

Pseudophakic pupillary-block glaucoma

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SUMMARY Four cases of iris-supported pseudophakic pupillary-block glaucoma were presented. Pupillary-block glaucoma is the first postoperative complication seen following the implantation of an intraocular lens, and in our series occurred at an incidence of 3·8%.

A short review was made of pupillary-block glaucoma with all types of intraocular lenses, with emphasis on the iris-supported lens. The role of inflammation, haemorrhage, and vitreous and lens material in obstructing aqueous flow at the pupil and peripheral iridectomy site was emphasised. Pitfalls in the diagnosis and management of this condition were reviewed. Methods of prevention and treatment were reviewed with emphasis on early mydriasis, along with carbonic anhydrase inhibitors and hyperosmotic agents as a primary medical treatment. Iridectomy, laser iridotomy, or transfixation of the iris was mentioned as a surgical treatment.

Pseudophakic pupillary-block glaucoma was reported by Ridley (1960). Interest at this time centred on posterior chamber lenses. However, Boberg-Ans (1961), Barraquer (1962), and Bresnick (1969) had focused attention on the same complication occurring with anterior chamber lenses. This unique form of glaucoma was briefly described by Nordlohne (1975) in association with iris-fixation lenses and iridocapsular lenses.

A total of 106 iris-supported lenses were inserted as primary procedures at the Jewish General Hospital in Montreal from March 1975 to November 1975. The most frequent and serious complication was pseudophakic pupillary-block glaucoma. It occurred in 4 patients for an incidence of 3·8%.

Although the problem has been identified in the literature, a thorough description of the entity is lacking. It is the purpose of this paper to review pseudophakic pupillary-block glaucoma, to illustrate the pitfalls in the diagnosis and management, and to recommend a simple method of prevention and treatment.

Case reports

CASE 1

A 75-year-old Caucasian female was admitted to the Jewish General Hospital on 14 August 1975 for cataract extraction and lens implantation. Physical examination showed no significant abnormalities,

with the exception of the eye findings. The best corrected visual acuity was 20/70 in the right eye and 20/400 in the left eye. Slit-lamp examination revealed anterior cortical opacities as well as nuclear sclerosis in the left lens. Intraocular pressure was 10 mmHg in the right eye and 8 mmHg in the left eye.

On 15 August 1975 the patient had an uncomplicated intracapsular cataract extraction in the left eye. The anterior chamber was entered through a 2-planned incision which was extended until it encompassed 180°. Alpha-chymotrypsin was used. A platinum clip iris-fixation lens was inserted into the anterior chamber and secured through a superior peripheral iridectomy. Acetylcholine chloride was injected into the anterior chamber to position the lens. The section was closed with 8 interrupted 8–0 silk sutures.

On the first postoperative day there was moderate epithelial oedema, a shallow anterior chamber, and an intraocular pressure of 44 mmHg. Therapeutic dosages of oral acetazolamide and intravenous mannitol were started. The intraocular pressure then fell to the mid-30s. By the fifth postoperative day, on the same medical therapy, the clinical situation was unchanged and the surgeon elected to perform a vitreous aspiration. 1·5 ml of fluid vitreous was aspirated through the surgical wound and the anterior chamber deepened. The acetazolamide and mannitol were discontinued. On the sixth postoperative day the intraocular pressure was 20 mmHg.

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Twenty-four hours later, however, the intraocular pressure had risen to 50 mmHg. The anterior chamber had shallowed and there was iridocorneal touch inferiorly. Acetazolamide and mannitol were again given. On the eighth postoperative day, mydriasis with phenylephrine 10% and tropicamide 1% was attempted. A good mydriatic response was achieved, but vitreous presented between the lens margin and the pupil in all areas except the inferior temporal quadrant. The intraocular pressure remained in the high 40s. On gonioscopic examination iris bombé was noted and the angle structures could not be visualised. On the ninth postoperative day the intraocular lens implant was removed. The intraocular pressure, however, remained elevated, and ecothiopate iodide (Phospholine Iodide) 0.06% twice a day was required to bring the intraocular pressure in the range of 20 mmHg. By October 1975 the intraocular pressure had stabilised in the high teens, controlled by 1 drop of 0.125% ecothiopate iodide daily.

Subsequently the intraocular pressure became elevated and was uncontrolled on maximum medical therapy. The patient required a combined trabeculectomy and anterior vitrectomy to lower the pressure. On her most recent examination the best corrected visual acuity was 20/400 and the intraocular pressure was 22 mmHg on pilocarpine 6% every 4 hours and acetazolamide 250 mg q.i.d.

CASE 2

A 68-year-old Caucasian male was admitted to the Jewish General Hospital on 10 April 1975 for cataract extraction. He was a mild diabetic and hypertensive, under good management. Physical examination was normal, with the exception of the ocular findings. His visual acuity was counting fingers at 3 ft (1 m) in the right eye and 20/40 in the left eye. Slit-lamp examination revealed cortical opacities and nuclear sclerosis in the right eye. The anterior chamber was deep in both eyes. The intraocular pressure was 18 mmHg, OU.

On 11 April 1975 the patient underwent an uncomplicated lens extraction with instillation of alpha-chymotrypsin prior to the delivery of the lens. A sprocket-type Federov iris-fixation lens was implanted and a single peripheral iridectomy was made at the 1 o'clock position. Acetylcholine chloride was injected into the anterior chamber during the final positioning of the lens. Eight 8-0 virgin silk sutures were used to close the 2-planed limbal section.

On the first postoperative day the patient was started on pilocarpine 4% q.i.d. One day later the intraocular pressure was recorded at 70 mmHg, and the anterior chamber was considered to be

shallow. The peripheral iridectomy could not be visualised because of the conjunctival flap. For 16 hours an unsuccessful attempt was made to control the elevated intraocular pressure with oral acetazolamide and intravenous mannitol.

It was then elected to perform a second surgical procedure. Fluid vitreous was aspirated through the original incision and a second peripheral iridectomy was made at 11 o'clock. Following this procedure acetazolamide was discontinued and 4% pilocarpine was given once a day for 2 days. On the fourth postoperative day the intraocular pressure was 18 mmHg. The anterior chamber was deep and the lens was in good position. By October 1975 the intraocular pressure was 11 mmHg, with the patient on no medication. The best corrected visual acuity in the right eye was 20/30.

CASE 3

A 76-year-old Caucasian female was admitted to hospital on 27 August 1975 for a cataract extraction. Visual acuity was hand motion in the right eye and 20/40 in the left eye. The right eye had a mature cataract. Intraocular pressure was 16 mmHg, OU.

On 28 August 1975 a right cataract extraction was performed through a 2-planed 160° limbal incision. Alpha-chymotrypsin was used. While the lens was being removed the posterior capsule ruptured and a small amount of liquid cortical material spilled into the anterior chamber. All remnants of cortical and capsular material were removed. During the insertion of a Worst medallion iris-fixation lens a leg of the lens was inadvertently hooked with the iris suture, and to correct this the lens was removed. At this point vitreous entered the anterior chamber and an anterior vitrectomy was done using cellulose sponges and scissors. When the anterior chamber was free of formed vitreous, the lens was repositioned and a peripheral iridectomy was made at the 12.30 position. Acetylcholine chloride was injected into the anterior chamber to constrict the pupil and re-form the anterior chamber. The section was closed with a running 10-0 nylon suture placed in a 'shoelace' fashion.

On the first postoperative day epithelial oedema, striate keratopathy, a shallow anterior chamber, and an intraocular pressure of 50 mmHg were found. The lens was in good position. Despite oral acetazolamide, betamethasone ophthalmic drops, and frequent hyperosmotic diuretics, the intraocular pressure remained elevated above 32 mmHg for 3 days. Gonioscopy revealed a closed angle inferiorly. Superiorly the iridectomy and angle were obscured by the epithelial oedema. On the fourth day the pupil was dilated with tropicamide 1% and phenylephrine 10%, every 3 hours. The lens moved superiorly,

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but its legs remained posterior to the iris. After 12 hours of this therapy the intraocular pressure had fallen to the high 20s, and within 24 hours the pressure was 17 mmHg, OU. Acetazolamide was discontinued, and 24 hours later the intraocular pressure was 16 mmHg. In September 1976 the visual acuity was 20/50 and the intraocular pressure was 18 mmHg, with the patient on no therapy.

CASE 4

A 64-year-old Caucasian female was admitted to hospital on 28 October 1975 for cataract extraction. The best corrected visual acuity was 20/70 in the right eye, 20/200 in the left. Intraocular pressure was 15 mmHg, OD, 18 mmHg, OS. Posterior subcapsular and anterior subcapsular opacities were noted, OU.

The patient underwent a left intracapsular cataract extraction the following day. Alpha-chymotrypsin was used. Formed vitreous was lost and 2.5 ml of fluid vitreous was aspirated through the surgical wound. A Worst medallion lens was inserted and secured to the iris with a 10-0 nylon suture. At this point a single peripheral iridectomy was made. The anterior chamber was re-formed with acetylcholine and the 2-planed incision closed with a running 10-0 nylon suture. After closure 40 mg of methylprednisolone acetate was injected subconjunctivally.

On the first postoperative day a moderate amount of epithelial oedema was noted. The lens was in good position and the anterior chamber was deep. Acetazolamide 125 mg by mouth 6-hourly was initiated. The intraocular pressure was not recorded. Nevertheless, by the third postoperative day the intraocular pressure was 42 mmHg and the anterior chamber appeared shallow. The acetazolamide was increased to 250 mg by mouth 6-hourly. 24 hours later the intraocular pressure was 50 mmHg and the iris was touching the cornea inferiorly. At this point phenylephrine 10% was given for 1 dose. The anterior chamber deepened but the pressure remained in the 40s. Intravenous mannitol was added, with no significant change in the intraocular pressure. On the fifth postoperative day, the anterior chamber remained shallow and the intraocular pressure remained elevated.

A second course of phenylephrine 10% was initiated. Following its instillation the anterior chamber deepened but the intraocular pressure remained unaltered. One dose of pilocarpine 4% was tried, but to no avail. The miotic was discontinued shortly thereafter and the patient placed on phenylephrine 10% and homatropine 5%, every 2 to 3 hours. On the sixth postoperative day the intraocular pressure fell to the low 20s, and

the patient was discharged 24 hours later on phenylephrine 10%, tropicamide 1%, and homatropine 5%, every 3 hours throughout the day. In November 1976 the visual acuity was 20/50, the intraocular pressure 24 mmHg, and the patient off all medication.

Discussion

The history of intraocular lenses has been associated with pseudophakic pupillary-block glaucoma. The first intraocular lens was a posterior chamber lens, introduced by Ridley. It was placed in the posterior chamber after extracapsular cataract extraction, and this frequently resulted in a marked inflammatory response. At this point a pupillary membrane could form, obstructing normal aqueous flow and thus resulting in pupillary-block glaucoma. As described by Reese (1960) and Ridley (1964), the treatment was dissection of the membrane, that is, corepraxia.

Anterior chamber lenses became popularised after the posterior chamber lenses. Here again inflammation or haemorrhage could create an obstruction to aqueous flow. This problem was discussed by Boberg-Ans (1961), Barraquer (1962), and Bresnick (1969). Another mechanism of pupillary block is the closure of the peripheral iridectomy by the supporting arms of the lens. Boberg-Ans (1961) suggested making fenestrations in the haptic support portion of the lens to prevent this.

At our institution only iris-fixation lenses have been used. The incidence of pupillary-block glaucoma with iris-supported lenses has been well documented. Nordlohne (1975) reviewed his own cases as well as those of Binkhorst and Worst. In all the cases reported by these authors, except for 1, the pupillary block occurred between the first and fourth postoperative days. Worst reported 1 case occurring 2 months after surgery. The incidence varied among surgeons. Binkhorst reported 2 cases in 534 operations (0.29%); Worst had 3 cases in 568 operations (0.45%); and Nordlohne had 5 cases in 73 operations (6.94%). In our series we had 4 cases in 106 operations for an incidence of 3.8%.

Iris-fixation lenses and iridocapsular lenses are of primary importance when considering pupillary-block glaucoma, for they are located in close proximity to the pupil along the normal route of aqueous flow. Iris-fixation lenses are usually implanted after intracapsular cataract extraction. Iridocapsular lenses are implanted after extracapsular cataract extraction and depend upon an inflammatory response between iris, lens, and posterior capsule to provide firm adhesions for stabilisation and support. With either type of lens, haemorrhage and/or inflammation enhance the possibility of obstructing the pupil.

Immediate postoperative mydriatics are not used with either lens, but in some cases miotics are used. This, too, can add to the potential for pupillary block. Regardless of the mechanism, once the pupillary pathway for aqueous is impaired, the patency of the peripheral iridectomy becomes of paramount importance. It can, however, be obstructed by blood, vitreous, lens material, trapped air, or by pigment epithelium from an incomplete iridectomy.

The diagnosis of pupillary block can easily be made, but it is frequently missed. There are several factors that should lead the surgeon to the correct diagnosis. It is usually the first postoperative complication, occurring within 48 hours of surgery. The patient frequently complains of ocular pain, disproportionate to the amount of surgical trauma. There is epithelial oedema and striate keratopathy. The anterior chamber is shallow, with iris bombé, and if an air bubble is present in the anterior chamber, it will be flattened against the back of the cornea. The intraocular pressure is elevated, usually above 40 mmHg. There is often a history of operative difficulties, such as rupture of the anterior vitreous face, with or without vitreous loss, operative hyphema, and unexpected extracapsular extraction.

The elevated intraocular pressure is usually ascribed to postoperative inflammation and/or the use of alpha-chymotrypsin. Topical steroids, carbonic anhydrase inhibitors, and hyperosmotic agents are administered, but the pathophysiological mechanisms persist and the pressure remains uncontrolled. The presence of a shallow anterior chamber may occur as a result of a wound leak, but when associated with elevated intraocular pressure, it generally implies pupillary block. In rare clinical situations, 'ciliary block' glaucoma due to cilio-vitreous adhesions, as described by Weiss (1972), may underlie the problem and present in a similar manner. This diagnosis is generally made after peripheral iridectomy fails to reverse the glaucomatous situation.

Once the diagnosis is made, the treatment of pseudophakic pupillary block is relatively simple. Nordlohne (1975) outlines a 3-step approach to this complication. If the mechanism is from an air-bubble block of the peripheral iridectomy, the patient should be repositioned and the air bubble may be displaced, establishing normal flow of aqueous. If this fails, miotics should be discontinued and mydriatics administered. It should also be emphasised that mydriatic agents be used simultaneously with carbonic anhydrase inhibitors and/or hyperosmotic agents. As a third step, an additional peripheral iridectomy or transfixation of the iris should be performed.

Tessler (1975) has described the conversion of an incomplete iridectomy to a through-and-through opening with the argon laser. Perkins (1973) has reported iridotomy with a ruby laser. We have not used the laser, but its use should be considered prior to surgical reintervention.

In our series the following errors were made in diagnosis and therapy. In case 1 mydriatics were not started until 7 days postoperatively, and only after a vitreous aspiration was attempted. In case 2 the first treatment was a vitreous aspiration with a peripheral iridectomy. Mydriatics were not tried. In case 3 it took 3 days before the correct diagnosis was made and appropriate therapy begun. In case 4 the diagnosis was delayed by 1 day, and then the correct treatment was not started until 24 hours had passed. Other authors have had similar difficulties. Of the 10 cases reviewed by Nordlohne (1975) only 5 were managed following the treatment outlined in his text. In these cases surgical treatment was performed before complete medical therapy had been tried.

Even when the diagnosis of pupillary block is entertained, a fear of lens dislocation may delay the use of mydriatics. The use of an iris suture or platinum clip lens helps to mitigate this anxiety. Either from fear of dislocation or from not appreciating the potential benefits of mydriasis many surgeons at this point go directly to the next step in treatment, which is another peripheral iridectomy. In many instances unnecessary surgery can be avoided. Confusion may arise after the mydriatics are used and the anterior chamber deepens, for at this point the intraocular pressure may remain elevated for another 24 hours (cases 3 and 4). With a deep chamber one must persist with mydriatics.

Steps may be taken at the time of surgery to avoid pupillary-block glaucoma. Boberg-Ans (1965) recommends 2 peripheral iridotomies. Nordlohne (1975) suggests that they not be made too peripheral because they then become more easily blocked. If miotics are required (case 2), weak ones should be used. We also feel (cases 3 and 4) that intraocular lenses should not be implanted in eyes where excessive postoperative inflammation from an extracapsular cataract extraction is anticipated; in cases of vitreous loss; and in eyes that develop large hyphemas at the time of surgery. The use of an iris-fixation suture or an iris clip lens will prevent dislocation when mydriatics are used. The surgeon will treat the complication earlier with mydriasis if he is confident that the lens is held securely by one of these additional supports.

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