The Inferior Oblique Muscle Adherence Syndrome

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Objective: To describe the clinical features, etiology, prevention, and treatment of the inferior oblique muscle (IO) adherence syndrome.

Methods: This series consists of 12 patients treated for a restrictive hypotropia in which the middle portion of the IO was scarred anteriorly, either into or near the inferior rectus muscle (IR) insertion, after prior surgery.

Results: Among the 12 patients treated, the mean hypotropia of the affected eye was 18.1±7.2 prism diop ters and the mean excyclotropia was 13.8°±3.3°. Causes of the IO adherence syndrome included IR surgery (with or without prior IO myectomy) and scleral buckling surgery. The syndrome responded well to surgically releasing or myectomizing the incarcerated IO, combined with ipsilateral IR recession.

Conclusions: The IO adherence syndrome is a complication of surgery on the IR or of scleral buckling surgery, which can result in a restrictive hypotropia and excyclotropia. It can be prevented by paying attention to the anatomic relationship between the IO and IR and can be effectively treated if it occurs.

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Price1 described a complication of lateral rectus muscle (LR) surgery in which the inferior oblique muscle (IO) is inadvertently scarred into the LR body. This was subsequently described in greater detail by Helveston and coworkers.2 This complication can occur after resection or resection of the LR and can result in a hypotropia or hypertropia of the affected eye. The vertical deviation is usually small. I have observed other clinical situations in which the middle portion of the IO is inadvertently scarred into or near the insertion of the inferior rectus muscle (IR). Because this results in a more profound alteration in the force vector of the IO than the complication reported by Price1 and by Helveston and coworkers,2 it has a greater effect on ocular motility. Proper recognition of this situation can lead to effective treatment. This entity, which I call the inferior oblique muscle adherence syndrome, occurs as a complication of scleral buckling surgery or after IR surgery, particularly if the latter was preceded by ipsilateral IO myectomy. The purpose of this article is to describe the clinical features, etiology, prevention, and treatment of the IO adherence syndrome.

Methods

I conducted a retrospective review of my patient database to identify all patients I operated on between September 15, 1974, and January 1, 2006, in whom the middle portion of the IO was scarred into or near the IR insertion. Because other ophthalmologists performed all prior ocular surgical procedures, I obtained the operative reports for all patients to help determine the causes of this complication.

The preoperative examination included alternate prism and cover testing at 6 m in the primary position, cardinal positions, and oblique fields in all patients. Measurements were also obtained with the head tilted right and left, and at 0.33 m in the primary position. Torsion was measured subjectively with the double Maddox rods and objectively using indirect ophthalmoscopy. Ductions and versions were also assessed.

In all cases, my surgery was performed with the patients pharmacologically paralyzed to assess intraoperative passive ductions. After 1981, all patients underwent exaggerated passive duction testing to assess the oblique muscles as described by Guyton,3 and, after 1985, patients underwent intraoperative rotary passive duction testing to determine whether a torsional restriction existed. This test has previously been described in detail.4 All patients were followed up for at least 12 months after their latest surgical procedure. When adjustable sutures were used, the initial amount of recession was determined at surgery by the amount of restriction felt and by spring-back balance testing as described by Jampolsky.5 All patients undergoing adjustable suture surgery in 2000 or later were operated on using the semiadjustable suture technique.6

This study was approved by the University of Wisconsin’s institutional review board and complied with the Health Insurance Portability and Accountability Act.
Twelve patients were identified who had the IO adherence syndrome. Their characteristics are presented in the Table. Four patients developed this syndrome after prior scleral buckling surgery, 4 after IR surgery that followed prior IO myectomy, and 4 after IR surgery with no prior surgery on the IO. In this latter group of 4 patients, 2 developed this complication after a planned routine recession or resection on an IR that had not previously undergone surgery. In the other 2, the complication developed after surgery on an avulsed IR or after secondary surgery on an IR that had slipped after prior recession. In all 12 patients, the prior operative reports gave no indication of untoward events and none described any manipulation of the IO.

Eleven of the patients had similar clinical findings in that there was a large hypotropia of the affected eye (mean deviation, 18.1 ± 7.2 prism diopters [PD]; range, 10-35 PD) and a large excyclotropia (mean deviation, 13.8° ± 3.3°; range, 10°-20°). All patients had a limitation of elevation in all upgaze fields. In some, the limitation was slightly greater in elevation in abduction than in elevation in adduction, and in an almost equal number the converse was true. At surgery, they all had abnormal passive ductions for elevation, confirming that the limitation was restrictive. Rotary passive duction testing confirmed a torsional restriction in all patients in whom that test was performed. However, the 12th patient (case 10) simultaneously had the IO adherence syndrome and the superior oblique tendon incarceration syndrome, which initially had 18° of incyclotropia. After the superior oblique tendon inclusion was relieved, he had 15° of excyclotropia from his remaining IO inferior inclusion.

The 4 patients who had undergone prior scleral buckling surgery (cases 1-4) showed similar findings at the time I performed surgery. In all patients the IO was found to have been drawn up over the encircling band near the temporal corner of the IR insertion, and the buckle in effect tucked the IO (Figure 1). The configuration was as depicted in Figure 1, with the more nasal aspect of the muscle compressed between the buckle and the globe. In all 4 patients, the IO was scarred and atrophied under the buckle, and I was unable to effectively pull the muscle out from under the buckle with it intact. I treated all 4 patients by doing a myectomy of the IO between where it crossed the buckle and the temporal edge of the IR. In all cases this improved but did not normalize passive ductions. There-
fore, I also recessed the ipsilateral IR using an adjustable suture technique during the same operation. This resulted in normal passive ductions and normal rotary passive ductions at the end of the surgical procedure.

Two patients (cases 5 and 6) developed the IO adherence syndrome after prior IR recession or resection; however, they had no history of having undergone intentional IO surgery. In these patients, the middle portion of the belly of the IO was scarred into the temporal corner of the IR insertion (Figure 2). In these patients, the hooking of the IR at the time of their prior surgery likely also engaged the IO. Although releasing the incarcerated IO and repositioning the belly further posteriorly greatly reduced the vertical and torsional restriction, I also performed a small recession of the IR using an adjustable suture technique in one patient (case 6) and a nonadjustable technique in the other (case 5). This seemed advisable because the presence of a long-standing hypertropia likely caused some degree of contracture in the IR, even though I felt only mild restriction after I detached the IO.

In the 4 patients who had undergone previous IO myectomy (cases 7-10), I discovered at surgery that the proximal end of the previously cut IO was scarred into the temporal corner of the IR (Figure 3A). Because the IO had been myectomized, this remaining proximal part of the IO was tight. In most cases, the IO seemed to blend in with or was scarred into the adjacent IR belly, and it was not immediately obvious that there were 2 muscles attached to the IR insertion. The 2 most helpful signs in identifying this situation were that the tissue attached to the temporal corner of the IR insertion was substantially tighter than the nasal tissue and that, when I inspected the muscle tissue posteriorly, the fibers ran in different directions: the IR fibers ran posteriorly and the IO fibers coursed nasally. For these patients I dissected the IO free and retroplaced it to a point approximately 4 mm directly posterior to the temporal corner of the IR insertion. In all 4 patients I also performed an adjustable suture recession of the IR as a safety measure for the same reason described in cases 5 and 6. The IO adherence syndrome presumably occurred in cases 7 through 10 because the proximal cut end of the IO adhered to the sclera adjacent to the temporal edge of the IR. If, at the time of the subsequent IR surgery, the IO was inadvertently captured when the hook was passed to engage the IR, the 2 muscles could have become incorporated together during the subsequent suturing (Figure 3B).

Two additional patients (cases 11 and 12) developed the IO adherence syndrome after attempted correction of a slipped or avulsed IR. In both patients the belly of the IO was scarred to the original scleral insertion site of the IR; however, the IR was not directly attached to the globe. The IO was released and mobilized in both patients, and the IR was successfully identified and reattached. In case 11, the IR was indirectly attached to the globe by an 8-mm-long empty capsule, with the muscle attached to the belly of the IO at the Lockwood ligament. Advancing this muscle to 9 mm from the limbus and releasing the adherent IO successfully corrected the primary-position hypertropia and excyclotropia. For the previously slipped IR (case 11), I used an adjustable technique involving a nonabsorbable suture. In the patient in case 12, the anterior edge of the avulsed IR was also connected to the Lockwood ligament. With a fixed, nonadjustable technique, I advanced it to only 12 mm from the limbus because it was quite tight. This slightly overcorrected the patient's hypertropia in the primary position and resulted in a large left hypertropia in downgaze because the IR underacted after this surgical procedure. The patient subsequently required a right IR recession with posterior fixation. This additional operation eliminated his diplopia in the primary position but left him diplopic in downgaze. He has declined further surgery.

In 10 of the 12 patients, I used an adjustable suture technique for recessing the IR; in 2 of these, I used the semiadjustable modification. Seven of the 10 patients did not require postoperative adjustment. In 2 patients the recession was decreased approximately 1 to 2 mm, and in one it was increased approximately 1 to 2 mm. In addition to the vertical muscle surgery to correct the IO adherence syndrome, 4 patients also underwent simultaneous horizontal surgery.

The results of surgery in this series of 12 patients was gratifying. Only 2 required surgery beyond the initial one to correct the IO adherence syndrome (cases 10 and 12), and all were cosmetically acceptable and visually comfortable. No patient had clinically significant torsion after surgery; however, 5 required some prism for distance viewing, downgaze at the reading distance, or both to attain single binocular vision. Figure 4 and Figure 5 depict a representative example of a case patient before and after surgical correction.

**COMMENT**

The IO adherence syndrome is a complication of ocular surgery in which the IO becomes scarred into or near the temporal corner of the IR insertion. This syndrome can occur after a scleral buckling procedure or when IR surgery is performed. In the latter, it may be more likely to occur if the IO had been previously myectomized.
The IO adherence syndrome responds well to surgical correction. This should include releasing the restriction by means of myectomy or by repositioning the IO and probably should also include ipsilateral IR recession. The use of an adjustable suture technique is helpful. The most important factor in treating this condition is to have an index of suspicion that it is present and to carefully identify it. One should suspect it whenever there is a hypotropia associated with restriction, a limitation of elevation, and a large excyclotropia. In addition, the patient should have a history of IR surgery or scleral buckling surgery. In this sense, the clinical presentation of the
IO adherence syndrome can be identical to the fat adherence syndrome, which can follow IO surgery. However, if the surgeon assumes that a patient has the fat adherence syndrome and does not consider the IO adherence syndrome, corrective surgery may not be successful. Similarly, if a patient has undergone scleral buckling surgery and has the clinical findings described herein, one might assume that the generalized restriction is a result of the prior retinal surgery. Unless one specifically looks for the IO adherence syndrome, the problem may not be corrected. Simply removing the encircling band will probably be unsuccessful because the IO will remain scarred to the sclera in its anterior position.

In some of the patients described, the IO was inadvertently hooked when the surgeon attempted to isolate the IR. This can be prevented by hooking the IR from the nasal side. If the surgeon prefers to hook the IR from the temporal side, special care should be taken to avoid passing the hook too far posteriorly. In theory, placing the IO stump through the opening in the Tenon capsule during an initial myectomy may decrease the risk of adherence and minimize the likelihood of inadvertent IO capture during any subsequent IR surgery.

Anterior transposition of the IO is a popular operation for converting the elevating action of the IO to an anti-elevating action. It is commonly used to treat dissociated vertical divergence, as well as other ocular motility disorders. In that procedure, the IO is intentionally sutured at or near the point where the IO is inadvertently attached in the IO adherence syndrome; however, IO anterior transposition does not cause a restrictive hypotropia or a large excyclotropia. This difference is because, in the anterior transposition operation, the distal insertion of the IO is sutured near the IR insertion. With the IO adherence syndrome, a somewhat more proximal part of the muscle that is near its middle is sutured near the IR insertion. Hence, the muscle fibers that are even more proximal to the attachment site are substantially stretched. In addition, it has been shown that the neurofibrovascular bundle serves as an ancillary functional origin for the IO when it is transposed anteriorly, which can not only restrict elevation but also cause an excyclotropia.

Although the midportion of the IO is scarred anteriorly, as occurs with the IO adherence syndrome, the neurofibrovascular bundle is substantially stretched, thus contributing to the restriction, as depicted by the small arrow in Figures 1, 2, and 3.

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REFERENCES


