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Management of spasticity in children with cerebral palsy

Beyin felçli çocuklarda spastisitenin tedavisi

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Management of spasticity is a major challenge to the rehabilitation team. The initial management has centered on the elimination of externally exacerbating causes, physical therapy, splinting and casting. Medical management has centered on anti-spasticity medication use, but more recently focal treatment methods including phenol blocks and botulinum toxin have been utilized. There has been an increased use of intrathecal baclofen in the management of refractory tone. Dorsal rhizotomy has been advocated for a selective population of children with spasticity. There is no standardized approach to spasticity management and this paper will discuss the importance of evidence-based treatment of spasticity that is adapted for the individual child.

Key words: Baclofen; botulinum toxin type A; cerebral palsy/drug therapy/surgery; child; dantrolene; injections, spinal; movement disorders; muscle spasticity/drug therapy; rhizotomy.

Spastisitenin tedavisi rehabilitasyon ekibi için zorlu bir iştir. Başlangıç tedavisi, ağırlaştırıcı dış nedenlerin ortadan kaldırılması, fizik tedavi, atel ve alçılama üzerine yoğunlaşmıştır. Medikal tedavide ise ağırlık spastisiteye karşı ilaç tedavisindedir; ancak, son zamanlarda fenol blokları ve botulinum toksini gibi fokal tedavi yöntemlerinden de yararlanılmaktadır. İlaçlara dirençli tonusun tedavisinde intratekal baklofen kullanımı giderek yaygınlaşmaktadır. Spastik çocukların seçilmiş bir kısmında dorsal rizotomi uygulaması da savunulan yöntemlerden biridir. Spastisitenin tedavisinde standartlaşmış bir yaklaşım bulunmamaktadır. Bu yazıda spastisitede çocuğa özel ve kanıta dayalı tedavinin önemi vurgulanmıştır.

Anahtar sözcükler: Baklofen; botulinum toksin tip A; beyin felci/ilaç tedavisi/cerrahi; çocuk; dantrolen; enjeksiyon, spinal; hareket bozuklukları; kas spastisitesi/ilaç tedavisi; rizotomi.

Cerebral palsy (CP) is defined as "a group of permanent disorders of development of movement and posture causing activity limitations that are attributed to non-progressive disturbances that occurred in the developing fetal or infant brain.[1]

Abnormalities of tone are an integral component and hypertonicity affects the majority of children with CP.[2] The most common form of hypertonicity is "spasticity" which is defined as "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the motor neuron syndrome."[3] Spasticity may occur focally in distinct muscle groups or more globally affect the majority of axial and appendicular skeletal muscles. It can interfere with movement and positioning, contribute to the formation of contractures and musculoskeletal deformities, and be a source of discomfort. It can also negatively impact function and make caregiver tasks, such as transfers and dressing more difficult.

There are a wide variety of treatment options for hypertonicity including oral medications, nerve blocks, and surgery. Determining whether abnormal tone is present globally or focally and the magnitude of its effect on an individual's musculoskeletal sys-

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tem, function, and comfort should guide one's treatment plan. The specific goals of tone reduction should always be determined prior to any intervention.

Oral medications

Oral medications are often used as an early treatment strategy for global spasticity. Medications that are most frequently used include baclofen (Lioresal®), dantrolene sodium (Dantrium®), clonidine, diazepam (Valium®), and tizanidine (Zanaflex®).[4] All of these medications, except dantrolene sodium, work through the central nervous system and, therefore, have the potential for sedation. None of these medications have been found to be universally effective in relieving spasticity[5,6] and evidence related to functional improvement is extremely sparse. The choice of medications is, therefore, often based on the impact of potential side effects on the individual patient.

Baclofen

Baclofen is a gamma-aminobutyric acid (GABA) analogue that acts at the spinal cord level to impede the release of excitatory neurotransmitters implicated in causing spasticity.[7] Low lipid solubility impedes passage through the blood brain barrier with more than 90% of the absorbed drug remaining in the systemic circulation.[7] As a result, large doses may be necessary to achieve an effect, which may result in dose-related side effects such as drowsiness. Very few studies have been published regarding the use of oral baclofen in CP. Two small double-blind, placebo-controlled, cross-over trials produced differing conclusions regarding the effectiveness of baclofen in reducing spasticity, but neither employed validated outcome measures.[8,9]

Benzodiazepines

Benzodiazepines have an inhibitory effect at both the spinal cord and supraspinal levels mediated through binding near but not at the GABA receptors and increasing the affinity of GABA for $GABA_A$ receptors. Diazepam is the most frequently used benzodiazepine and oldest antispasticity medication that is still in use,[6] but like other oral medications in CP, its effectiveness has not been well evaluated in CP. It is rapidly absorbed, reaching peak drug levels an hour after drug administration. The positive effect of diazepam may be related to general relaxation that permits improvements, especially in those individuals with athetosis and spasticity.[10]

Dantrolene sodium

Dantrolene sodium is unique in that it works primarily through actions on the skeletal muscle and not through central nervous system pathways. It inhibits the release of calcium from the sarcoplasmic reticulum, thereby uncoupling electrical excitation from muscle contraction and reducing contraction intensity. It is well absorbed within 3 to 6 hours after ingestion and is metabolized in the liver to 5-hydroxydantrolene with peak effect in 4 to 8 hours.[11] Doses in children range up to 12 mg/kg/day. It is often suggested that dantrolene be considered for the treatment of spasticity of cerebral origin because its mode of action is not central nervous system mediated, thus, it is less likely to be sedating.[6] Side effects from treatment, though, can include mild sedation as well as nausea, vomiting, and diarrhea. Use of dantrolene is also associated with hepatotoxicity.[11] Liver function studies should be done prior to instituting treatment and periodically while on maintenance therapy.[6] There are a few published trials of Dantrium in CP. One report on long-term use of dantrolene in children with spastic diplegia indicated that young children achieved greater levels of function than predicted prior to dantrolene administration and older children were able to move more easily and maintain their highest level of function.[12]

Additional oral medications used to treat spasticity in children with CP include alpha-2-adrenergic agonists such as clonidine and tizanidine, as well as certain anticonvulsants including gabapentin (Neurontin®). The alpha-2 adrenergic agonists result in decreased motoneuron excitability by decreasing the release of excitatory amino acids.[11] The side effects associated with these agents are frequently the cause of their more limited use and include nausea, vomiting, hypotension, sedation, dry mouth, and hepatotoxicity. In addition, reversible liver enzyme elevations have been noted in 2 to 5% of patients.[6] Gabapentin is structurally similar to GABA, readily crosses the blood-brain barrier, and is not protein bound. It does not activate GABA, but results in increased brain levels of GABA.[6] Reports of its use in children with spasticity are not available as of yet.

Chemical denervation

Chemical denervation should be considered for the treatment of significant focal increases in tone. The advantage of an injected, locally administered agent

is to limit the systemic effects while targeting a specific nerve or muscle.[13]

Phenol

Phenol motor point blocks have been used for many years to reduce focal increases in tone. Phenol injections, with 3 to 5% solutions, either at motor points of selected muscles or perineurally, denature proteins and disrupt efferent signals from hyperexcitable anterior horn cells by inducing necrosis of axons.[14,15] Nerves that are more commonly treated with phenol include the musculocutaneous and obturator nerves, given the reduced sensory function of these nerves and the lower risk for dysesthesias.[16] The low cost of phenol, coupled with reports of duration of action exceeding 12 months, render phenol injections an attractive treatment option in selected patients with focal spasticity. [17] In children, they are frequently done under general anesthesia, thereby, causing additional risks and costs.

Botulinum toxin (BoNT)

BoNT is a protein composed of a heavy chain which binds nerve terminals at the neuromuscular junction, and a light chain which is transported into the nerve terminal blocking the release of acetylcholine presynaptically and thereby weakening the force of muscle contraction produced by the hyperexcitable motor neurons. BoNT-A is marketed as Botox® in the United States and as Dysport® in Europe. BoNT-B is marketed as Myobloc®.[13]

Muscles commonly treated with BoNT include the gastrocsoleus complex, hamstrings, hip adductors,[18-20] and flexor synergy muscles of the upper extremity.[21-23] Intramuscular injections can be localized by surface landmarks, electromyographic guidance/stimulation, and/or ultrasound. Following injection, muscle relaxation is evident within 48 to 72 hours and persists for a period of 3 to 6 months.[24] Dosing is not equivalent amongst the various brands. It is dependent upon both body weight and size of the target muscle(s). Universally accepted dosing guidelines do not exist, but a consensus statement[19] and systematic reviews[25] of dosing and injection techniques are available for guidance.[26] Injections are typically spaced a minimum of three months apart due to concerns for antibody formation in an estimated 5% of patients, resulting in potential resistance.[27]

Many studies in the literature describe the effects of BoNT-A in children with CP. A systematic review of the literature summarized 17 controlled trials.[28] The literature supports improvement in gait over the 1-3 months following injections into the gastrocnemius muscles for spastic equinus.[29-31] Two small open-label studies found modest improvements in either gait kinematics or muscle length following injection into the hamstrings.[32,33] Several small trials evaluating the effectiveness of casting of the ankle in addition to BoNT-A failed to show any additional benefit.[33,34] Injections into the hip adductors resulted in improved range of motion[35] and decreased postoperative pain in children undergoing adductor lengthening.[36] More research needs to be done to determine the optimal choice of muscles, the most appropriate dose, the number of injection sites, the safety of repeated and long-term injections, and the risk for development of secondary resistance to BoNT due to antibody formation.[28]

Side effects are rare with BoNT, but may include pain during injection, infection, bleeding, a cool feeling in injected limbs, rash, allergic reaction, flu-like symptoms, excessive weakness, and fatigue.[31] Reports of serious or potentially life threatening side effects from BoNT are extremely rare. The United States Food and Drug Administration issued a statement on February 8, 2008 identifying cases of respiratory failure and mortality in children with CP linked to injection with botulinum toxin serotypes A and B. Rare cases of serious systemic effects have been reported in the literature in children receiving higher doses of BoNT.[37,38] Caution is recommended when injecting children with pseudobulbar palsy.

Intrathecal baclofen

Intrathecal baclofen (ITB) was first described by Penn and associates in 1984 and was FDA approved for the treatment of spasticity of cerebral origin in 1996. Baclofen is delivered directly to the cerebrospinal fluid via a catheter connected to an implanted device in the abdomen. The device contains a peristaltic pump, a battery with an operational life of 4 to 7 years, a reservoir for baclofen, and electronic controls that permit regulation of the pump by telemetry.[39] This feature allows baclofen infusion rates to be either continuous throughout the day or at varied dosages in order to accommodate the patient's specific needs. By infusing baclofen directly into the subarachnoid space around the spinal cord, potentiation of GABA-mediated inhibition of spasticity can be achieved while minimiz-

ing side effects related to high levels of baclofen in the brain.[27] Administration of ITB produces levels of baclofen in the lumbar cerebrospinal fluid that are 30-fold higher than those attained with oral administration.[27] The half-life of ITB in the cerebrospinal fluid is five hours.[13]

Candidates for ITB have severe, generalized tone that has not been successfully managed with oral medications and other more conservative measures. The increased tone must have a significant effect on function, ease of care, or comfort. Intrathecal pumps can be implanted in children generally greater than 15 kg in body weight.[40] Prior to surgical implantation, a test dose of 50-100 μg of intrathecal baclofen is typically given via lumbar puncture to verify a reduction in tone.

The ITB pump is typically programmed post-operatively to deliver baclofen at a continuous rate, typically at a daily dose similar to the dose given during the trial. The dose is not related to age or weight, and ITB dosages typically increase over the first year of treatment, then stabilize.[39] Refills of intrathecal baclofen are generally needed every 1-6 months depending on baclofen infusion dosage, the size of the pump, and the concentration of the baclofen being used.

Complications from ITB can result from programming error, pump failure, catheter failure, and infection. The majority of these problems involve breakage or disconnection of the catheter, but can also include blockage and kinking.[6,41]

Catheter or pump dysfunction can result in decreased baclofen delivery and baclofen withdrawal. Intrathecal baclofen withdrawal can also be seen in cases of battery failure without low battery alarm warning.[6] Early symptoms of withdrawal include pruritis, dysphoria, irritability, increased spasticity, tachycardia, fever, and changes in blood pressure.[42] If not recognized and managed optimally, baclofen withdrawal may progress to serious and life-threatening complications including severe hyperthermia, seizures, rhabdomyolysis, disseminated intravascular coagulation, altered mental status, psychomotor agitation followed by multisystem failure and death. Immediate treatment with high dose oral baclofen and/or diazepam, as well as referral to an emergency room setting is recommended in these scenarios.[13] Investigations into the causes for withdrawal should then ensue, including plain radiographs to assess pump and catheter placement in comparison to previous radiographs. Further studies may include dye or isotope studies to assess for catheter placement, leakage, and kinking.

Treatment for withdrawal can include any combination of oral baclofen, intravenous diazepam, or infusion of intrathecal baclofen through use of a lumbar drain.[43] Cyproheptadine, a serotonin antagonist, has also been used as an adjunct to baclofen and diazepam for treatment of severe intrathecal baclofen withdrawal.[44] Dantrolene sodium use should also be considered in patients with suspected rhabdomyolysis as a result of withdrawal. Intravenous physostigmine or withdrawal of 30 to 40 ml of cerebrospinal fluid can be tried in severe overdoses.[42]

A number of studies have reported on the outcomes of ITB. Randomized controlled trials have shown a significant decrease in spasticity.[41,45] Non-controlled trials have demonstrated improvements in joint range of motion, reduced pain, ease of care, and function.[46,47] Treatment with intrathecal baclofen is also associated with an increase in weight gain velocity.[48]

Selective dorsal rhizotomy

Selective dorsal rhizotomy (SDR) is a neurosurgical procedure that involves partial sensory deafferentation at the levels of L_1 through S_2 nerve rootlets.[49] Operative technique involves the performance of single or multilevel osteoplastic laminectomies, exposing the L_2-S_2 roots. Motor and sensory roots are separated to allow for electrical stimulation of individual sensory roots. The selection of rootlets for cutting is based on the lower-extremity muscular response to electrical stimulation of the rootlets. Although there is variability in percentages of rootlets cut, in general, about 50% of the sensory rootlets at any level are cut.[27,50]

Following the procedure, the reduction in spasticity often unmasks a significant amount of lower extremity weakness. As a result, intensive therapy is necessary to guide the patient through appropriate motor patterns and strengthening programs. Ideal candidates for SDR include children between the ages of 3 and 8 years of age who are GMFCS levels III or IV.[51]

A meta-analysis of three randomized controlled studies comparing SDR plus physical therapy with physical therapy alone has been completed.[51] Findings included a clinically important decrease in spas-

ticity, as well as a small but statistically significant advantage in function (GMFM-88) with SDR plus physical therapy. The subjects in these studies were primarily ambulatory children with spastic diplegia, and those with dystonia, athetosis, and ataxia were excluded. An additional larger nonrandomized controlled study compared SDR with physical therapy to physical therapy alone in children with spastic paraparesis, having GMFCS levels I to III.[52] Results of this study were similar to studies in the meta-analysis including gains in strength, gait speed, and overall gross motor function in children who received SDR plus physical therapy.[52]

Although immediate perioperative complications are not uncommon with SDR, long-term complications such as sensory dysfunction, bowel or bladder dysfunction, or back pain are infrequent.[53]

Conclusion

Management of spasticity is a major challenge to the rehabilitation team. Initial management should focus on the elimination of externally exacerbating causes. If the spasticity interferes with function, causes pain, and produces deformity, then clear treatment goals should be established. This rehabilitation management often requires a variety of different approaches including oral medications, peripheral nerve blocks, intrathecal medication, and often surgical interventions such as selective dorsal rhizotomy and orthopedic surgery. There is not a standardized approach. The treatment needs to be evidence-based and adapted for the individual.

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