Eye (2017) 31, 148–151

www.nature.com/eye

CLINICAL STUDY

Augmented vertical recti transposition with intraoperative botulinum toxin for complete and chronic sixth nerve palsy

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Abstract

Purpose **To evaluate the results of augmented vertical rectus muscle transposition (VRT) with intraoperative botulinum toxin (BTX) for complete and chronic sixth nerve palsy.**

Methods **During a 10-year period (2004–2014) all patients with chronic and complete sixth nerve palsy and contracted medial rectus (MR) who underwent augmented VRT and BTX injection into the MR enrolled in this study.**

Results **In total, 29 patients (5 bilateral) were enrolled in this study. Preoperative deviation was 45 ± 17.5 Prism Diopter (PD), which was improved to -3.1 ± 13.2 after the operation ($P<0.001$). Mean preoperative and postoperative abduction limitation was -4.4 ± 1.1 and -1.8 ± 0.9, respectively ($P<0.001$). The success rate was 76% (deviation within 10 PD of orthotropia). Four patients (13.7%) had hypotropia. In 19 patients with preoperative deviation ≤ 45 PD, four patients had consecutive exotropia.**

Conclusion **Intraoperative BTX injection with augmented vertical rectus transposition is an effective procedure. In deviation ≤ 45 there is a risk of overcorrection.**

Eye (2017) **31,** 148–151; doi:10.1038/eye.2016.226; published online 4 November 2016

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Received: 28 March 2016
Accepted in revised form: 14 September 2016
Published online: 4 November 2016

Introduction

Patients with sixth nerve palsy present limitation of abduction, moderate-to-severe esotropia and diplopia in primary position, and usually abnormal head posture.[1] Various surgical procedures have been described the management of complete sixth nerve palsy, including partial and full vertical recti muscles transposition (VRT) to induce the abduction of the eye. Full VRT is among the most effective treatment for the management of the complete sixth nerve palsy.[2] Foster-introduced augmented VRT to enhancing abduction. In long-standing palsy, the medial rectus muscle (MR) is contractured and weakening of the MR may be required. Simultaneous operation on MR has the risk of anterior segment ischemia.[3] To avoid this complication, some authors advocated botulinum toxin (BTX) injection to the MR.[4–7]

The use of intraoperative BTX to weaken the antagonist MR in transposition procedures has been proposed by Scott.[8] Scott and Kraft postulated that BTX reduces contracture of the MR and allows for more complete recovery of LR muscle function.[9]

In this study, we describe the results of augmented transposition of vertical rectus muscles with intraoperative BTA in the management of chronic sixth nerve palsy.

Methods

The Medical Ethics Committee of Tabriz University of Medical Sciences approved this study, and informed consent was obtained from patients. During a 10-year period between 2004 and 2014, patients with complete and chronic sixth nerve palsy enrolled in this study. We defined complete palsy as deficient abduction beyond midline and chronic at least 6 months from the onset of palsy. Patients included in the analysis had at least 6 months follow-up. The senior author (RN) performed all surgeries.

The etiology of the palsy was classified at the time of enrollment as traumatic, congenital, neoplastic, vascular, and idiopathic. Abduction deficit was graded from −8 (globe immobilized in extreme adduction) to −4 (abduction to midline) to 0 (full abduction). A forced muscle

generation test confirmed the presence of complete sixth nerve palsy and forced duction testing confirmed the MR contracture. Patients must have MR contracture to be included in the study. The following data were collected and analyzed: age at enrollment, etiology, gender, involved eye, pre- and postoperative deviation, and abduction deficit and duration of follow-up. Patients with history of previous surgery or BTX injection and visual acuity of $<20/200$ were excluded. Angle of deviation was measured by simultaneous prism and cover tests in the primary position at distance fixation. Augmented vertical rectus muscles transposition was performed according to the technique described by Foster.[2] In previous studies, 2.5–5 IU of Botox was injected to MR.[4–7] After the completion of transposition, 12.5 IU of BTX A (Dysport, Ipsen, Berkshire, UK) were injected into MR muscle (3 units of Dysport is equivalent to 1 unit of Botox).[8] Chloramphenicol eye drops were prescribed for 1 week and betamethason eye drops were prescribed for 2 weeks. Motility and complete eye examination, including signs and symptoms of anterior segment ischemia, were performed regularly. The SPSS 17 and the Kolmogorov–Smirnov test were used for descriptive data, and pre-and postoperative results were analyzed by the Wilcoxon signed-rank test. *P*-values of ≤ 0.5 were considered significant. Postoperative alignment within 10 prism diopters (PD) of orthotropia was defined as success.

Results

In total, 29 patients (24 unilateral and 5 bilateral) with sixth nerve palsy enrolled in the study, of which 15 of the patients were male (51.71) and 14 (49.3%) were female. Etiology and laterality are summarized in Table 1. The most common cause of sixth nerve palsy in this study was trauma (48.1%), followed by congenital causes (31%). The pre- and postoperative data are summarized in Table 2.

The mean age was 26.5 ± 19.8 years (range 1–62 years). Follow-up period was 24.2 ± 24.3 months (range 6–91 months). Twenty-two (76%) patients were within 10 PD of orthophoria, and the rates of undercorrection and overcorrection were 3 (10.3%) and 4 (13.7%), respectively. The mean preoperative deviation was 45 PD that reduced to -3 (exotropia shift) postoperatively ($P<0.001$). The effect of operation was 48.1 ± 17.7 PD (range 12–90). In 3 patients with under correction, preoperative ET were 90, 60 and 30 PD, and postoperative ET were 30, 15, and 18, respectively. In overcorrected patients, preoperative ET were 45, 40, 25, and 20 PD with postoperative results of 18, 20, 40, and 35 PD of XT in primary position. In 19 patients, preoperative ET were ≤ 45, in which 4 patients (21%) had more than 10 PD XT after operation. Two of these 4 patients had limited adduction of -1 and -2. In 5 patients (16.9%), we observed postoperative limitation of adduction ranging from -1 to -2. All of the patients with consecutive XT underwent reoperation and achieved orthotropia. In three patients, we performed unilateral or bilateral MR resection and in one patient LR recession and MR resection. In two patients with consecutive XT and limitation of adduction, MR resection resulted in full adduction. None of these 4 patients developed anterior segment ischemia. Four patients (13.7%) had postoperative hypotropia of 30, 20, 10, and 8 PD. The patient with 30 PD hypotropia underwent 2 operations on transposed IR that diminished hypotropia to 10 PD. The other 3 patients with hypotropia refused reoperation.

Preoperation abduction limitation was -4.4 ± 1.1 (range -4 to -8), and that improved to -1.8 ± 0.9 (range 0 to -4), which was statistically significant ($P<0.001$).

We compared congenital and traumatic cases. The results were marginally different between the 2 groups and this difference was not statistically different (Table 3).

Table 1 Etiology, laterality and involved eye

	Unilateral	*Bilateral*	*OD*	*OS*
Trauma	12	2	8	8
Congenital	8	1	4	6
Neoplasia	1	—	—	1
Vascular	1	—	—	1
Convulsion	—	2	2	2
Unknown	2	—	—	2

Discussion

In complete sixth nerve palsy, when the globe cannot be excursed beyond midline, transposition procedure is indicated. Various transposition procedures have been described for management of sixth nerve palsy. The goals of surgery are to improve eye alignment in primary position, increase the binocular diplopia-free field and improve abduction deficit. Full-tendon VRT was first described by Schillinger[10] in 1959. In 1997, Foster[2] introduced augmented VTR to enhance abducting force. MRI evaluation of this technique shows increased lateralization of the transposed muscles. In chronic sixth palsy, passive abduction may be limited secondary to MR contracture.

To improve abduction, MR contracture must be alleviated. Considering the risk of anterior segment ischemia with simultaneous MR recession, we used intraoperative BTX injection of MR.

In our study, the most common cause of palsy was trauma, which was compatible with other studies.[4,11] Our success rate was 76%. Armenia,[7] Fitzsimons,[5] and Leiba *et al*[12] performed unaugmented VRT with intraoperative

Table 2 Pre-and postoperative data

Age	*Deviation*		*LR underaction*		*Postop MR underaction FU*	
(26.5 ± 19.8)	Preop	Postop	Preop	Postop	5 (16.9%)	—
	45 ± 17.5	−3.1 ± 13.2[a]	−4.4 ± 1.1	−1.8 ± 0.9	−0.2 ± 0.4	24.2 ± 24.3
	(20 to 90)	—	(−8 to −4)	(0 to −4)	(range, −1 to −2)	(6 to 91mo)
Effect:	48.1 ± 17.7 (12–90)					
Success:	22 (76%)					
Undercorrection:	3 (10.3%)					
Overcorrection	4 (13.7%)					

[a]Negative post-operative deviation referred to consecutive exotropia.

Table 3 Comparing traumatic and congenital 6th nerve palsy

	Traumatic	*Congenial*	P-*value*[a]
Preop deviation	48.3 ± 18.5	45 ± 21.9	0.48
Postop deviation	−0.67 ± 11.20[b]	−2.66 ± 15.3	0.95
Preop LR underaction	−4.8 ± 1.5	−4.1 ± 0.3	0.35
Postop LR underaction	−1.7 ± 0.9	−1.6 ± 1.1	0.82
Postop adduction deficiency	−0.06 ± 0.2	−0.1 ± 0.3	0.86

[a]Mann–Whitney Test. [b]Negative postoperative deviation referred to consecutive exotropia.

BTA and gained 60, 50, and 59% success rates, respectively.

In the present study, the mean final reduction in esotropic deviation was 48 PD. In previous studies that used unaugmented VRT with BTX, exotropic shift was between 32 and 66 PD, however, some authors have included patients with previous surgery.[4–7,12,13] In the current study, mean abduction deficit improved from −4.4 to −1.8, which seems to be slightly better than most of previously-mentioned transposition procedures.[4–7,11] Four (13.7%) of our patients were overcorrected. In all these patients, preoperation esotropia was ≤45. We assessed LR rectus function by office force generation test, which is a qualitative test. Saccadic velocity assessment may be the best way to confirm the palsy. Suggested laboratory saccadic velocity tests are electro-oculograghy, infrared scleral reflection technique, and scleral search coil technique. The latter technique is regarded as the most sensitive method.[14] Our overcorrected cases may have had some residual LR activity, and if we could use above mentioned techniques, some of these cases may be excluded as having complete sixth nerve palsy. Because of limited cost and availability, they have not been widely used and this was applied to our patients. In 1997, Foster[2] reported that augmentation of VRT significantly increased tonic abducting force of the transposition without decreasing adduction. In a few patients, stiffness of MR decreased; and in most patients, MR weakening was not needed. According to our results, we suggest that in preoperation deviation of <45, it is reasonable to perform augmented VRT without BTX to MR. We observed limitation of adduction in 5 patients (−1 to −2), and 3 of these patients were among overcorrected cases. In all 4 patients with consecutive XT, unilateral or bilateral MR resection was performed, which leads to orthotropia and improvement of adduction deficit. We did not observe anterior segment ischemia in these patients.

We also compared the results of congenital and traumatic patients. The results are comparable without any statistically significant difference between the two groups.

One of the most important and challenging complications of VRT is the undesired vertical deviation, which has been reported between 5% and 30% of the time. Four of our patients had hypotropia ranging from 8 to 30 PD.

The reason of this complication is not well understood. Laby and Rosenbaum[15] believed that differences in tension between nasal and temporal portions of the new insertion causes vertical deviation. Holmes *et al*[16] hypothesized that undesirable asymmetry of superior rectus (SR) and inferior rectus (IR) leads to relatively tight IR with resultant hypotropia. Also, anatomical differences between SR and IR may have a role. Some methods suggested to prevent this complication include adjustable suture technique,[11] intraoperative monitoring of torsion,[16] and modification of Foster technique using single posterior fixation suture.[17] Ruth *et al*[18] performed vertical forced ductions at the end of procedures to ensure there are no vertical restrictions. They managed induced hypotropia by releasing scleral suture and then recessing IR until the forced duction became negative, then posterior fixation suture replaced 8 mm posterior to LR insertion along the inferior and superior lateral border of IR. A patient with induced hypotropia of 30 PD underwent two consecutive corrective surgeries, according to the method described above, which reduced hypotropia to 10 PD. Three other patients with induced

hypotropia declined corrective surgery. Recently, Holmes *et al*[16] suggested intraoperative monitoring of torsion to reduce the rate of induced vertical deviation after VRT. To the best of our knowledge, our study is the first to report of long-term results of augmented VRT with intraoperative BTX.

Conclusion

Augmented VRT with intraoperative BTX injection to MR has a high success rate and favorably improves abduction deficit, especially in severe forms of LR palsy.

Summary

What was known before

- Complete sixth nerve palsy needs transposition of vertical recti toward LR.
- Some advocated injection of Botulinum toxin to MR (intraoperative or postoperation).

What this study adds

- We performed augmented vertical recti transposition with intraoperative injection of botulinum toxin to MR (to the best of our knowledge there was no published study about this technique).
- The success rate of this technique is promising, especially in improving abduction deficiency.
- The most important disadvantages of this technique are induced hypotropia, and overcorrection in moderate angle deviation.

Conflict of interest

The authors declare no conflict of interest.

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