Ocular Hypotony after Baervaeldt Shunt Implantation for Glaucoma

Julinda Jaho¹, Suzana Nuellari², Elona Bucaj¹

¹Department of Ophthalmology, University Hospital Center of “Mother Theresa”, Tirana, Albania
²Polyclinic of Specialities No 3, Tirana, Albania

Abstract: We are reporting a case of a 61 years old, male patient, with refractory glaucoma, who underwent uneventful Baervaeldt shunt implantation in the left eye. The eye was stable on antiglaucomatous therapy after surgery. He developed almost flat anterior chamber on the ninth postoperative day. Injection of air and balanced salt solution (BSS) in the anterior chamber was performed. The patient came back the next day, in the same situation, with almost flat anterior chamber. Injection of Sodium Hyaluronate in the anterior chamber was performed, hoping that the flow through the shunt would stabilize. The next day, the anterior chamber was flat. Opening of the conjunctiva and ligation of the tube with a new suture (8-0 Vicryl) was performed. The anterior chamber stabilized after this third intervention. There are specific measures that should be taken to prevent hypotony after Baervaeldt shunt implantation, especially in patients predisposed for postoperative hypotony. Immediate intervention is necessary in cases of extreme hypotony where preservation of the globe is threatened.

Keywords: Baervaeldt shunt, ocular hypotony, glaucoma surgery

1. Introduction

Although trabeculectomy is still considered as gold standard surgical procedure for glaucoma, the glaucoma drainage devices are increasingly used, especially in refractory glaucoma patients.

The mechanism of glaucoma drainage devices is to divert the aqueous humor from the anterior chamber to an external reservoir formed by the fibrous capsule around the device. 4-6 weeks are necessary for the reservoir to be formed. Baerveldt tube shunt is one of these devices, which is part of the non-valved implants. This means that the aqueous humor can flow in both directions of the shunt, contrary to valved devices, which allow the aqueous humor to flow only from the anterior chamber to the reservoir.

Studies that compare the trabeculectomy outcome to tube shunt surgery, report a higher success rate of tube shunts to trabeculectomy with mitomycin.[1] However, even tube shunt surgery has its own complication risks, such as risk of postoperative hypotony, diplopia, strabismus, proptosis, tube erosion, failure, corneal decompensation, endophthalmitis and visual loss.[2]

The aim of this case report is to discuss postoperative hypotony after Baervaeldt shunt implantation.

2. Method and Materials

We are reporting a case of a 61 years old male patient, pseudohypakic, with pseudoexfoliative glaucoma in the left eye and cornea guttata in both eyes. He had history of failed trabeculectomy and canaleoplasty in the left eye. He reported a history of hypotony following the trabeculectomy. His preoperative best corrected visual acuity (BCVA) was 0.4 in this eye (Snellen chart). His intraocular pressure had recently increased to 31 mmHg regardless of the use of three topical antiglaucomatous medications. The patient underwent uneventful Baervaeldt shunt implantation. The eye was stable on antiglaucomatous therapy after surgery. He presents to the clinic on the ninth postoperative day, complaining of sudden low vision and light pain in the eye. The slit lamp examination revealed almost flat anterior chamber and intraocular pressure (IOP) of 6 mmHg (measured with applanation tonometry). The cornea was slightly edematous. Injection of air and balanced salt solution (BSS) in the anterior chamber was performed. The patient was advised to stop all the antiglaucomatous medication in this eye. He reported back the next day, in the same situation, with almost flat anterior chamber. Injection of Sodium Hyaluronate in the anterior chamber was performed, hoping that this would stabilize the flow through the shunt. Atropine drops were prescribed. The next day, the anterior chamber was flat. No evidence of choroidal effusion or hypotony/maculopathy was present during the examination. Premature suture loosening was suspected and exploration of the extra ocular part of the tube was decided. Opening of the conjunctiva and ligation of the tube with a new suture (8-0 Vicryl) was performed. The anterior chamber stabilized after this third intervention, but he developed relevant corneal decompensation within six months. His BCVA decreased to 0.16 (Snellen chart). The IOP could not be measured accurately, because of the corneal decompensation, but the globe was soft during the 4 year follow-up without the need of any antiglaucomatous medication. No evidence of choroidal effusion was present at any point during the follow-up. Even though the ocular hypotony was managed in time, the visual acuity decreased because of the corneal decompensation. The patient has been informed about the possible benefits of a corneal transplant.

3. Discussion

Hypotony and complications related to it may occur after glaucoma drainage devices insertion, especially after non-valved devices, such as Baervaeldt shunt. This has been a
major concern in the past. In a study published in 1995, hypotony occurred in 32% of 103 eyes where Baerveldt shunt was implanted.[3] Nowadays, hypotony owing to overfiltrations an uncommon complication of glaucoma drainage devices surgery.[4]

Postoperative hypotony is divided into early (occurring within 2 weeks of surgery) and late (occurring more than 2 weeks after surgery). Sometimes, the observation and adjustment of medications is enough to stabilize the IOP. In some cases, injection of viscoelastic in AC is necessary to get the IOP under control. However, if the hypotony persists, its management is possible only by surgical intervention. Joshua D. Stein et al reported that most of the patients with persistent hypotony included in his study were treated by GDD ligation. He reported that the hypotony was treated successfully in 50% of eyes in which polyglactin suture was used, compared to 80% receiving prolene sutures. He concluded that in a small subgroup of patients receiving GDD, the eye is hypotinous, either because it cannot form a capsule, has persistently low aqueous production from postoperative iritis, from too wide sclerostomy with flow around rather than through GDD itself, or from other causes.[5] We suspect that the ligation suture loosened prematurely in our patient.

However, as the old phrase says, prevention is the best cure. There are a few intraoperative measures that can be taken to prevent early hypotony in patients with non valved tube shunts. External tube occlusion (ligature) can restrict the flow of aqueous humor through the tube in the early postoperative period. This can be done using a non-absorbable suture with a releasable knot or 7-0 or 8-0 absorbable polyglactin suture tied around the tube. We used an 8-0 absorbable polyglactin suture in our patient. Alternatively, the tube can be ligated using a 9-0 nylon or 10-0 prolene suture in the distal end, which can be later cut with laser.

Another method to prevent early hypotony is to insert a 4-0 or 5-0 prolene or nylon suture through the tube, limiting the aqueous flow through the tube. This suture is to be removed after the fibrous capsule forms. We did not insert such a suture in our patient.

In specific cases predisposed for early postoperative hypotony, a two-stage surgery can be performed, first implanting the plate and tube in the subconjunctival space, and then inserting the tube in the anterior chamber after a period of 4-6 weeks.

Incomplete occlusion of the tube is one of the reasons for early postoperative hypotony. A test injecting balanced salt solution in the tube after its ligation was performed intraoperatively, which showed that the occlusion of the tube by ligation was complete.

The history of hypotony after trabeculectomy could have been one of the points to take into consideration for taking extra measures in preventing the hypotony in our patient. In addition, presence of cornea guttata and pseudexfoliation could have been considered by us for being more cautious in ensuring a deep anterior chamber postoperatively. Both of these conditions negatively influence the vitality of endothelial corneal cells, and consecutively the resistance of endothelium to devastating factors such as flat anterior chamber with iridocorneal or phacocorneal touch.

We were lucky that the eye got stable immediately after ligation of the tube with 8-0 polyglactin suture, although corneal decompensation was later developed.

4. Conclusion

There are specific measures that should be taken to prevent hypotony after Baerveldt shunt implantation, especially in patients predisposed for postoperative hypotony. Immediate intervention is necessary in cases of extreme hypotony, where preservation of the globe is threatened.

References