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Recurrent corneal oedema following late migration of intraocular glass

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Abstract

This is a report of very late complications following intraocular penetration of numerous fragments of glass as a result of a test tube explosion. Fifteen years after the initial injury glass splinters began to migrate from the vitreous into the anterior chamber, causing acute episodes of corneal oedema. Four such episodes occurred over the past nine years, the corneal oedema each time disappearing within a few days following surgical extraction of the glass splinters. The literature on intraocular glass and its movement within the eye is reviewed.

Penetration of glass splinters into the eyeball is much rarer than the penetration of metallic foreign bodies.[1] In about 4–7% of eye injuries there is involvement of glass,[2] with a similar percentage found in children.[2] According to Rathmann and Aerztl[3] only one of 62 eye injuries by glass resulted in perforation, with glass retained within the eye. Unless it is photochromic glass is inert and does not cause any chemical reaction.[3] Damage is due either to the original impact of the injury or to late secondary movement of the glass within the eye causing mechanical tissue damage.

There are few reports describing late spontaneous movement of intraocular glass fragments. The present report describes recurrent migration of glass splinters from the vitreous cavity into the anterior chamber, which started 15 years after the initial injury and continued intermittently over nine more years.

Case report

A 16-year-old school boy was admitted to our department on February 1965 because of bilateral perforating eye injuries caused by a chemical explosion of a glass test tube. Numerous glass splinters covered his face and chest. In both eyes the cornea was studded with splinters, which were also seen within the lens. An intummescent cataract developed in the left eye and was removed. This eye regained full visual acuity (6/6).

In the right eye the lens absorbed spontaneously, and only needling of the posterior capsule was eventually required. Because of pigmentary changes in the macula, vision in this eye remained at 6/20. Four years after the initial injury no posterior vitreous detachment could be seen, but a localised flat retinal detachment was diagnosed in the upper nasal quadrant. Numerous immobile glass splinters could be seen within the vitreous of the eye, and these did not cause any disturbance for about 15 years. However, during all this time glass splinters were extruded from both corneae, with small corneal erosions and short episodes of tearing and pain.

In 1980 there was the first episode of severe corneal oedema in the right eye. Gonioscopy revealed two glass fragments, 2–3 mm in diameter, within the lower part of the angle. When the patient was prone these fragments were seen to lie free on the corneal endothelium. Both fragments were surgically removed through a limbal incision, and thereafter up to December 1988 the patient underwent three more similar operations with removal of glass from the anterior chamber angle, always because of recurrence of moderate to severe corneal oedema. After each of the four operations the oedema disappeared completely within two to five days.

When last seen in June 1989, the right eye was quiet and the cornea was clear, with minute glass splinters within the stroma. The anterior chamber was deep. The pupil was slightly irregular but reacted normally. There was aphakia with a large central opening in the posterior capsule. The vitreous, which showed advanced syneresis, contained numerous glass splinters distributed both anteriorly and posteriorly within it. The flat retinal detachment in the upper nasal quadrant remained unchanged. The disc was normal, and there were old pigmentary changes in the macular area. Vision remained 6/20 with aphakic correction.

In the left eye no further changes have occurred, and vision remained 6/7 with aphakic correction.

Discussion

Cases in which glass splinters penetrate the eye are relatively rare owing to the low weight of glass. In most cases glass penetrates the eyeball as a result of an explosion, in which the high kinetic energy compensates for the low weight. More rarely penetration is due to broken spectacle lenses, window panes, etc.

Cutko (quoted by Milkowski[2]) collected 100 cases with retained intraocular glass fragments which were seen between 1942 and 1960. In the majority, 73 eyes, the localisation was within the anterior chamber; in the remaining cases the fragments were described within the lens, ciliary body, vitreous, retina, and choroid.

Unlike other intraocular foreign bodies, glass tends to be localised within the anterior segment of the eye. Fragments in the posterior segment are relatively immobile and usually less injurious. Cohen[4] described a glass fragment stuck in the retina, remaining asymptomatic over a period of 28 years. Santoni[1] described a fragment of glass near the disc remaining asymptomatic over a period of 11 years; while another fragment from

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the same injury caused episodes of bullous keratitis for three years until it was discovered and removed from the anterior segment. Glass fragments may remain for years within the lens and cause only minimal local cataract.[5]

There are rare reports of late migration of glass fragments from the posterior to the anterior segment of the eye. Löhlein[6] described migration from the ora serrata causing traumatic cataract five years after the original injury, when the glass eventually found its way into the anterior chamber. Cutko (quoted by Milkowski[2] described migration of a fragment from the posterior chamber into the anterior chamber, which occurred six weeks after the injury. Cauer[7] described a fragment which was stuck between the sclera and ciliary body remaining asymptomatic for 23 years and was then found in the anterior chamber.

Migration of intraocular glass is always from the back forwards and, in 85% of the cases, also downwards.[2] The last direction of movement is easily understood, since glass is heavier than aqueous. It is more difficult to explain the forward movement, and various factors need be considered. Simple dislocation of glass within the eye could be triggered or influenced by repeated trauma, sporting activities, or head and eye movements. The overall forward movement is probably influenced by intraocular flow of the aqueous. When splinters are retained within the lens they may fall into the anterior chamber as a result of a slow process of spontaneous absorption of the lens.

In cases of aphakia, especially after intracapsular cataract extraction, splinters can more easily move from the posterior to the anterior segment. In the present case there were large openings in the posterior capsule that allowed such movement already within the first year following the injury. The fact that glass splinters started to appear in the anterior chamber and caused corneal oedema 15 years after the initial injury is probably due to the slow and progressive liquefacation of the vitreous over this period of time.

The tendency to vitreous liquefaction increases with age and probably occurs sooner and more often following trauma that involves the posterior segment of the eye. In the present case the initial injury occurred at the age of 16, and vitreous liquefaction was sufficiently advanced to allow migration of glass from it into the anterior chamber at the age of 31. So far no late injury has occurred within the posterior segment from the free movement of glass within the vitreous. The late, localised, flat retinal detachment in the right eye appeared relatively early, did not progress over the years, and no retinal tear could be found. Probably it resulted from vitreous traction following the original injury. At the time of the initial injury, in 1965, as well as four years later when the localised detachment was diagnosed, vitrectomy did not exist. Later on it was considered when fragments of glass migrated into the anterior chamber, causing corneal oedema. However, in view of the very large number of glass splinters within the vitreous this procedure seemed to be risky, especially in the presence of a localised retinal detachment that has not progressed over a long time.

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