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Brown's superior oblique tendon sheath syndrome

Abstract

Brown's syndrome (superior oblique tendon sheath syndrome) presents as an inability to raise the adducted eye and a positive forced duction test. Since first recognized in 1950, this syndrome has been the subject of much controversy concerning its etiology and consequent treatment. Catagorized by Brown himself into true, simulated, acquired, intermittent and spontaneous recovery cases, the origins for this mobility anomaly range from a short anterior sheath of the superior oblique tendon to stenosing tenosynovitis and trauma. Treatment is difficult and is accomplished largely by tenotomy or tenectomy. This article provides the reader with an overview of the syndrome, its signs, history of etiology, prognosis and treatment.

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BROWN'S SUPERIOR OBLIQUE TENDON SHEATH SYNDROME

RESPECTFULLY SUBMITTED
BY

JANET L. LEASHER

IN ORDER TO FULFILL THE
REQUIREMENTS FOR THE
DOCTOR OF OPTOMETRY DEGREE

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PACIFIC UNIVERSITY
COLLEGE OF OPTOMETRY
FOREST GROVE, OREGON

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ABSTRACT

Brown's syndrome (superior oblique tendon sheath syndrome) presents as an inability to raise the adducted eye and a positive forced duction test. Since first recognized in 1950, this syndrome has been the subject of much controversy concerning its etiology and consequent treatment. Catagorized by Brown himself into true, simulated, acquired, intermittent and spontaneous recovery cases, the origins for this mobility anomaly range from a short anterior sheath of the superior oblique tendon to stenosing tenosynovitis and trauma. Treatment is difficult and is accomplished largely by tenotomy or tenectomy. This article provides the reader with an overview of the syndrome, its signs, history of etiology, prognosis and treatment.

Key words: Brown's syndrome, Superior oblique tendon sheath syndrome, Pseudoparalysis of the inferior oblique.

INTRODUCTION

Superior oblique tendon sheath syndrome was first described by Harold Whaley Brown at the Strabismus Ophthalmic Symposium in 1950[3]. This ocular motility anomaly features restricted elevation in adduction either passively or actively. It is prevalent in roughly one in every 450 cases if strabismus.[21] While this finding might seem to point to a paralysis of the inferior oblique, a positive forced duction test rules out any such paresis. Thus Brown originally proposed the syndrome could be a mechanical restriction at the trochlea due to a congenitally short anterior sheath of the superior oblique tendon.

Since that time, many other etiologies for this motility dysfunction have been suggested, causing Brown in 1973[4] to further catagorize superior oblique tendon sheath syndrome into two groups: true and simulated (see Table I). The true syndrome group includes only those cases where a congenitally short anterior superior oblique tendon sheath has been found. True cases may be either typical or atypical: the typical cases being those with no coexisting paralysis of the ipsilateral superior rectus and the atypical cases those with a significant paresis of that muscle. The simulated cases are those caused by any reason other than a congenitally short anterior tendon sheath of the superior oblique. Brown further classified the simulated group into spontaneous recovery cases, intermittent cases, and acquired cases. These cases might be a result of inflammation, trauma, or surgery to the eye. Also considered under the heading of simulated Brown's syndrome are those cases of congenital etiology differing from that of a defectively short anterior tendon sheath of the superior oblique.

The definitive diagnosis of superior oblique tendon sheath syndrome involves eye movement testing, cover testing,

Hess-Lancaster testing, range of binocularity testing, and forced duction testing. Surgical intervention is currently the only form of treatment for true Brown's syndrome and should be considered solely in cases where there exists a disfiguring head tilt and/or hypotropia of the affected eye in primary gaze.

The discussion which follows explains in further detail the clinical features, etiology, diagnosis, treatment and prognosis of Brown's syndrome while highlighting current theories and recent research in the still controversial areas of etiology and treatment of this syndrome.

CLINICAL FEATURES

The following characteristic findings of superior oblique tendon sheath syndrome are universally accepted as pathognomonic:[4,19,21]

1) severely limited elevation in adduction of the affected eye (ususally not above the mid-horizontal plane); the degree of limitation will be the same whether the movement be a duction, version, or passive rotation;

2) full elevation possible in abduction and straight up gaze;

3) normal or near normal muscle activity of the ipsilateral superior rectus muscle;

4) little, if any, overaction of the ipsilateral superior oblique (sometimes a slight downshoot of the involved eye may be noted);

5) usually, widening of the palpebral fissure on adduction;

6) "V" pattern exotropia in upward gaze;

7) compensatory backward head tilt;

8) infrequent hypotropia in primary gaze;

9) unequivocably positive forced duction test on attempted elevation in adduction;

10) usually unilateral but may be bilateral;

11) an audible or palpable (often painful) "click" in the trochlear region of the adducted elevating eye (usually found in intermittent cases or those inflammatory in origin).

ETIOLOGY

The true etiology of superior oblique tendon sheath syndrome remains unclear and many etiologies have been suggested.

TRUE

Brown maintains that the true congenital anomaly is a mechanical problem due to an abnormally short anterior sheath of the superior oblique tendon. In 1950 he described the mechanism as follows:

> "The tendon sheath of the superior oblique, according to Whitnall, is fixed to and terminates at the pulley and its scleral insertion of the fused tendon and sheath, results, if the tendon is short, in restriction of elevation in the nasal field. The linear distance increases on adduction of the eye and decreases on abduction. Therefore, if the tendon sheath is taut when the eye is in primary position, adduction will be possible only as the eye is depressed. Normally this sheath acts as a check-ligament for the inferior oblique muscle."

Thus Brown proposed that the abnormally short sheath might be

secondary to either a congenital paralysis or to a delay in the function of the inferior oblique muscle. Unfortunately the relationship to inferior oblique paresis has not been supported by further clinical research.

SIMULATED

Brown's original concept regarding an anomalous short anterior tendon sheath of the superior oblique has not stood unchallenged, and other apparently congenital structural anomalies have been reported. Brown acknowledged these as causes for simulated Brown's syndrome. In 1973[4] he remarked that a congenitally thick area in the posterior tendon and possibly firm attachment of the sheath to the posterior tendon could also prevent movement of the tendon through the trochlea on adduction.

Parks and Brown[19] refuted the validity of Brown's original theory for the true syndrome, noting the widespread poor results of surgical weakening of the tendon sheath. Furthermore, in 25 patients studied, none showed a shortened or taut sheath. In most of the patients, limp tendons suggested the cause for the restriction might be adhesions of the muscle fasciae posterior and inferior to the globe. In two of their patients, however, the actual tendon, not the sheath, was taut and shortened.

Crawford[7,8] examined the results of tenotomies and tenectomies done on Brown's syndrome patients and concluded that the cause of the "true" syndrome must be due to a tight superior oblique tendon.

Scott and Knapp[24] concurred with Park and Brown's idea that a restrictive band posterior to the globe might be the cause of the inability to elevate the eye on adduction. They raised the question whether true Brown's could result from birth trauma to the orbital floor.

Raab[20] reported a case where exposure of the superior oblique tendon and sheath revealed an anomalous band of connective tissue from the anterior sheath to the sclera, thus suggesting yet a different etiological basis for the pathogenesis.

Parks[18] later concluded that the syndrome was problably due to a taut tendon and could be treated by tenectomy. He noted that the superior oblique tendon does not have a true sheath.

Sevel[25], in a search for an embryological explanation for Brown's syndrome, reviewed 54 embryos and fetuses for the development of the superior oblique muscle, trochlea, superior oblique muscle traversing the trochlea, the trochlea-tendinous connections and the musculo-tendinous junctions. His investigation reveals

> "fine trabeculae between the tendon and trochlea persist into adulthood and most likely act as tethering strands to control and limit the excursions of the tendon in the trochlea. No accumulation of mesenchymal or fibrous tissue was noted between the superior oblique muscle and tendon thus it is unlikely that this is the etiology of Brown's syndrome. It is postulated that persistence of the thickened embryological analage of these trabeculae could well account for the condition of Brown's syndrome."

Evidence of a hereditary factor in superior oblique tendon sheath syndrome is rare. Nonetheless, Katz, et al.[13] presented a case where Brown's syndrome was found in monozygotic twin girls. It was speculated that if genetics did not play a part in this instance, it was highly likely that very early embryological insult occurred, thus causing mirror imaged Brown's syndrome in the twins.

Weiss and Urist[31] reported a case of Brown's syndrome coincident with Duane's syndrome and microcornea.

Abnormal innervation has also been pursued as a possible etiology for Brown's syndrome. Electromyographical studies

reported by Catford and Hart show conflicting evidence. Breinin in 1957 revealed normal innervation to the inferior oblique in Brown's syndrome cases while Stein and Papst's research[5] implies that abnormal innervation to the superior and inferior oblique muscles might ultimately cause the inability to raise the adducted eye. Catford and Hart support the theory that Brown's syndrome is a mechanical anomaly and is not due to abnormal innervation.

ACQUIRED

Inflammation

Sandford-Smith[22,23] firmly believes that Brown's syndrome is due to stenosing tenosynovitis, where localized swelling at the trochlea associated with constrictive hypertrophy of the sheath prevents passage of the tendon through the trochlea. Mein[15] and Herman[11] show similar findings. It is postulated that this is the etiology in intermittent cases where occasionally the tendon swelling is reduced sufficiently to allow passage through the trochlea. Consequently if the swelling reduces sufficiently the patient may spontaneously recover.

Acquired inflammatory Brown's syndrome has also been reported as a consequence of rheumatoid arthritis.[1,14,17,26,30] The clinical features of extreme tenderness and swelling in the area of the trochlea usually improve with administration of anti-inflammatory steroids if the condition does not spontaneously resolve.

Helveston,et al[10] suggested that local inflammation in the trochlear area may result in fluid accummulation in the bursa between the sheath and trochlea. Also vascular engorgement in the sheath itself may occur, both of which might limit movement of the tendon through the trochlea.

Post-surgery

The condition known as Brown's syndrome has also been related to post-surgical trauma. Among those cases reported are: tucking of the superior oblique[15], ethmoidal and frontal sinus surgery[2], blepharoplasty[32], and scleral buckling procedure. Wright et al[33], in a clinicopathologic study of acquired inflammatory superior oblique tendon sheath syndrome, noted that perisheath scarring following trauma, sinusitis or surgery can manifest Brown's syndrome.

Trauma

Zipf and Trokel[34] presented two cases where Brown's syndrome was simulated following orbital floor fracture. Jackson et al[12] discussed another case where an orbital floor fracture with entrapment, edema, and hemorrhage were masked by simulated Brown's syndrome.

Quite a variety of research exists with respect to the etiology of superior oblique tendon sheath syndrome. Brown believes at least 20% of the cases are of the simulated type[4]. Thus one cannot assume one cause for a patient with an inability to raise the adducted eye and a positive forced duction test. Each case must be investigated very carefully.

DIFFERENTIAL DIAGNOSIS

Eye movement testing, whether saccadic fixations, ductions, or versions, will reveal an inability to elevate the adducted eye with a slight tendency for downshoot in the affected eye. The patient may attempt a compensatory torticollis by tilting his head back. A slow and gradual increase of elevation in abduction is also observed. Diplopia in upward gaze is a common subjective symptom.

Unilateral and alternate cover testing may reveal either orthotropia or hypotropia of the affected eye in the primary position of gaze. Ususally a "V" pattern exodeviation can be observed (and in inferior oblique palsies an "A" phenomenon is usually noted[16]). Rarely, an excyclodeviation is also involved. If the patient has adapted to the condition with an abnormal head position, the cover test should also be performed in the habitually tilted position to determine if orthotropia is possible.

The Hess-Lancaster test usually identifies an underaction of the inferior oblique and a coexisting overaction of the superior rectus. The Park Three Step Method may be used to verify the anomaly.

The definitve diagnostic test used to determine the superior oblique tendon as the source of the mobility defect is the forced duction test. It will separate an inferior oblique paresis from a superior oblique tendon sheath problem and should be considered conclusive. The test is performed under general or topical anesthesia, and the procedure is as follows:[28]

> "The eye is grasped near the limbus with forceps and moved in the direction opposite that in which the mechanical restriction is suspected."

In the case of Brown's syndrome, a positive forced duction test reveals the eye resisting passive supraduction in adduction. If there were an inferior oblique paresis, supraduction would be produced upon adduction.

Electro-oculography measurements of vertical saccadic velocities in Brown syndrome patients reveal no reduction in velocities on adduction. Metz[16] believes this differentiates Brown's syndrome from an inferior oblique palsy where the vertical saccades in

adduction are slowed.

The range of single binocular vision is a very important assessment. A variety of tests can be utilized, for example, Worth-4-dot, Bagolini lenses, red/green anaglyphs or polaroid vectograms, and the major amblyoscope or the troposcope. It is crucial to determine if fusion can be utilized, and if so, to what extent.

TREATMENT

Surgical intervention for congenital (true and simulated) Brown's syndrome is indicated when:[9]

1) a disfiguring ocular torticollis is necessary in order to maintain an adequate field of single binocular vision,

2) hypotropia and no stereopsis in primary position of gaze exists, or

3) a manifest deviation has developed but evidence of binocular function is still present.

The type of surgical treatment varies with differing etiologies. The true etiology can not be determined until surgical examination of the superior oblique tendon, sheath and trochlea has been accomplished. Therefore true diagnosis as well as choice of treatment for that cause is undertaken at the time of surgery.

Since the syndrome was first described in 1950, several surgical procedures have been used to correct the motility defect. One of these, stripping of the tendon sheath with or without postoperative traction sutures have been performed.[3,4,7,8,24] Unfortunately the results were unreliable and in most cases did not cure the problem.

Crawford[7,8] investigated superior oblique tendon weakening procedures. By carrying out either a Z tenotomy, split tendon lengthening, complete tenotomy, or tenectomy in patients with Brown's syndrome, he concluded that tenotomy (cutting the tendon just medial to the superior rectus muscle) provides the best results.

Parks and Brown[19] found that, of the 25 patients in their study mentioned previously, tenotomy or tenectomy worked on two of their patients with taut superior oblique tendons.

VonNoorden and Olivier[29] recently performed tenectomies of the superior oblique tendon and sheath on 12 patients and found that post-operative motility in elevation and adduction was normal. Unfortunately, they found that additional surgery was necessary after several months due to superior oblique palsy. They, along with Crawford[7,8], feel that secondary surgery is indeed necessary in order to provide excellent permanent results. In most cases a myectomy of the inferior oblique solves the problem, but numerous other procedures may be necessary including recession of the contralateral inferior rectus or contralateral superior rectus.

If inflammatory acquired simulated Brown's syndrome is diagnosed, steroid therapy may help reduce the swelling[9,17,22,23]. Evacuation of pus and systemic antibiotics may be necessary to treat the actual inflammation.

If Brown's syndrome is caused by some sort of trauma, that specific cause must be remedied.[2,12,26,32]

If non-inflammatory in cause, and congenital Brown's syndrome has been ruled out, spontaneous recovery may eliminate the need for tenotomy. Mein[15] claims some writers advocate strenuous exercise of the eye and possibly repitition of the forced duction test daily

to cure intermittent cases. Orthoptics may prevent the need for a cosmetically unacceptable head position, suppression, or even amblyopia. Prism and occluder therapy is initiated to help toe diplopia. Monocular skills such as motilities, convergence and accommodation are strengthened. Then remedial and enhancement binocularity/stereofusion training is incorporated to strengthen what fusion the patient may have.

PROGNOSIS

Without surgery, the cure of single binocular vision in all fields of gaze without cosmesis problems is practically impossible. The author has found no research done on the effects of orthoptic training on Brown's syndrome. However, Clark and Noel[16] speculate

> "one wonders why a child would assume a head position (chin elevation and face turn) appropriate for Brown's syndrome but fail to demonstrate fusion. The anomalous head position suggests that at some point the child enjoyed binocular single vision but that with the passage of time this facility was lost."

It is the author's opinion that visual training, although not capable of producing a cure, is indeed necessary for preventative and visual enhancement measures, and should not be ignored when dealing with the Brown's syndrome patient.

Clarke and Noel classified 28 children with diagnosed true Brown's syndrome into four groups: 1)Those with uncomplicated Brown's syndrome; ie. no anomalous head tilt, and some fusion in forced primary position of gaze. 2)Those with fusion in the assumed head position and hypotropia in forced primary gaze. 3)Those with fusion in primary gaze but also intermittent hypotropia and large angle exotropia. 4)Those with no fusion even in the assumed head position. They found those in group 1 with no head tilt maintained orthophoria or minimal heterophoria and full stereopsis (titmus stereotest) in primary position of gaze. These

were not considered for surgery. Those in group 2 underwent surgery and showed marginal improvement. Group 3, after surgery, could elicit full fusion and steropsis but showed some remaining intermittent exotropia or hypotropia. Those in group 4 following surgery still showed a lack of fusion and stereopsis, and an anomalous head tilt, vertical and horizontal deviation and several were amblyopic.

A longitudinal study of the effects of tenotomy on 28 patients after several years was done by Crawford et al[17]. Their results are grouped as follows: 1) Those patients with excellent single binocular vision preoperatively who maintained it after surgery. 2) Those patients with partial residual Brown's syndrome postoperatively which gradually improved spontaneously over a period of several years. 3) Those patients who had minimal or moderate superior oblique paresis, secondary overaction of the ipsilateral inferior oblique and excellent binocular single vision who improved spontaneously. 4) Those patients with marked superior oblique paresis and secondary ipsilateral inferior oblique overaction who did not improve spontaneously and required inferior oblique plication. They suggest not only assessing patients immediately after surgery but also after an extended period of time.

In patients with inflammatory acquired Brown's syndrome, several authors[2,14,17] have found excellent prognoses with steroid therapy.

SUMMARY

Brown's superior oblique tendon sheath syndrome is an esteablished clinical identity which manifests the inability to elevate the adducted eye either actively or passively. It is currently recognized that the defect may originate from several causes. Among these are congenital structural anomalies, stenosing

tenosynovitis, rheumatoid arthritis, trauma, and infection to the orbit. The key differentiation between Brown's syndrome and an inferior oblique paresis is the positive forced duction test result in Brown's syndrome. Today the widely accepted treatment is firstly tenotomy or tenectomy followed by secondary surgery to other overacting or underacting extraocular muscles which may become evident several months postoperatively. The prognosis for excellent binocular vision following surgical intervention is guarded. However, inflammatory Brown's syndrome can usually be successfully managed with steroid therapy.

TABLE I: CLASSIFICATION OF BROWN'S SYNDROME

TRUE
congenital short anterior sheath of the superior oblique tendon

TYPICAL
No coexisting paralysis of ipsilateral superior rectus

ATYPICAL
Coexisting paralysis of the ipsilateral superior rectus

ACQUIRED
trauma
post-surgical inflammation

INTERMITTENT
signs of the syndrome are present only occasionally

SPONTANEOUS RECOVERY
those cases which disappear without any treatment

SIMULATED
all other etiologies including other congenital possibilities

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