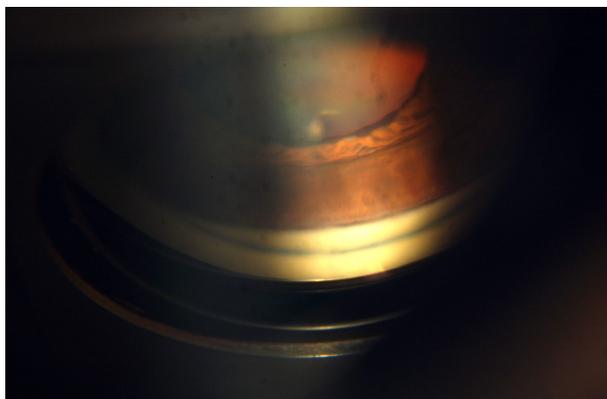


Figure 2. Gonioscopic view of superior angle: Sampaolesi line just visible with no evidence of angle recession.

tomy augmented with mitomycin was performed the same day. One month after surgery, best-corrected visual acuity was 20/30 and the IOP improved to 16 mm Hg. Sixteen weeks postoperatively, the patient developed a visually significant cataract with a best-correct visual acuity of 20/60. Phacoemulsification of the cataract with posterior chamber intraocular lens implant was performed; despite the sustained contusion, the lens was stable perioperatively, with no evidence of zonular dehiscence. Two weeks after phacoemulsification, uncorrected Snellen visual acuity was 20/30, and IOP was 14 mm Hg.

Discussion

Three factors were responsible for this patient's development of glaucoma: (1) blunt ocular trauma, (2) steroid response, and (3) pigment dispersion. Although von Hippel proposed in 1901 that anterior chamber pigment

could be a causative factor in elevating IOP through trabecular meshwork blockage, it was not until 1949 that pigmentary glaucoma was described in a case series of two white male patients by Sugar and Barbour,¹ whose original paper united the clinical findings of myopia, corneal endothelial pigment deposition, iris transillumination defects, trabecular meshwork pigmentation, and elevated IOP exacerbated by mydriatic use.¹ Subsequent work established predilections toward white, myopic males. The condition now referred to as pigment dispersion syndrome is typically bilateral. Although many cases are sporadic, genetic linkage analysis by Andersen et al² has elucidated an autosomal dominant form linked to a gene tracked to the long arm of chromosome 7q35–q36.

Pigment dispersion is an uncommon clinical entity following ocular trauma but has been reported by Ritch et al and McKinney and Alward within the context of angle recession.^{3,4} Although angle recession is known to close within weeks of a sustained injury,⁵ our case is unusual in that it is, to our knowledge, the first reported case of unilateral pigment dispersion secondary to trauma without angle recession at presentation.

It has long been recognized that glaucoma may occur as a late complication of blunt ocular injury. The incidence has been reported at 3.39% at 6 months⁶ and, in cases of angle recession secondary to blunt trauma, 6%–7% at 10 years.^{7–8} A recent anecdotal report indicates that glaucoma may occur as late as 23 years after blunt trauma.⁹ This variation in reported incidence data may not only reflect time frame differences but also differences in specific etiology and consequent severity of the injury. Sports-related injuries are commonly implicated causative agents, but a variety of other causes have been reported, including airbags, elastic cords injuries, bottle caps and corks, airsoft pellets, and paintball guns.^{10–15}

Elevated IOP in blunt trauma can be secondary to angle recession, inflammation, intraocular bleeding, lenticular dislocation, and, in severe blunt trauma, phacoanaphylaxis secondary to capsular rupture.^{16–17} Multivariate analysis of 6,021 cases in the US Eye Injury Registry identified key features associated with the development of traumatic glaucoma: advancing age, presenting visual acuities <6/60, iridial or lenticular injuries, hyphema, or angle recession. Other risk factors include corneal injuries, lens displacement, presence of optic atrophy, vitreal injuries, increased pigmentation of the angle, elevated presenting IOP and traumatic cataract.^{18–20} Many of these features reflect the degree of anatomical disruption and hence the severity of the forces involved in the blunt injury. In a study of 37 patients with blunt ocular trauma

without gonioscopic angle recession, Kashiwagi et al demonstrated a greater increase in anterior chamber depth in iris periphery relative to central iris.²¹ This may relate to relatively greater stretch damage sustained by the iris in blunt ocular trauma near its fixed insertion at the angle. Canavan and Archer²² reported damage at this fixed insertion point—angle recession—in a series of 212 eyes as being the most common form of damage following contusion. In our case, we believe that the blunt ocular trauma sustained was insufficient to cause angle recession but culminated in a greater peripheral increase in anterior chamber depth, iridial concavity, and ultimately the irido-zonular contact necessary for pigment dispersion.^{23–24}

In our patient, IOP elevation occurred 1 month following initial presentation and steroid use. Steroid-induced glaucoma typically occurs within weeks of initiation of topical steroid therapy, with pressures as high as 60 mm Hg reported following prolonged use.^{23–24}

Literature search

The authors searched PubMed without date or language restriction in June 2010 using the following combinations of terms: *pigment dispersion; glaucoma AND pigment dispersion; glaucoma AND trauma*.

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