OnabotulinumtoxinA Injection as an Adjunct in the Treatment of Posterior Shoulder Subluxation in Neonatal Brachial Plexus Palsy

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OnabotulinumtoxinA Injection as an Adjunct in the Treatment of Posterior Shoulder Subluxation in Neonatal Brachial Plexus Palsy

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Background: Botulinum toxin A is used to treat contractures in children with spasticity by temporarily interfering with neural transmission at the motor end plate. In infants with brachial plexus palsy, posterior shoulder subluxation and dislocation are the result of muscle imbalance, in which neurologic recovery is evolving, and spasticity is not a deforming force. We postulated that temporary weakening of the shoulder internal rotator muscles with botulinum toxin A would facilitate reduction of the glenohumeral joint in such infants with early posterior shoulder subluxation or dislocation.

Methods: Thirty-five infants with posterior subluxation or dislocation of the shoulder due to brachial plexus palsy were treated with botulinum toxin A between January 1999 and December 2006, and were followed for a minimum period of one year. Records were reviewed for the severity of the palsy, age at time of treatment, recurrence of subluxation or dislocation, and the subsequent need for further treatment to reduce the glenohumeral joint.

Results: The average age at the time of shoulder reduction and botulinum toxin-A injection was 5.7 months. Six patients had a second injection. Reduction of the shoulder was maintained in twenty-four (69%) of the thirty-five patients. There were no complications related to the use of botulinum toxin A.

Conclusions: Although there may be specific risks associated with its use, botulinum toxin-A injection into the internal rotator muscles is a useful adjunct to the treatment of early posterior subluxation or dislocation of the shoulder in infants with neonatal brachial plexus palsy, and may help to avoid the need for open surgical procedures to restore or maintain shoulder reduction.

Level of Evidence: Therapeutic Level IV. See Instructions to Authors for a complete description of levels of evidence.

Loss of passive external rotation and posterior subluxation or dislocation of the shoulder are well-recognized results of muscle imbalance between the weak external rotator and the strong internal rotator muscles of the shoulder in infants with neonatal brachial plexus palsy[1-6]. Maintenance of glenohumeral congruency and prevention of permanent dysplastic changes during neurologic recovery are important goals of early treatment in these infants. These dysplastic changes in the shoulder can occur as early as three to six months after birth[6,7]. Loss of passive external rotation beyond neutral, when accompanied by posterior subluxation of the humeral head, with or without residual articular contact on imaging studies is an indication to reduce the glenohumeral joint and restore passive external rotation of the shoulder. *Subluxation* is the term used if there is some contact between the posterior aspect of the glenoid and the humeral head. *Dislocation* refers to the position of the humeral head posterior to the articular surface of the glenoid and the posterior aspect of the labrum.

Treatment of late posterior dislocation of the shoulder in neonatal brachial plexus palsy includes release of contractures,

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open reduction of the humeral head, and rebalancing tendon transfers in a young child without fixed dysplastic changes[8-10]. The older child with an established dysplastic glenohumeral joint or fixed dislocation may benefit from an external rotation osteotomy of the humerus[11]. Prevention of this dysplastic deformity of the shoulder depends on early detection of the posterior subluxation or dislocation followed by reduction of the glenohumeral joint.

Botulinum toxin type A has been used off-label in children with cerebral palsy, and as an adjunct to surgical treatment of neonatal brachial plexus palsy[12-14]. Its use is based on the peripheral inhibition of the release of acetylcholine at the presynaptic neuromuscular junction, temporarily weakening the force of muscle contraction. It is effective in the temporary improvement of function in the lower limbs of children with spastic cerebral palsy[15-19] and may improve function in the upper limbs of some of these children as well[20-22].

The safety of botulinum toxin A has been questioned recently by reports of complications, including death, following injection. In the spring of 2009, the United States Food and Drug Administration (FDA) issued directives about these safety issues and stated what information must be provided to the patient or family as part of obtaining informed consent for the use of botulinum toxin A. The complications associated with the injection of botulinum toxin A have occurred with large doses, multiple sites of injection, and in patients with comorbidities. The lack of bioequivalence of the various proprietary preparations of botulinum toxin A was also found to pose a risk to the patient. The FDA specified new names to distinguish the preparation and strength of the various forms of botulinum toxin A on the market. The use of botulinum toxin A in the treatment of spasticity related to cerebral palsy, or its use in children, has not yet been approved by the FDA, despite extensive experience and reports in the literature[12-22]. Up-to-date information is available on the FDA web site: www.fda.gov/Drugs/DrugSafety/PostmarketDrugSafetyInformationforPatientsandProviders/DrugSafetyInformationforHeathcareProfessionals/ucm174959.htm.

We postulated that the adjunctive use of onabotulinumtoxinA injection into the tight internal rotator muscles in infants with posterior shoulder subluxation or dislocation would facilitate maintenance of reduction of these shoulders with standard therapy and immobilization methods. The present study describes the outcomes of the treatment of the at-risk shoulder in patients with neonatal brachial plexus palsy with closed reduction and cast immobilization and supplemental onabotulinumtoxinA injection into the internal rotator muscles. All patients were treated by the senior author (M.E.).

Materials and Methods

With approval of the institutional review board of the University of Texas Southwestern Medical Center, we reviewed medical records to identify infants with brachial plexus palsy treated with closed reduction of the shoulder and injection of botulinum toxin A (onabotulinumtoxinA) into the shoulder internal rotator muscles. During the period from January 1999 to December 2006, forty-two patients met these criteria; seven patients were excluded because they had been followed for less than one year after treatment despite attempts to contact the families, leaving thirty-five patients in this series. All patients were treated with a standard home-exercise program focused on maintaining passive external rotation and full range of motion of the glenohumeral joint prior to injection of botulinum toxin A unless the diagnosis of shoulder subluxation or dislocation was made at the initial evaluation.

Surgical Technique

All infants underwent closed manipulative shoulder reduction. Following examination to determine the degree of contracture and whether the shoulder could be reduced after muscle relaxation, a slow steady manipulation was done until the shoulder could be brought into a fully reduced position and had a minimum of 60° of external rotation. The specific method of manipulation was done with the scapula manually stabilized, the shoulder maximally adducted to selectively stretch the subscapularis muscle, the elbow flexed >90° to stabilize the elbow collateral ligaments, and the forearm fully supinated to stretch the interosseous membrane and protect the radial head from dislocation. If passive external rotation of ≥60° and palpable forward translation of the humeral head could not be achieved, surgical release of contractures was done, and those patients were not included in this study.

Informed consent for the patients in the botulinum toxin-A group included disclosure of the off-label use of onabotulinumtoxinA (BOTOX; Allergan, Irvine, California) for this treatment, as well as the potential risk of prolonged weakness, and the need to seek emergency care in the event of respiratory depression or swallowing problems. A maximum total dose of 10 U/kg of reconstituted onabotulinumtoxinA was injected in equally divided doses into the subscapularis, the teres major, and the pectoralis major muscles (2 to 3 U/kg per muscle) at multiple intramuscular points. The latissimus dorsi muscle was not separately injected. The dilution used was either 50 or 100 U/mL, and the volume injected was never >0.5 mL per injection site. The target muscles were identified by palpation and localized with use of a nerve stimulator electrode (Stimuplex Dig RC; B. Braun Medical, Bethlehem, Pennsylvania) attached to a standard 1.5-in (3.8-cm) 25-gauge needle at the lowest current required to produce one twitch per second, usually 1 to 2 mA.

Early in the study period, six patients did not have immediate casting, but the remaining twenty-nine had the shoulder immobilized in a modified shoulder spica cast. The cast was applied in two parts with use of so-called soft or flexible casting tape (3M Scotchcast Soft Cast; 3M Health Care, St. Paul, Minnesota). A well-padded long-arm cast was applied first, with the elbow flexed 90°, the forearm fully supinated, and the thumb-index space protected with felt padding because of possible diminished sensation at the C6-C7 vertebral level. A double layer of stockinette tubing was placed around the hips and trunk, and the infant was placed on two 12 × 10 × 8-in (30 × 25 × 20-cm)

blocks, one supporting the head and shoulders and the other, the pelvis and legs. A generous abdominal relief pad was placed under the stockinette to allow room for abdominal distention and feeding, and cast padding was wrapped around the body. Pieces of felt padding were placed over the anterior superior iliac spines and the sacrum, and then a band of casting tape was wrapped as a girdle around the lumbar area, allowing the hips to flex fully. A strut of three to four thicknesses of regular fiberglass casting splints, twisted in the middle to create a "goose-neck" and flattened at the ends, secured the posterior proximal forearm of the long-arm cast to the anterior aspect of the girdle cast. The shoulder was immobilized in adduction and external rotation to provide the greatest stretch of the internal rotator muscles[23] (Fig. 1). The cast was checked at two weeks, and the abdominal portion was expanded if necessary by wedging the cast at the posterior midline. The cast was removed after four weeks, and range-of-motion exercises were restarted.

Records were reviewed for the severity of brachial plexus involvement (Narakas type)[24], age at the time of treatment of the shoulder subluxation, treatment provided, recurrence of the subluxation, and subsequent treatments needed to reduce or rebalance the glenohumeral joint. Follow-up clinical and imaging findings for the involved shoulders were reviewed.

Clinical indicators of glenohumeral congruency and stability included passive external rotation of the shoulder and assessment with the Active Movement Scale[25] or a modified Mallet scale in the older child during the course of treatment[26,27]. Imaging was by radiography and/or ultrasonography. Ultrasonographic data were evaluated for α angle and percentage of posterior displacement of the humeral head[28].

Source of Funding
No external funds were used in this study.

Results

There were seventeen boys and eighteen girls, with seventeen right and eighteen left sides involved. Twenty-eight infants had a Narakas type-1 brachial plexus lesion (involvement of the C5 and C6 nerve roots); five, a type-2 lesion (involvement of the C5, C6, and C7 nerve roots); and two, a type-3 lesion (involvement of the entire plexus). The average age at the time of diagnosis of subluxation or dislocation was 5.2 months (range, two to sixteen months). Twelve infants were found to have shoulder subluxation at the initial clinic visit, while all patients followed prior to the diagnosis of subluxation or dislocation failed an exercise program that stressed passive external rotation with the shoulder in adduction. The average age at the time of treatment was 5.7 months (range, three to sixteen months). The average duration of follow-up was thirty-seven months (range, twelve to eighty months).

Early in the series, six patients did not have immediate casting of the shoulder (Fig. 2). Of this group, two shoulders remained reduced, with one shoulder later undergoing tendon transfers to augment external rotation. Of the four shoulders that redislocated in this group, two underwent a second course of onabotulinumtoxinA with casting, and both

Fig. 1
The cast used for immobilization of the shoulder in an infant keeps the chest free and the abdomen relieved for feeding. The position of the shoulder can be modified according to the procedure done. Removal of the cast does not require a cast saw because of the casting material used.

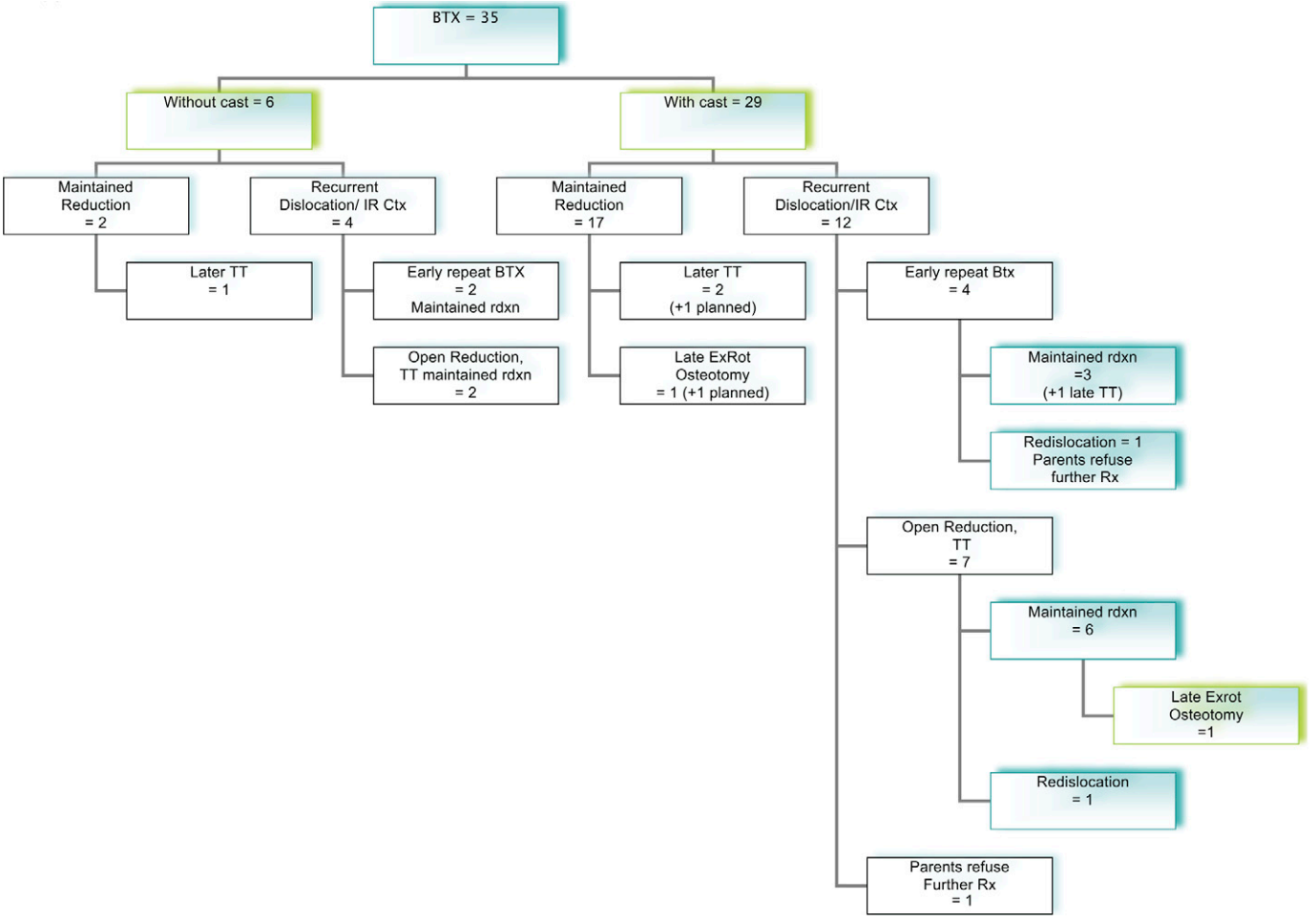

Fig. 2

The results of treatment on the reduction of the shoulder in infants treated with casting and botulinum toxin A. BTX = onabotulinumtoxinA; IR Ctx = internal rotation contracture, TT = tendon transfers, rdxn = reduction, ExRot = external rotation, and Rx = treatment.

maintained glenohumeral reduction. The other two shoulders required later open surgical reduction and then remained reduced.

Twenty-nine of the thirty-five patients underwent injection of onabotulinumtoxinA and concomitant casting. Seventeen of the twenty-nine shoulders maintained the reduction, with two that later underwent tendon transfers and one that had a late external rotation humeral osteotomy to augment external rotation. Two additional patients were scheduled for either tendon transfer or osteotomy at the time of follow-up. Twelve of the twenty-nine patients had recurrence of the shoulder dislocation. The parents of one of these twelve patients refused additional treatment. Four of the twelve shoulders had an early redislocation and received a second course of onabotulinumtoxinA; three of the four shoulders remained reduced, and one redislocated but the parents refused further treatment. Seven of the twelve patients underwent an open procedure to reduce and rebalance the shoulder, and six of the seven shoulders remained reduced (with one undergoing a late external rotation humeral osteotomy) and one shoulder redislocated in a child with a Narakas type-3 palsy. Therefore, twenty-six (74%) of the thirty-five patients who received supplemental onabotulinumtoxinA injections into the internal rotator muscles for early shoulder dislocation and/or subluxation did not require early open surgical procedures to reduce the shoulders. Of these, twenty-one had a single injection of onabotulinumtoxinA and five had two injections of onabotulinumtoxinA.

During this same period of case collection, an additional forty-one open contracture releases and shoulder reductions were done in our center on infants under the age of one year, either because manipulative reduction had failed or preoperative imaging showed a fixed posterior dislocation.

The eleven infants who experienced a redislocation, even after a second injection of onabotulinumtoxinA, included the three oldest (ten, eleven, and fifteen months of age) in the series, although the average age was 6.6 months (range, two to fifteen months). For those with redislocation, eight were Narakas type 1, two were type 2, and one was type 3. This group also included the two infants whose parents refused further treatment.

Complications

There were no complications, either local or systemic, related to the botulinum toxin-A injections or related to the casting.

Discussion

Although many infants with neonatal brachial plexus palsy can be expected to have complete or nearly complete recovery of shoulder function, some have permanent deficits in shoulder motion and strength. During normal shoulder development, the concentric position of the humeral head within the glenoid fossa is maintained both by the shape of the articulating surfaces and the stabilizing muscles. Each side of the joint induces the morphology of its opposing cartilaginous surface[7,29-34]. Functional loss of active motion due to muscle weakness, combined with loss of passive motion due to soft-tissue contracture, and a change in the shape of the shoulder joint eventually result in a permanent functional deficit.

Posterior shoulder subluxation or complete dislocation may develop early in neonatal brachial plexus palsy[6,10,35]. Moukoko et al. reported an 8% prevalence of posterior shoulder dislocation in 134 patients with neonatal brachial plexus palsy before the age of one year[6]. The hallmark clinical sign of the at-risk shoulder is loss of external rotation. Other clinical signs are shortening of the humeral segment, extra skin folds in the arm, and a deep axilla on the affected side. Ultrasonographic imaging is both a valuable screening tool and a dynamic means of evaluating the position and congruency of the still unossified shoulder[28], so it is possible to identify potential shoulder dislocation in the very young infant.

Although microsurgical nerve reconstruction, tendon transfers, and humeral osteotomies all have a role in the treatment of this condition, outcomes correlate best with the severity of the initial injury[8-11,36-39]. Congruency of the involved shoulder joint also affects the potential for the recovery of shoulder function. Waters and Bae postulated that "progressive glenohumeral joint deformity may be arrested if appropriate surgical procedures are performed when the patient is relatively young and the shoulder is in an early stage of development."[9]

Open procedures to rebalance a shoulder joint following brachial plexus palsy have been standard treatment options in children with established deficits who are past the period of anticipated neurologic recovery[40-43]. Preservation or restoration of passive range of motion, prevention of fixed contractures, and maintenance of a congruent glenohumeral joint during the early recovery period following brachial plexus palsy should optimize function after either spontaneous recovery or surgical rebalancing.

Botulinum toxin type A was first reported as a treatment for spasticity in children with cerebral palsy in 1993[44]. Botulinum toxin A is one of seven neurotoxin serotypes produced by *Clostridium botulinum.* Botulinum toxin A has been the most studied in terms of therapeutic application. By preventing release of acetylcholine at the presynaptic neuromuscular junction, it reduces the ability of a muscle to contract. There is usually a twenty-four to seventy-two-hour delay between toxin injection and clinical effect[45]. Although its antispasticity effect may last up to twelve weeks in children with cerebral palsy, improved range of motion and kinematics may last for up to twenty-four weeks after treatment[46]. More recently, botulinum toxin A has been used to treat nonspastic contractures of the biceps and triceps in children with neonatal brachial plexus palsy[12], and has also been used as treatment of the dysplastic shoulder[47]. Price et al. reported better functional outcomes in the seventy-four patients injected with botulinum toxin A as an adjunct to the surgical treatment in comparison with the seventy-four without botulinum toxin A at a minimum follow-up of two years[14]. DeMatteo et al. reported on three children with neonatal brachial plexus palsy who received injections in the latissimus dorsi and pectoralis major and concluded that botulinum toxin A can facilitate improved muscle function in the affected limb[13]. Our present study focuses on the early treatment of the imbalance between shoulder external rotation and internal rotation with closed manipulation and immobilization in a spica cast augmented with botulinum toxin-A injection into the contracted internal rotator muscles in an attempt to improve final shoulder function and reduce the need for open reconstructive shoulder surgery.

Our present study confirms that shoulder dysplasia commonly develops early in the first year of life, as the average age of our patients with posterior subluxation of the shoulder was four to five months. Heightened awareness of the clinical findings in these shoulders has led us to a more aggressive approach to restore and maintain passive external rotation and shoulder joint congruency. In our experience, early reduction has been facilitated greatly by supplemental onabotulinumtoxinA injection, leading to better outcomes defined as a decreased need for open procedures to release contractures and reduce the shoulder joint. Although it is not possible to prove with significance that this approach is superior to treatment without onabotulinumtoxinA, the number of patients at Texas Scottish Rite Hospital for Children who have required an open procedure to reduce the shoulder has decreased substantially over the past decade, despite an increasing number of new patients seen in our clinic. We believe this can be directly associated with the use of onabotulinumtoxinA as an adjunct to the treatment of these infants.

Another study weakness may be the lack of magnetic resonance imaging of these children. In our institution, we have become comfortable and familiar with ultrasonographic imaging of shoulders in very young children. Ultrasonography is available on the same day and requires no sedation or anesthesia, and it has proven to be reliable and cost-effective for this very young group of patients. Although there are no direct comparison studies of ultrasonography and magnetic resonance imaging to validate the findings in posterior shoulder dislocation, it is unlikely that such a study would be approved by a review board since it would require sedation or the use of an anesthetic agent. Incidental imaging at the time of evaluation of the brachial plexus and cervical nerve roots may provide an opportunity to examine this issue[34].

In conclusion, closed manipulation and immobilization of the glenohumeral joint augmented with injection of

onabotulinumtoxinA into the shoulder internal rotator muscles is an effective approach in the treatment of the infant with neonatal brachial plexus palsy with early, reducible posterior subluxation or dislocation of the shoulder. ■

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