Superior Oblique Tendon Incarceration Syndrome

Burton J. Kushner, MD

Objective: To describe the clinical features, etiology, and management of superior oblique tendon incarceration syndrome.

Methods: This series consists of all patients I treated between September 15, 1974, and March 1, 2006, for restrictive hypertropia in which the superior oblique tendon was found scarred to the superior rectus muscle insertion after prior surgery.

Results: Twenty eyes in 18 patients were included in this series. The mean ± SD hypertropia of the affected eye was 15.4 ± 9.0 prism diopters, and the mean ± SD incyclotropia was 15.0° ± 3.5°. Causes of superior oblique tendon incarceration syndrome included prior superior rectus muscle resection, recession, plication, or transposition; superior oblique tendon recession, disinsertion, or posterior tenectomy; and scleral buckling surgery. The syndrome was difficult to treat and required a mean ± SD of 1.9 ± 0.7 additional surgical procedures to correct.

Conclusions: Superior oblique tendon incarceration syndrome is a complication of surgery on the superior rectus muscle or superior oblique tendon that can result in restrictive hypertropia and incyclotropia. Proper handling of the connection between the superior oblique tendon and superior rectus muscle at the time of surgery may prevent this complication, which can be difficult to treat.

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The superior oblique muscle has a triple function that causes intorsion, depression, and to a lesser degree, abduction. The specific location of the insertion of the superior oblique tendon and the angle made by the course of the tendon and the anterior-posterior axis of the globe determine how the muscle’s force is distributed among these 3 functions. Any substantial anterior displacement of the tendon’s insertion will greatly increase the intorsional force vector,1,2 and any movement nasal and anterior to the center of rotation of the globe may restrict depression.3,4

Several iatrogenic clinical situations exist in which the superior oblique tendon may become scarred to the nasal corner of the superior rectus muscle insertion.3,5 This occurrence has been referred to as superior oblique inclusion syndrome; however, I believe the name superior oblique tendon incarceration syndrome is more appropriately descriptive. To my knowledge, no reports have described this syndrome in detail, including its characteristic findings, etiology, prevention, or management. A search of the PubMed database for the keywords superior oblique inclusion or superior oblique incarceration found only 2 references to this clinical entity. One was a 4-case series by Metz and Norris6 of patients who developed torsional diplopia after scleral buckling. It included 1 patient in whom the torsion was caused by the superior oblique tendon becoming scarred anteriorly to the encircling band. The other was a report of different weakening procedures of the superior oblique tendon by Castanera de Molina et al3 in which they reported 2 cases of superior oblique tendon incarceration syndrome. In addition, Jampolsky5 mentioned the existence of this entity in published transactions of the New Orleans Academy of Ophthalmology. None of these reports described the syndrome in detail. I have observed and treated many patients with this syndrome. The purpose of this study is to provide information regarding the characteristics, etiology, prevention, and management of superior oblique tendon incarceration syndrome.

Methods

This is a retrospective review of all patients I operated on who were found at surgery to have the superior oblique tendon scarred to the nasal corner of the superior rectus muscle insertion. This review was accomplished by searching my patient database to identify patients in

Author Affiliation: Department of Ophthalmology and Visual Sciences, University of Wisconsin Hospital and Clinics, University of Wisconsin, Madison.
whom I observed that clinical finding between September 15, 1974, and March 1, 2006. For patients in whom prior surgery was performed by other ophthalmologists, the operative reports from the prior surgical procedures were obtained and studied to help determine the cause of this complication.

My preoperative examination included alternate prism and cover testing at 6 m in the primary position, in the secondary fields (gazes right, left, up, and down), and with the patient’s head tilted 30° to the right and left (Bielschowsky head tilt test). Measurements were also made at one-third meter. In most patients the misalignment at 6 m in the tertiary (oblique) fields was also quantified, unless the patient was too young to allow for this additional testing. Subjective torsion was tested in all sufficiently cooperative patients with double Maddox rods. Ductions and versions were also assessed.

Surgery was always performed under general anesthesia with the patients being pharmacologically paralyzed to permit accurate assessment of intraoperative passive ductions. After 1981 all patients underwent exaggerated passive duction testing to assess the oblique muscles, as described by Guyton. In addition, after 1985, patients underwent rotary passive duction testing at the time of surgery to determine if there was a torsional restriction. This test is performed by fixating the eye at the 6-o’clock and 12-o’clock positions with forceps and rotating the eye counterclockwise and clockwise until resistance is felt. If the eye could be passively intorted further than it could be extorted, a restriction to excyclorotation was present (Figure 1). All patients were followed up for at least 6 months after their last surgical procedure. This study was approved by the University of Wisconsin institutional review board and was compliant with the Health Insurance Portability and Accountability Act.

RESULTS

The review identified 20 eyes in 18 patients who were treated for superior oblique tendon incarceration syndrome. Their characteristics are presented in the Table. All patients in this series had hypertropia of the affected eye (mean ±SD, 15.7 ±8.7 prism diopters [PD]; range, 8-35 PD), except the 2 who had bilateral superior oblique tendon inclusion. All patients had some limitation of depression, but it was variable, ranging from –1 to –4 (on a 5-point scale of 0 to –4); consequently, the hypertropia increased in downgaze in all patients. In most the limitation was moderate (–2 to –3). In some patients depression was more limited in adduction and in others in abduction. Because of the overall complexity of these patients and the presence of other findings (eg, slipped muscles, restrictions secondary to Graves orbitopathy, or underacting muscles due to prior large recessions), one could not attribute all the limitation of depression to the incarceration of the superior oblique tendon. This can also explain the wide range observed with respect to downgaze limitation. All but 2 patients could be tested for subjective torsion, and they all had incyclotropia of the affected eye (mean ±SD, 15.1° ±3.5°; range, 10°-20°). Results of the Bielschowsky head tilt test were inconsistent. In some patients no substantial increase in hypertropia on head tilt to either side was found, and in others a small to moderate increase on ipsilateral head tilt was found. Only 2 patients showed a small increase on head tilt to the contralateral side. In general, the test was not useful diagnostically.

Vertical passive duction test results tended to be abnormal for depression, and they seemed to correlate with the size of the primary position hypertropia; the larger the deviation, the greater the restriction of passive ductions. All of the patients with unilateral involvement who underwent exaggerated forced duction testing had substantially more prominent tightness of the superior oblique tendon in the affected than in the contralateral eye. All patients who underwent rotary passive duction testing had a notable restriction of excyclorotation in the affected eye, which was abnormal to a greater degree than the exaggerated passive duction test results.

I had performed prior surgery in 1 of the patients (case 3) who had undergone 3 prior strabismus surgical procedures. Two of them involved surgery on her right superior rectus muscle before my caring for her. I advanced her right superior rectus muscle from 14 to 7 mm from the limbus, which resulted in superior oblique tendon incarceration syndrome. In the remaining 17 patients, someone else had performed the prior surgery. I was able to obtain previous operative reports for all 17, and thus I was able to tell what surgical procedure predisposed patients to superior oblique tendon incarceration syndrome. However, only 1 of the 17 reports indicated any unusual find-
ings at the time of surgery that suggested the superior oblique tendon might have gotten scarred to the superior rectus muscle insertion. In that case (case 12), unique findings were observed. This patient had previously undergone a right superior oblique tenectomy and right inferior oblique muscle myectomy to treat oscillopsia secondary to superior oblique myokymia by another pediatric ophthalmologist. These procedures eliminated his oscillopsia but left him with a 45-PD right hypertropia and an inability to elevate his right eye. He underwent additional surgery by the same physician, at which time his right superior rectus muscle was discovered to have been avulsed. The surgeon thought that there were 2 separate slips of muscle: a larger temporal slip and a smaller nasal slip. They were both sutured to the original superior rectus muscle insertion site, and the right inferior rectus muscle was recessed using an adjustable suture. After surgery the patient had no vertical misalignment in the primary posi-

Table. Characteristics of the Study Patients

| Case No. | Sex/Age, y | Affected Eye | Primary Position Hypertropia, PD | Incyclotropia, deg | Limitation of Depression in Most Limited Field | Downgaze Field of Greatest Hypertropia | Suspected Cause of Incarceration Syndrome | First Attempt to Correct Incarceration Syndrome | No. of Surgical Procedures to Correct Incarceration Syndrome |
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Abbreviations: deg, degrees; SR, superior rectus muscle; SO, superior oblique tendon.

a Case numbers were assigned thematically with patients being grouped according to similar prior surgical procedures rather than chronologically.

b Lists only the surgical procedure that specifically caused superior oblique tendon incarceration and does not include history of other muscles operated on.

c Superior rectus muscle had to be disinserted to free and mobilize the incarcerated superior oblique tendon.

d Does not include description of any horizontal muscle surgery to correct horizontal strabismus.
tion but had 15° of right incyclotropia with markedly limited elevation and depression of the right eye. His symptoms of oscillopsia had returned. I operated on him 3 months later and found the right superior oblique tendon scarred to the nasal corner of the superior rectus muscle insertion (Figure 2). Presumably, what was thought to be a nasal slip of superior rectus muscle at the time of his second surgical procedure was in fact the remaining superior oblique tendon, which was purposefully sutured to the superior rectus muscle insertion. After I performed a tenectomy of the remaining superior oblique tendon, he manifested 12° to 15° of right excyclotropia and still had limited elevation and depression of the right eye. During a subsequent operation, I found that the proximal end of his myectomized right inferior oblique muscle was tight and scarred to the temporal corner of the right inferior rectus muscle. Presumably, it had been inadvertently hooked and incorporated with the right inferior rectus muscle at the time of his right inferior rectus muscle recession. This condition was treated with an additional myectomy of the right inferior oblique muscle. In addition, a simultaneous posterior fixation of the contralateral left inferior rectus muscle was performed to treat a right hypertropia in downgaze. Although this patient’s findings are presented in the Table, I did not include them in calculations of the mean amount of hypertropia and incyclotropia in this series because he had the unique situation of having incarceration of both the superior oblique tendon and the inferior oblique muscle in the respective adjacent vertical rectus muscle insertion. I similarly excluded the 2 patients with bilateral superior oblique tendon incarceration from the calculation of the mean hypertropia in the primary position; however, I included them in the calculation of the mean amount of incyclotropia with each eye entered as a separate data point.

My approach for treating this entity is still in a state of evolution and is not fully satisfactory. Also, in addition to surgery on the incarcerated superior oblique tendon, many patients underwent simultaneous surgery on other vertical or horizontal muscles, which was dependent on the magnitude of the deviation, the presence of restriction that persisted after the incarcerated tendon was freed up, and their incomitant pattern. Consequently, I cannot provide a standard treatment approach for all patients with this syndrome. This confirms the idea that complex strabismus may begin with a specific cause (eg, a restriction) and then become multifactorial as other muscles become contractured. In this series, the most common manifestation of this fact was the presence of contracture of the superior rectus muscle in the affected eye. When that was found, the contractured muscle was recessed. Despite the heterogeneity of these patients, some general treatment principles have evolved. Initially, I simply tried to free the incarcerated tendon; however, in 3 of the 4 patients on whom I tried this approach, the adherence recurred shortly thereafter. This treatment was successful only in case 4 (Figure 3). Castanera de Molina described a similar lack of success in treating the syndrome by simply freeing the incarcerated tendon (written communication, August 22, 2006). In the subsequent 2 patients, I fashioned a sling of 6-0 polyglactin suture material and used that to hold the tendon approximately 5 mm posterior to the nasal cor-

Figure 2. Surgical findings in case 12. The superior oblique tendon (outlined in white dots) is scarred to the insertion of the superior rectus muscle (outlined in black dashes). This tendon is seen to be anterior to the superior rectus muscle. It was inadvertently sutured to the sclera after prior tenectomy during an attempt to repair an avulsion of the superior rectus muscle.
number includes the operation at which the superior oblique tendon incarceration was diagnosed and initially treated, it does not include any previous surgical procedures. One patient (case 8) had initially undergone bilateral superior oblique tendon recessions using a suspension technique (“hang-back”) by another ophthalmologist to treat marked overdepression in adduction, which resulted in a worsening of his findings. When I operated on this patient, I found superior oblique tendon incarceration syndrome bilaterally and treated him with split tendon lengthening in each eye, which only slightly improved his symptoms. Subsequent dynamic magnetic resonance imaging suggested that he had lateral rectus instability, as described by Oh et al.\textsuperscript{11} His lateral rectus muscles slipped superiorly on abduction, which may have been the cause of his initial overdepression in adduction. Further surgery is planned to stabilize the lateral rectus muscles inferiorly; however, it has not yet been performed.

Case 4 is unique in that the cause of the patient’s problem is somewhat unclear. She was born with left hypotropia associated with monocular elevation deficiency. Another pediatric ophthalmologist treated her initially with a 3-mm recession of her left inferior rectus muscle combined with a transposition of her left medial rectus muscle and left lateral rectus muscle to the left superior rectus muscle (Knapp procedure)\textsuperscript{12} at 8 months of age. He positioned the transposed horizontal rectus muscles to keep their new insertions on the spiral of Tillaux. This approach resulted in an overcorrection characterized by a 20-PD left hypertropia and limited depression of her left eye. Four months later the same surgeon recessed the left superior rectus muscle 5.5 mm. At the time of this second operation, the surgeon described substantial scarring around the superior rectus muscle and the transposed medial rectus muscle. This second operation had essentially no effect on the hypertropia. When I examined her at 18 months of age, she had 18 PD of left hypotropia and limited depression of her left eye. At surgery 1 month later, I found the left superior oblique tendon scarred to the nasal corner of the left superior rectus muscle insertion and the superior corner of the transposed left medial rectus muscle insertion (Figure 3B). I freed the superior oblique tendon and repositioned it in its normal anatomical position and the Knapp procedure was reversed, the patient had negligible left hypertropia.

Superior oblique tendon incarceration syndrome is a complicated iatrogenic disorder of ocular motility that can occur after surgery on the superior rectus muscle or su-
The superior oblique tendon is adherent to the undersurface of the superior rectus muscle by tenuous areolar tissue, which Jampolsky called the frenulum. He pointed out that when the superior rectus muscle is recessed, this frenulum will cause the superior oblique tendon to retract with the superior rectus muscle, as long as the frenulum is left intact. Prieto-Díaz reported similar observations. If the frenulum is severed, the superior oblique tendon will not retract and can scar to the superior rectus muscle insertion if the superior rectus muscle is recessed. Jampolsky therefore advocated preserving the frenulum when recessing the superior rectus muscle. This advice seems prudent to me. However, he also indicated that the frenulum may prevent the superior rectus muscle from taking up the slack if a recession of greater than 10 mm is desired using a suspension technique. In that circumstance, Jampolsky advocates cutting the frenulum. It seems to me that separating this connection between the superior rectus muscle and the superior oblique tendon could predispose the patient to superior oblique tendon incarceration syndrome, particularly because a recession of approximately 10 mm would place the new insertion directly over the superior oblique tendon. Prieto-Díaz observed that this does occur. However, Castanera de Molina has indicated that perhaps the adverse effects of superior oblique tendon incarceration might not be severe if it occurs close to the equator of the globe because the vector forces would not be as disadvantageous in that position (written communication, June 28, 2006). It is not clear from prior operative records if the frenulum had been severed in my 3 patients who developed superior oblique tendon incarceration syndrome after prior ipsilateral superior rectus muscle recession. However, in all 3 the recession was performed using a suspension technique. Conversely, the frenulum will cause the superior oblique tendon to be drawn forward if the superior rectus muscle is resected and the frenulum is left intact. Transposition of the superior rectus muscle temporally, as is performed to treat sixth cranial nerve palsy, can drag the superior oblique tendon with it if the frenulum is not severed. I am similarly unclear as to how the frenulum was handled in my patients who had prior superior rectus muscle resection or transposition temporally. It is also possible that in my patients who had prior recession, resection, or transposition of the superior rectus muscle, the superior oblique tendon was inadvertently hooked when the superior rectus muscle was identified and was inadvertently incorporated in the sutures.

The normal anatomical course of the superior oblique tendon has its functional origin (the trochlea) anterior to its insertion. Consequently, the distance between the superior rectus muscle insertion and the superior oblique tendon is shorter along the nasal edge of the superior rectus muscle than along the temporal edge. Thus, if the superior oblique tendon is recessed following its normal anatomical course, the insertional end will move forward and be closer to the superior rectus muscle insertion. Any recession procedure that does not fix the superior oblique tendon to the sclera at the desired point may result in this restrictive syndrome. Seven eyes in 5 of my patients developed this complication after prior surgical procedures on the superior oblique tendon, either in the form of a disinsertion or recession using a suspension technique. I know of no way to prevent this syndrome with either of those surgical procedures, unless there is minimal stripping of the frenulum at the time of surgery. However, if the frenulum is left essentially intact, the tendon will be constrained and unable to effect the desired recession. This is consistent with Parks’ observations reported in his Doyne Memorial Lecture. He stated that complete disinsertion of the superior oblique tendon will have a minimal weakening effect unless the anterior and posterior connective tissue around the tendon are incised, in which case the weakening effect is profound. Interestingly, Castanera de Molina et al compared different superior oblique weakening procedures and found the highest incidence of persistent overdepression in the infra-adducted position with disinsertion (62.5%), which they referred to as an uncontrolled procedure. They also reported cases of superior oblique tendon incarceration syndrome with that procedure, which is consistent with my observations. According to Castanera de Molina et al, in 1974 Prieto-Díaz reported in a transaction of the Consejo Latino-Americana de Estrabismo (CLADE) Congress that superior oblique tendon inclusion syndrome can occur after a variety of surgical procedures on the superior oblique tendon or superior rectus muscle.

In the 3 patients who developed this complication after scleral buckling surgery, I found that the encircling band had been placed posterior to the superior oblique tendon insertion. This approach had the effect of tucking the tendon and drawing it anteriorly. Obviously, care must be taken to avoid this situation during placement of the encircling band. These cases are somewhat different from the one described by Metz and Norris. In their patient, the superior oblique tendon was scarred to the posterior edge of the encircling silicone band, which in turn was posterior to the superior rectus muscle insertion. Their patient was successfully treated with freeing of the scarred tendon. In my patients, the superior oblique tendon was incarcerated between the superior rectus muscle insertion and the encircling band, which in effect tucked the tendon (Figure 4). It was impossible to simply mobilize the tendon without removing the silicone band, and the tendon beneath the band was atrophic. In this series, I excluded patients who had the findings at surgery described by Metz (eg, the superior oblique tendon scarred to the posterior edge of the buckle) because I believe that represents a somewhat different entity.

The lack of uniform response to the Bielschowsky head tilt test is not surprising given the heterogeneity of the initial strabismic conditions in this patient group. Some had dissociated vertical divergence, suspected superior oblique muscle palsy, partial third cranial nerve palsy, Graves orbitopathy, and comitant primary vertical strabismus. Many had undergone prior surgery on multiple vertical muscles. These factors can all result in misleading findings with the 3-step test, which is designed to identify isolated vertical muscle palsy. This study needs to be viewed in light of its limitations. Being retrospective in nature, measurements were
not obtained in a masked manner or with the rigor that is inherent in prospective studies. Nevertheless, I believe the qualitative descriptions are sufficiently accurate to provide the ophthalmic surgeon with the information needed to recognize this entity. Also, it can be hard to extrapolate meaningful treatment protocols from this series because the patients had such complex and heterogeneous histories and findings. In addition, because this series represents my experience during a period of more than 30 years, there was presumably a learning curve in my ability to identify and strategically treat this condition.

I believe that this study justifies some conclusions. Superior oblique tendon incarceration syndrome should be suspected in any patient who had prior superior rectus muscle surgery of any type or a scleral buckling procedure and in whom there is hypertropia of the affected eye, incyclotropia of 10° or more, and limitation of depression. Rotary passive ductions and exaggerated passive ductions at the time of surgery can be helpful in diagnosing this complication. It can be difficult to treat and may require more than 1 additional surgical procedure. Simply freeing the incarcerated tendon is often unsuccessful. Recession, tenectomy, or split lengthening of the tendon can eliminate the syndrome but may result in subsequent motility problems that require treatment. Until further studies are performed to clarify the behavior of the superior oblique frenulum during different surgical procedures, the following guidelines (based on theoretical considerations) seem prudent for prevention of superior oblique tendon incarceration syndrome: (1) do not separate the superior oblique frenulum when recessing the superior rectus muscle up to 10 mm; (2) separate the frenulum for superior rectus muscle resections or transpositions; (3) to prevent superior oblique tendon incarceration syndrome caused by superior oblique tendon disinsertions or resections with a suspension technique, strip the frenulum to effect the desired recession; and (4) whenever surgery is performed on the superior rectus muscle, identify the superior oblique tendon and confirm that it is not inadvertently incorporated in the sutures.

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Correspondence: Burton J. Kushner, MD, Department of Ophthalmology and Visual Sciences, University of Wisconsin Hospital and Clinics, 2870 University Ave, Suite 206, Madison, WI 53705 (bkushner@wisc.edu).

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REFERENCES