

fore be effective, by decreasing vascular permeability. The spontaneous resolution of choroidal ischemia has been reported previously [5], but in our case, because the patient showed no signs of any functional or anatomic improvement after observation for a week, we decided to treat the patient with IVB, which led to prompt visual and anatomical recovery.

This is the first case report of the occurrence of choroidal ischemia following brain tumor surgery and complete recovery of BCVA after IVB. In patients with severe visual loss and SRD associated with choroidal ischemia, IVB may be a viable treatment option.

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Conflict of Interest

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***In Situ* Peripheral Iridoplasty in Phakic Eyes for the Treatment of Symptomatic Peripheral Iridotomy**

Dear Editor,

Laser peripheral iridotomy (LPI) is commonly used to treat acute angle-closure glaucoma (AACG) and to prevent glaucoma before posterior chamber phakic intraocular lens (IOL) implantation. Patients may experience some of the following visual symptoms after LPI: diplopia, ghost images, lines, glare, and haloes. These symptoms are associated with high lid position or large iridotomy size [1]. The typical treatment for such symptoms is a tinted contact lens. When patients cannot tolerate tinted lenses, surgery

may be necessary. Here we effectively adapted a simple iridoplasty technique for the treatment of symptomatic LPI.

LPI was performed in the first case to treat AACG. A 46-year-old woman presented postoperatively with glare in the left eye. Her left eye best-corrected visual acuity (BCVA) was 20 / 20 and intraocular pressure (IOP) was 13 mmHg. Slit-lamp microscopy revealed two LPIs 1 / 2 to 2 / 3 of pupil size at 10 and 2 o'clock as well as incipient cataracts (Fig. 1A). In the second case, LPI was performed for the prophylaxis of glaucoma after the implantation of a phakic IOL in a 42-year-old female. Postoperatively, the patient complained of glare and photophobia in the left eye. Her left eye BCVA was 20 / 15 and IOP was 12 mmHg. The slit-lamp examination revealed a clear lens and two LPIs at 11 and 1 o'clock that were partially covered by the upper lid (Fig. 1A).

Both patients agreed to undergo peripheral iridoplasty.

The surgery was performed using a modified Siepser slipknot technique (Fig. 1B and 1C) [2-4]. The primary incision was made in superior cornea (Fig. 1B-1 and 1C-1). A secondary incision was made near the iridotomy to allow for the injection of viscoelastics and insertion of the IOL manipulator. Sodium hyaluronate 1.4% (Healon GV; Pharmacia-Upjohn Ophthalmics, Kalamazoo, MI, USA) was injected to the anterior chamber and iridotomy site in a step-by-step manner to protect the lens (Fig. 1B-2 and 1C-2). A CTC-6 long, curved needle was passed through the main incision with 10-0 polypropylene suture. The needle was used to elevate and pick up one side of the iridotomy with counter-pressure provided by the IOL manipulator (Fig. 1B-3 and 1C-3) [5]. The needle was then passed through the other side of the iridotomy with the same support and finally exited through the corneolimb angle (Fig. 1B-4 and 1C-4). The IOL manipulator was introduced through the main incision, used to create a suture loop, and then retracted through the main incision (Fig. 1B-5 and 1B-6;

1C-5 and 1C-6). The free proximal end of the suture was passed through the loop three times (Fig. 1B-7 and 1C-7). The first slipknot was gently slipped through into the anterior chamber, then engaged *in situ* around the iridotomy site as each free end of the suture was pulled (Fig. 1B-8 and 1C-8). This maneuver was repeated twice to secure the knot (Fig. 1B-9 to 1B-12, 1C-9 to 1C-12). Both ends of the knot were then cut using long Vannas scissors. Depending on the size of the iridotomy, it was secured using one to two additional slipknots. The viscoelastic material was removed by manual irrigation. In order to prevent AACG recurrence in the first patient, iridotomy size was reduced by 80%. Postoperatively, two patients reported that their symptoms disappeared completely (Fig. 1A-2 and 1A-4). Their postoperative BCVAs were not decreased and IOPs were not elevated. No other postoperative complication was detected.

The use of peripheral iridoplasty to close symptomatic LPI without disturbing the lens represents a technical chal-

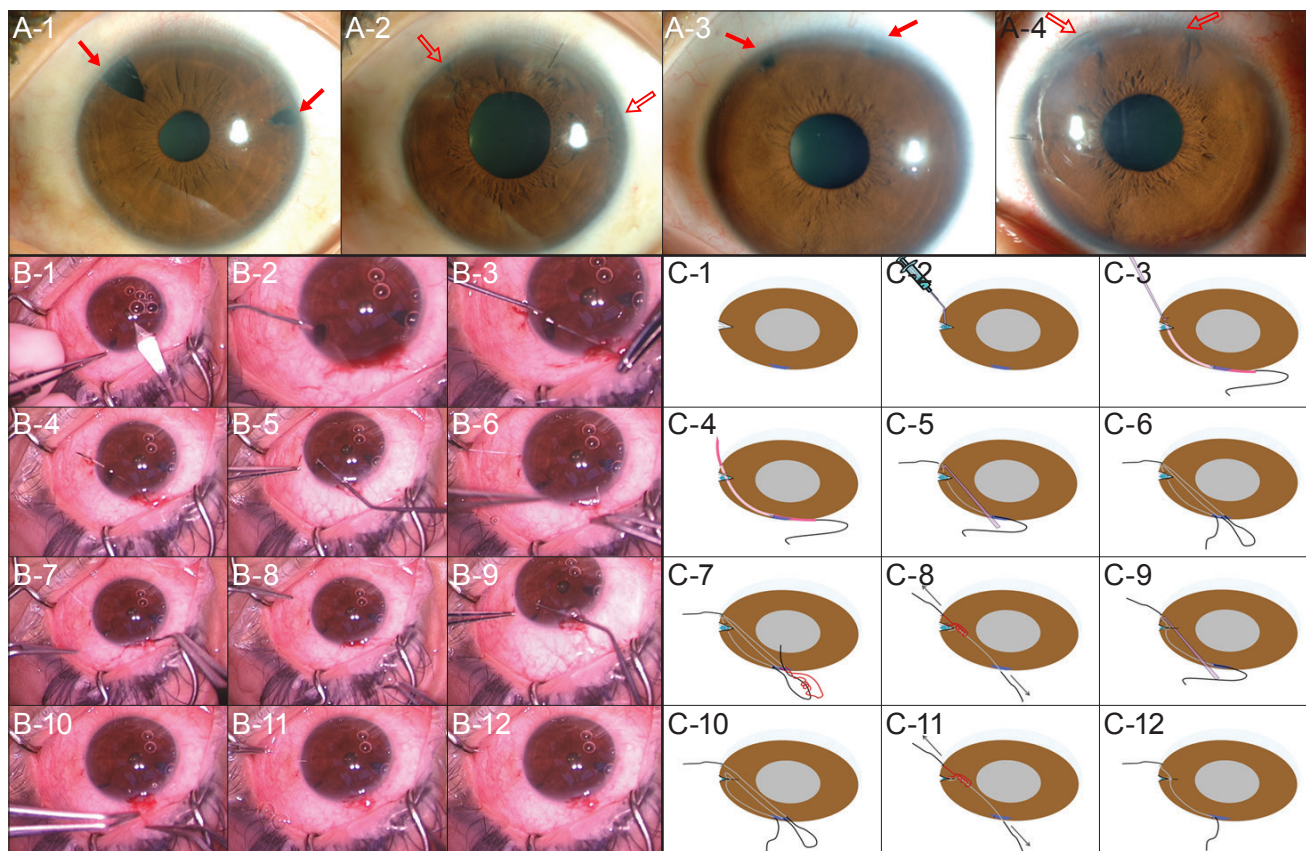


Fig. 1. Figures (A) presented preoperative and postoperative photographs of cases (case 1, A-1 and A-2; case 2, A-3 and A-4). Solid arrows indicate preoperative laser peripheral iridotomies (LPIs), and open arrows indicate closed LPIs. Figures (B) showed intraocular Siepser slipknot technique. Figures (C) presented schema of (B). Numbering is consistent with (B).

lenge. Surgery can lead to iridodialysis, cyclodialysis, and subsequent bleeding [5]. The following steps prevented these complications in the two patients presented here. First, we used a modified Siepser technique for creation of the knot *in situ*. This type of knot minimizes the force pulling on the iris from the direction of the corneolimb angle. Second, we used an IOL manipulator to create a counter-force when the needle was passed through the iris. Third, the viscoelastics is inserted in a step-wise manner. This was performed to prevent iris prolapse through the primary incision, which would subsequently trigger a sharp spike in IOP. Finally, the needle was maneuvered extremely gently when passing horizontally through the anterior chamber to as to avoid contact with the lens. This is the first report to describe the adaptation of a modified Siepser slip-knot technique to treat symptomatic LPI in phakic eyes.

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Ethmoidal Sinus Mucocele as a Cause of Acquired Brown Syndrome

Dear Editor,

A mucocele is composed of a respiratory epithelial lined mucus-containing lesion with accumulation of mucoid secretion. Sometimes, it produces remodeling of the bony structure and influences the adjacent tissues [1]. In this case, extension of the ethmoid sinus mucocele and remodeling of the orbital structure were major causes of acquired Brown syndrome. Although various causes of acquired

Brown syndrome have been described, this is the first reported case of acquired Brown syndrome due to ethmoidal sinus mucocele in Korea.

A 69-year-old male presented to our institution with a newly-developed diplopia. There was no history of trauma or any medical illness. At initial examination, visual acuity was 20 / 30 in both eyes. The intraocular pressure was 17 mmHg in the right and 20 mmHg in the left eye. There was no relative afferent pupillary defect and color vision was normal.

A soft compressible tender mass measuring 2 × 2 mm on the superonasal aspect of the left upper lid extending from the trochlea to the medial canthal tendon was noted. A motility examination showed an under-elevation in adduction of the left eye mimicking Brown syndrome (Fig. 1A). A