Are Most Sixth Nerve Palsies Really Paralytic?

Ayse Gul Altintas, MD; Hasan Basri Arifoglu, MD; Derya Dal, MD; Saban Simsek, MD

ABSTRACT

Background: Etiology and date of palsy are two important parameters that affect the treatment protocol of sixth nerve palsy. This study evaluated the treatment protocols and outcomes of treatment in sixth nerve palsies.

Methods: Thirty-four patients who had sixth nerve palsy were included. Botulinum toxin A (BTX) injection was performed on patients with acute sixth nerve palsy and paresis (BTX group), whereas chronic cases received only horizontal surgery (surgery group). All patients in the BTX group received a BTX injection into the ipsilateral medial rectus muscle. Patients in the surgery group underwent either ipsilateral medial rectus muscle recession or recession combined with lateral rectus resection without the transposition procedure.

Results: Fifteen patients were treated with a BTX injection to the medial rectus muscle. One patient underwent ipsilateral medial rectus muscle recession and 6 patients received both medial rectus recession and lateral rectus resection in the same session. Measurement of esotropia was 24.9 prism diopters (PD) (range: 18 to 35 PD) before treatment in the BTX group. The recovery rate was 86.6% (13 of 15) without any residual deviation. In the surgery group, the mean preoperative deviation was 35.1 PD (range: 14 to 75 PD), which decreased to mean 2.57 PD (range: 0 to 10 PD) postoperatively. The achievement of orthotropia rate was 85.7%.

Conclusion: BTX injection was found to be an effective treatment because it prevented medial rectus contraction in acute sixth nerve palsies. Correction of deviation with the recession of contracted medial rectus muscles and resection of lateral rectus muscles without the need of transposition in chronic sixth nerve palsy testified that most sixth nerve palsies involve partial paralysis rather than complete paralysis.

INTRODUCTION

Sixth nerve palsies caused by different etiologies have different prognoses. Clinical management is influenced by the expected rate of spontaneous recovery, which is high in acute sixth nerve palsies due to microvasculopathy, such as diabetes mellitus and hypertension. For acute traumatic sixth nerve palsies, the spontaneous recovery rate has been reported to be lower than in cases with microvasculopathy. The severity and the laterality are also important factors affecting final prognosis. Patients with bilateral and complete palsies have less chance for recovery than those with paresis, which explains partial deficits. The neuromuscular sequelae of complete unrecovered or persistent sixth nerve palsies developed contracture on the unopposed medial rectus muscle in a few months, which causes esodeviation and compensatory head position in some cases. This contracture of the medial rectus muscle may prevent complete resolution of esodeviation and head posture despite complete recovery of lateral rectus function. Esodeviation may be corrected by preventing medial rectus muscle contracture or releasing the contracting medial rectus muscle.
In this study, we compared the final outcome in patients who received botulinum toxin A (BTX) to treat sixth nerve palsy during the acute phase and horizontal muscle surgery in cases with unrecoverable chronic sixth nerve palsy.

**PATIENTS AND METHODS**

We included a total of 34 patients with a mean age of 50.2 years (range: 6 months to 80 years) who had sixth nerve palsy in this study. Data, including age, sex, date of palsy, presence of trauma or microvasculopathy (such as diabetes mellitus and hypertension), systemic condition, degree of abduction deficit, angle of deviation, presence of diplopia, and compensatory head position, were examined. All patients underwent complete ophthalmic examination including a force duction test.

To evaluate the degree of paresis and medial rectus contracture, abduction deficits were graded from 0 to -6 according to the scale described by Scott and Kraft: 0 = full abduction; -1 = can rotate eye from midline to 75% full rotation; -2 = to 50% full rotation; -3 = to 25% full rotation; -4 = no abduction beyond the midline; -5 = inability to abduct to the midline; -6 = abduction that is only 15° nasal to the midline.1,4-6,7 The angle of deviation was measured by simultaneous prism and alternate cover test in the primary position at both near and distance fixation. Patients were classified into two treatment groups according to the time interval between the onset of sixth nerve palsy and the enrollment visit. Chronic sixth nerve palsy and paresis was defined as more than a 6-month duration since the onset of pathology, whereas the acute phase was defined as less than a 6-month duration period.

A BTX injection was performed on patients with acute sixth nerve palsy and paresis (BTX group), whereas chronic cases received surgery (surgery group). All patients in the BTX group received a BTX injection into the ipsilateral medial rectus muscle while under topical anesthesia. Doses ranged from 2.5 to 5 units (U), which were determined by the size of deviation and abduction deficiency. A total of 5 U of BTX was used in cases with a -4 to -6 abduction deficit and 2.5 U of BTX was injected in cases with a -2 to -3 deficiency.

Patients in the surgery group underwent either ipsilateral medial rectus recession or recession combined with lateral rectus resection with intraoperative adjustable surgery technique. Intraoperative adjustable surgery was performed under both topical and anterior subconjunctival anesthesia in all patients.

Recovery was defined as the absence of head position and diplopia equal to 10 prism diopters (PD) or less distance esotropia in primary position and improved duction and version in the field of lateral rectus action at least 6 months after intervention.

**RESULTS**

The etiology of the sixth nerve palsy is given in the table. Fifteen patients were treated with a BTX injection into the medial rectus muscle in the eye with acute sixth nerve palsy. Measurement of the esotropia was 18 to 35 PD with an average of 24.9 PD before treatment in the BTX group. The mean esodeviation was 1.16 PD (range: 0 to 8 PD) in the posttreatment period.

Thirteen patients were orthotropic 1 week after the BTX injection and complete recovery of duction and version motility was determined by version testing. Head position and diplopia disappeared. It was even considered a success when two patients had minimal residual esotropia. One patient had 6 PD and the other had 8 PD 1 week after treatment, and both had microvascular etiology. Both patients adopted a small compensatory face turn to alleviate diplopia, which was gone at the second visit. No one required prismatic correction (Fig. 1). Two patients had transient minimal ptosis without pupillary occlusion, which went away in 1 week. Only one pa-

<table>
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<th>Variable</th>
<th>BTX</th>
<th>Surgery</th>
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<tr>
<td><strong>Etiology</strong></td>
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<tr>
<td>Microvasculopathy</td>
<td>13</td>
<td>3</td>
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<tr>
<td>Trauma</td>
<td>2</td>
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<td><strong>Interval between palsy onset and treatment (d)</strong></td>
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<tr>
<td>Range</td>
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<td><strong>Age (y)</strong></td>
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<tr>
<td>Female</td>
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BTX = botulinum toxin A.
tient received a second injection 6 months after the first treatment when the effect of BTX wore off. No patient needed more than two injections in all follow-up periods after the 6-month follow-up.

The recovery rate was 86.6% (13 of 15) without any residual deviation and 100% with two patients having 6 and 8 PD of residual deviation (Fig. 1). In the surgery group, the mean preoperative deviation was 35.1 PD (range: 14 to 75 PD), which decreased to mean 2.57 PD (range: 0 to 10 PD) postoperatively.

One patient underwent ipsilateral medial rectus muscle recession and 6 patients received both medial rectus recession and lateral rectus resection in the same session for chronic sixth nerve palsy (Fig. 2). We tried to perform the force duction test preoperatively, but because of significant medial rectus muscle contracture we could not distinguish whether the impairment of the lateral rectus muscle was either partial or total. Therefore, the final amount of manipulation, either medial rectus recession only or a combination of medial rectus recession with ipsilateral lateral rectus resection, was decided intraoperatively. The muscle functions and deviations were evaluated by simultaneous prism and alternate cover test intraoperatively with the help of intact muscle function due to anterior subconjunctival anesthesia (Fig. 3).

Five patients had orthotropia and one had residual deviation less than 10 PD. The success rate of achievement to orthotropia was 85.7%. One patient had 10 PD residual esodeviation in distance fixation without complaining of diplopia. None of our patients had more residual deviation. Therefore, no one needed BTX injection to the medial rectus muscle after surgery for residual deviation.

Three patients with acute sixth nerve palsy without treatment did not return to the hospital for treatment or follow-up. Therefore, we did not have any information about whether their acute palsies recovered on their own.

DISCUSSION

Patients with sixth nerve palsy mainly caused by microvasculopathy, such as diabetes mellitus and hypertension, are usually observed for a period of 6 months to allow for spontaneous recovery. To alleviate symptomatic diplopia and compensatory head posture, a patch over one eye or prism glasses can be used for relatively small deviation while waiting for recovery. Both of these are not acceptable cosmetically and have no permanent therapeutic effect.

Patients with traumatic sixth nerve palsy that had a low spontaneous recovery rate and recovery
period required a longer recovery period than a patient who had microvascular pathology that increased the risk of medial rectus contracture. Early BTX injection not only eliminates symptoms and improves the patient’s quality of life, but also reduces contracture of the ipsilateral medial rectus muscle, therefore allowing for complete recovery of lateral rectus muscle function. Metz and Dickey reported that 93% of patients with sixth nerve palsy due to different etiology did not require surgery and 76% had complete recovery with BTX injection. Holmes et al. found a 73% recovery rate in traumatic sixth nerve palsy with BTX injection. Hung et al. reported a recovery rate of 64.3% in traumatic sixth nerve palsy with BTX injection, but only a 26.3% recovery rate in the conservative treatment group with the same etiology. We found an 86.6% recovery rate with BTX injection in sixth nerve palsies due to different etiology.

BTX pharmacologically denervates the antagonistic medial rectus muscle and prevents contracture of that muscle. As the paralytic lateral rectus muscle recovers, a BTX injection helps to achieve a slack medial rectus muscle without contracture, which allows for the restoration of alignment mainly in the primary position with a marked improvement in abduction. BTX treatment in sixth nerve palsy is considered a safe and effective alternative treatment to surgery in most patients. We did not have a conservative observation group because we prefer to treat all patients in an ethical and medical manner either with a BTX injection in acute palsies within 3 months of onset or surgery at least 6 months after onset.

Three of 12 patients who had acute sixth nerve palsy had not received any treatment and did not return for follow-up. Holmes et al. assumed that all such patients failed to return for treatment or follow-up because their symptoms resolved on their own. According to Holmes et al., assumption of the recovery rate would be 76% in the BTX group in traumatic sixth nerve palsy, which is similar to our result. We prefer the recession–resection procedure without the need of transposition surgery. Holmes et al. reported that the success rate was 7 (50%) of the 14 recession–resection procedures without the intraoperative adjustable suture technique to correct deviation and ocular motility. This high success rate in horizontal surgery without the need of transposition surgery may also testify that horizontal deviation in chronic sixth nerve palsy is due to the contraction of the medial rectus muscle but not persisting in sixth nerve palsy.

We did not analyze our results statistically because our study was limited by small numbers and it was neither randomized nor prospective. According to the high success rate of both the BTX group in early stage sixth nerve palsy and the surgery group with recession–resection procedure without the need of transposition surgery in chronic sixth nerve palsy, we believe that paretic cases are more frequent than the true paralytic cases. This finding may be important in guiding the clinician regarding correct diagnosis and in choosing therapeutic modalities.

**REFERENCES**