Motility Restriction after Resection of an Extraocular Muscle

Shin Jeong Kang, MD, Jeung Hun Jang, MD

Department of Ophthalmology, Inje University Sanggye Paik Hospital, Seoul, Korea

Restriction of eye movement after surgery is an unusual but troublesome complication. A patient presented with a limitation of abduction after a 5 mm resection of medial rectus muscle and an 8 mm recession of lateral rectus muscle. Since the forced duction test was positive, restrictive factors were suggested to be implicated. A reparative operation was performed at the postoperative 9 month, and the forced duction test was negative after releasing the resected medial rectus muscle. The patient showed an improved abduction after resecting the resected muscle. Even after an uneventful surgery, resection of an extraocular muscle may cause restriction of ocular rotation caused by muscle scarring to the sclera or by an increased tightness of the muscle.

Key words: extraocular muscle, resection, restriction

INTRODUCTION

When a patient presents with a limitation of ocular rotation in one or more directions, it is assumed that there are both innervational deficiencies and/or mechanical restrictions of the extraocular muscles. Mechanical restrictions may be more common than purely paretic causes of reduced ocular rotations.1 Restrictions frequently occur after an orbital floor fracture, endocrine ophthalmopathy, Brown’s syndrome, following multiple strabismus procedures, orbital or retinal detachment surgery, or muscle transposition surgery. When such a restriction occurs after a surgery, there are several causes: excessive resection of an extraocular muscle or excessive recession of its antagonist; a slipped muscle; Tenon’s capsule scarring, and so on.2

We treated a patient who showed limited abduction postoperatively after resection of the medial rectus muscle. Since the forced duction test was positive, restrictive causes were implied. The literatures were reviewed, and possible factors affecting outcome are herewith suggested.

CASE REPORT

An 18-year-old man visited our clinic complaining of an intermittent deviation of his left eye. He rarely noticed the deviation in his left eye until 2 years previously at which time his friend’s knee hit his left eye. Although his vision remained unchanged, he noticed that the frequency of the intermittent deviation had increased, especially when he was talking to others.

Ocular examination showed a visual acuity in right eye of 20/20 and in left eye 20/40, which was corrected to 20/20 with -1.25 D cyl x 180°. Th
anterior and posterior segments were normal. Prism and alternate cover tests showed a 40-prism diopter of intermittent exotropia in the primary position at near and 35-prism diopter of intermittent exotropia at distance after correcting for his refractive errors. Ductions in the both eyes were normal, and the amount of deviation was the same in all gaze directions. An overaction of the oblique muscles was not found. On the Worth 4 dot test, he demonstrated fusion at near and left suppression at distance. An operation on his left eye was performed, which recessed the left lateral rectus muscle 8 mm and resected the left medial rectus muscle 5 mm by means of limbal incision using local anesthesia (retrobulbar injection of 2% lidocaine). Phenylephrine 2.5% was instilled into the conjunctival sac prior to the operation, and excessive cautery was avoided during the procedure.

On the first postoperative day, the patient showed a 20-prism diopter esotropia in the primary position at near and distance. A limitation of abduction was noted in the left eye not crossing the midline. During subsequent visits, the esotropia in the primary gaze improved and he showed orthotropia at the primary position at one month postoperatively (Fig. 1). The limitation of abduction, however, did not improve and his left eye did not cross the midline on an attempted lateral gaze (Fig. 2). The forced duction test revealed a resistance of the lateral gaze in the left eye. During his visit at the postoperative 9th month, he was orthotropic in the primary gaze but the limitation of abduction in his left eye still persisted. Since there was no improvement in the abduction of left eye, an exploration of the previous operation site and/or release of the resected medial rectus was planned.

When the resected medial rectus was exposed, the overlying conjunctiva, Tenon’s capsule and the muscle sheath did not show signs of excessive fibrosis. The new insertion site of the resected muscle was 5 mm from the limbus, as originally planned. After dissecting the conjunctiva and Tenon’s capsule, a forced duction test was performed to see whether fibrosis of overlying soft tissue caused the limitation. The same amount of resistance still was felt on abduction as occurred during the preoperative state. After the medial rectus muscle was secured by 6-0 vicryl and detached from its insertion, there was no limitation of abduction on the forced duction test. The medial rectus muscle was reattached to the sclera 4 mm from its insertion, which resulted in a 1 mm resection effect compared to the original preoperative state.

On the first postoperative day, the patient showed a slight exophoria in his primary gaze at near and distance. Compared to the preoperative state, the lateral gaze of his left eye improved, but a -2 grade of limitation still remained on attempted abduction. On the 7th postoperative day, there was an 8 prism diopter of exophoria in his primary gaze at near and distance with good fusional movement. The limitation on abduction did not change, and showed a -2 grade of limitation. The patient was lost to further follow-up.
DISCUSSION

Limitations of ocular rotation in one or more directions may have several causes. These can be divided into: (1) a diminished innervational effect, and (2) a mechanical, restrictive effect. In practice, restrictions are more commonly seen. Various disorders of ocular motility have been shown to have significant restrictive elements that cause a limitation of the full rotation of the eye. These include: (1) secondary to trauma; (2) endocrine ophthalmopathy; (3) congenital disorders; (4) postoperative sequelae; (5) longstanding, large deviations; (6) transposition surgery; (7) orbital tumor; and (8) orbital inflammation. Postoperative restrictions can be seen following retinal detachment surgery, orbital surgery, and multiple strabismus operations.

Our patient showed a limitation of abduction on the postoperative 1 day. There can be several causes to explain this: an excessive resection of a medial rectus muscle; scarring and hemorrhage on the medial side of the globe that causes restriction due to tightness of Tenon’s capsule and scar tissue; when a suture has become untied or torn soon after lateral rectus muscle surgery; or when a lateral rectus muscle has been recessed excessively. Since the forced duction test showed strong resistance in abduction and the patient was orthotropic at the primary postoperative period, restrictive factors were regarded as being the cause. We excluded the possibilities of a slipped lateral rectus muscle or an excessive recession.

In order to prevent these complications, the technique used for the strabismus surgery is important. Rough handling of tissues, “popping” the orbital fat into the surgical field and an excessive or poorly controlled hemorrhage all contribute to form adhesions around the globe and therefore a limitation of full motility. Insufficient surgery on Tenon’s capsule can cause muscle stretching and deformation, with a resultant restriction but without harm to muscle tissue and elasticity. More damage is done by improper muscle manipulation, which can cause stretching and scarring of Tenon’s capsule. This leads to a restricted motility that is frequently accompanied by muscle scarring as well. In such cases, very tightly scarred conjunctiva and Tenon’s capsule are seen over the restricted muscle, with Tenon’s capsule and a conjunctiva severely scarred to sclera from the muscle to the limbus. Our patient, however, did not show any signs of excessive scarring postoperatively, and the conjunctiva and Tenon’s capsule were not severely scarred at the time of reparative surgery.

When an eye has been exotropic by more than 30 prism dipters for a prolonged period of time, the lateral rectus and lateral tenons and conjunctiva can become contracted. This does not explain the findings in our patient, because the duration of time that our patient noticed the deviation was about 2 years, and the deviation was intermittent, not constant.

If an excessive amount of resection of a rectus muscle is performed, or if several resections have been done, the muscle can be very tight and thus limit ocular excursion. We did not regard a 5 mm resection of medial rectus muscle to be excessive for a 35 prism dipter of exotropia.

A change in the muscle length or arc of contact produced by strabismus surgery may affect the resting tension of agonist and antagonist muscles. This change in tension could result in a compensatory hypertrophy of the extraocular muscle. Compensatory hypertrophy is a response of muscle fibers to an increased load or tension on a muscle. Christiansen et al studied the compensatory hypertrophy of extraocular muscle by performing a large right lateral rectus resection in 16 rats. A large resection of the rectus muscle loaded its antagonist and induced a hypertrophic response. A large rectus resection also resulted in loading of the resected muscle. The hypertrophic response was not sustained beyond 4 weeks. This was possibly due to compensatory changes that decreased the loading effect over time. Even if a compensatory hypertrophy of resected medial rectus muscle occurred, it is difficult to explain the limitation that occurred on postoperative 1 day.

We regard muscle scarring to the sclera to be one of the causes, because the forced duction test was negative after releasing the resected medial rectus muscle. Although most postoperative restrictions are not primarily caused by muscle scarring to sclera but rather by scarring of Tenon’s capsule to muscle, sclera, and conjunctiva, it can be caused by
muscle scarring, particularly when the muscle is scarred to the sclera. This presents an unusual and difficult problem in surgical management. All tissue should be handled as gently as possible to prevent this restriction.

Resecting a muscle which has been shown to be tight preoperatively may have a large effect and produce a restriction. Even if the patient does not show any signs of a tight medial rectus muscle preoperatively, it cannot be excluded that such a resection might increase the underlying tightness of medial rectus muscle. Performing the forced duction test after the conjunctiva has been closed just prior to the completion of surgery may help to predict a tight muscle.

We think that the most probable cause of restriction is caused by muscle scarring, because we found no evidence of excessive fibrosis in the surgical fields, or a preoperative tightness of the medial rectus. The fact that a forced duction test was negative after releasing the resected medial rectus muscle increases the possibility of muscle scarring. With muscle scarring, even a 5 mm resection could lead to limitation of rotation. However, we do not know the reason why the limitation of abduction appeared just one day after surgery.

We have observed a complication of resecting an extraocular muscle even after an uneventful surgery, although the exact cause can not be determined. We think that the factors we describe should be considered before a resection procedure is performed.

REFERENCES

Fig. 1. White lens material on the anterior and posterior surface of the intraocular lens in the right eye.

Fig. 2. Gonioscopy of the right eye showing a wide-open angle with white cortical lens material in the inferior angle.

Fig. 3. Anterior chamber aspiration specimen showing macrophages with engulfed lens particles (Papanicolaou stain, ×400).

by Papanicolaou stain (Fig. 3).

DISCUSSION

In lens particle glaucoma, the lens protein can cause an obstruction of aqueous outflow like that experienced in phacolytic glaucoma, although free particulate lens material is the major component.1,2 The cellular reaction to these lens particles may also contribute to the glaucoma, however these lens particles themselves can mechanically impair trabecular drainage.3 The macrophages may obstruct the aqueous outflow, but may also act to remove lens debris by phagocytosis and thereby normalize the intraocular pressure.1,2

Generally, there is a delay of days or weeks between the cataract surgery and the onset of lens particle glaucoma. However, it has been reported that lens particle glaucoma has occurred 2 to 65 years after extracapsular cataract operations for congenital cataracts,4 and postulated that the delayed onset may have been due to the time required for the infantile residual lens material to denature to a heavy molecular weight and subsequently break into small pieces and release soluble lens protein, which in turn resulted in phacolytic and lens-particle glaucoma.5 However, in our case the glaucoma occurred in an adult who already possessed heavy molecular weight protein in his lens. We hypothesize that the lens material became sequestered in the space between the anterior and posterior capsular adhesion.