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Use of botulinum toxin in children with cerebral palsy

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Abstract
Cerebral palsy in children is a syndrome of disorders resulting from damage to the central nervous system in its early development [1]. Damage to the structure of the upper motor neuron during its early development leads to dysfunction of the central motor control system and, consequently, to impairment of the child's functional development [2,3]. The consequences of damage to the central motor control system include the presence of muscular tension disorders, especially spasticity [4,5]. Botulinum toxin is one of the many pharmacological agents used to reduce spasticity. Botulinum toxin therapy is one of the most effective and currently safest methods for the treatment of spasticity in children [6].

Keywords: cerebral palsy, spasticity, botulinum toxin

Introduction
Cerebral palsy in children is a syndrome of disorders resulting from damage to the central nervous system in its early development [1]. Cerebral palsy in children is defined as persistent, non-progressive but not changing postural and motor disorder resulting from damage to the developing central nervous system during pregnancy, childbirth or early life after birth. The frequency of CP occurrence according to various statistics varies from 2 to 3 per 1000 births [1]. Many factors influence the development of cerebral palsy in children. The most important are: prematurity, hypoxia and ischaemia of the fetus, extremely small mass of the fetus, multiple pregnancy,

infections during pregnancy and the age of mother under 16 and over 40 years of age. [2]. Cerebral palsy in children is the above mentioned syndrome of abnormal posture and locomotion symptoms occurring in various clinical forms, mainly in spastic, hypotonic and rarely dyskinetic forms [2]. The damage to the structure of upper motor neuron during its early development leads to disorders of the central motor control system and, consequently, to impairment of the child's functional development [2,3]. The consequences of damage to the central motor control system include the presence of muscular tension disorders, such as spasticity, pathological reflexes, muscle clonuses, global movement patterns – so-called synergy, muscle weakness, loss of selective control of movement, as well as deformations and loss of efficiency [4,5].

Cerebral palsy is a complex therapeutic issue, there is no possibility of a complete cure and in such a situation the symptoms should be alleviated so that the child could be as independent as possible in life [2]. In the rehabilitation process of children with CP, the aim is to create optimal functional patterns [2]. According to the general principles, the rehabilitation of children should be comprehensive, early, continuous and complex – although there are some additional principles in this rehabilitation, specifically "paediatric" [3].

The forms of cerebral palsy in children vary according to the location of damage and severity of symptoms, and each small patient should be treated individually. Early diagnosis and detection of irregularities in the child's development is the most important issue. Quick implementation of rehabilitation process enables to improve the quality of patient's life at a given development stage, but also in the future [4,5]. In the rehabilitation of children with CP, aids are often used which do not directly improve, but rather alleviate individual symptoms [7,8,9]. Botulinum toxin is one of the many pharmacological agents used to reduce spasticity. Botulinum toxin therapy is one of the most effective methods of combating spasticity in children [6]. However, it is important to remember that not every patient is eligible for this therapy. Prior to botulinum toxin treatment, each child should be clinically evaluated to determine the possibility of therapy [6]. The use of botulinum toxin preparations facilitating the normalization of muscle tension in children with CP is important and has an impact not only directly on the child, but also indirectly beneficially on other applied methods of improving treatment [10].

Botulinum toxin and spasticity

Botulinum toxin, also known as botulism – abbreviated as BTX (Latin botulus) belongs to neurotoxins, exotoxin produced by absolutely anaerobic bacteria – bacillus with the species name Clostridium botulinum, as well as by a few other representatives of the Clostridium species [11].

There are seven types of botulinum toxin: A, B, C, D, E, F, G. Type A is the strongest toxin of bacterial origin known to date. Each of them is produced by a different strain of bacteria, consists of serologically different proteins and differs in its mechanism of action [12]. Types are most important for human health: A, B and E. Botulinum toxin in the human body is combined with neuromuscular junction causing inhibition of acetylcholine release from presynaptic terminals [13]. Botulinum toxin type A (BTX-A), administered intramuscularly, is most commonly used in the treatment of dystonia and spasticity accompanying cerebral palsy, stroke, spinal cord injuries and multiple sclerosis [14,15,16]. Currently, botulinum toxin therapy is one of the most effective methods of combating spasticity in children [9]. Prior to BTX-A treatment, each child should be clinically evaluated in order to determine the treatment options as not every patient is eligible for BTX-A treatment [9,13]. The number of muscle groups occupied by spasticity has a huge role in BTX-A therapy.

The best results are obtained with BTX-A in case of focal spasticity (local), which significantly impairs function, e.g. gait, with relatively small coexisting paresis. An example of such a situation is the so-called spastic diperesis in children with CP, with a typical gait on fingers or crossing of lower limbs during walking [17]. In situations where spasticity is more generalized, the muscles selected are those that may have a key influence on the improvement of function, e.g. gait [9,13,17]. The BTX-A has a very special position among the drugs used in spasticity.

Planning the therapy of spastic muscles of upper and lower limbs, attention should be paid to the impact of specific muscle dysfunctions on the performance of the so-called high motor skills by the patient (sitting, crawling, standing, walking) and to the analysis of gait. For this purpose, a number of studies and tests may be used to assess spasticity, including: Modified Ashworth Scale, Modified Tardieu Test, Adductor Muscle Tension Scale, and Passive and Active Mobility [9].

In the upper limb, we usually want to improve the grip function of the hand, and in the lower limb we want to improve the gait [18].

Botulinum toxin administered to specific muscles reduces muscle tension by means of so-called chemical denervation [9,17]. The effect is inhibition of acetylcholine release in presynaptic nerve endings in muscles [17,18]. After injection BTX-A is bound and internalized to synaptic endings, the process usually takes 24-72 hours [9,18]. After this time we can expect the first clinical effects, after another 14 days the maximum effect is achieved and after an average of 12 weeks the clinical effect gradually disappears due to the phenomenon of new neuromuscular endings (so-called "sprouting") [9,17,18]. Repeated injections prevent soft tissue lesions and development of permanent contractures [18].

The BTX-A administration is based on both short and long term treatment, as well as comprehensive support [18].

Conclusions

Botulinum toxin therapy is currently one of the most effective and safest methods for treatment of spasticity. This therapy effects depend on the correct classification of the patient for treatment, the correct choice of muscles to be injected in patients with focal spasticity and reduced function, as well as the correct dose selection. However, treatment with botulinum toxin is an element in the improvement process of the patient with cerebral palsy in children. We should not forget about the continuous complex neurodevelopmental therapy of a small patient.

The benefits of BTX-A include improved function of upper and lower limbs, i.e. gait, grip, reduction of discomfort related to painful tension, prevention of permanent contractures, facilitation of muscle growth and development of normal movement patterns [9,18].

Bibliography

1. Milewska A. , Mileańczuk-Lubecka B.,A. , Kochanowski J. , Werner B.: Analiza czynników ryzyka mózgowego porażenia dziecięcego. Nowa Pediatria 2011, 4, 79-84.
2. Gugała B, Snela S: Mózgowe porażenie dziecięce – rys historyczny i poglądy na temat istoty schorzenia. Pielęgniarka i Położna 2006; 3: 25-26.
3. Matyja M., Nowotny J.: Zasady rehabilitacji dzieci z uszkodzeniami ośrodkowego układu nerwowego-aspekty teoretyczne i praktyczne. Zeszyty Metodyczno-Naukowe, AWF, Katowice 1996:8, 5–14.
4. Pogorzelczyk M , Gajewska E.: Terapia Dziecka z Mózgowym Porażeniem Dziecięcym z Punktu Widzenia Fizjoterapeuty. Polski Przegląd Nauk o Zdrowiu 1 (38) 2014, 43-47.
5. Nowotny J., Czupryna K., Domagalska M.: Aktualne podejście do rehabilitacji dzieci z mózgowym porażeniem dziecięcym. Neurologia Dziecięca, 2009, 35: 53-60.
6. Mirska A, Kułak W.: Terapia spastyczności toksyną botulinową w mózgowym porażeniu dziecięcym. Neurologia Dziecięca. Vol. 18/009, nr 6, 59-63.
7.Domagalska M., Czupryna K., Szopa A. et al.: Specyficzne i alternatywne sposoby terapii dzieci z porażeniem mózgowym. Neurol. Dziec., 2005:14, 7–15.
8. Nowotny J.: O racjonalne podejście do usprawniania dzieci z mózgowym porażeniem dziecięcym. Neurol. Dziec., 1993:1, 29–36.
9. Domagalska M., Czupryna K., Szopa A. et al.:Wzorce postawno-lokomocyjne dzieci z m.p.dz. a programowanie rehabilitacji. Fizjoter. Pol. 2007:7, 320–331.
10. Wilk T., Malinowska-Matuszewska M., Bilski J., Niewęgłowska-Wilk M.: Ocena skuteczności terapii toksyną botulinową u dzieci chodzących samodzielnie ze spastyczną postacią diplegii oraz hemiplegii w przebiegu mózgowego porażenia dziecięcego. Zeszyty Naukowe WSSP, TOM

17 – 2013, 129-134.
11. https://pl.wikipedia.org/wiki/Jad_kiełbasiany
12. Domżał T.M.: Toksyna botulinowa w praktyce lekarskiej. Wyd. Czelej, Lublin 2002
13. https://www.umb.edu.pl/photo/pliki/WNoZ_jednostki/wnoz-k-rehabilitacji-dzieciecej/artykul_toksyna_botulinowa.pdf
14. Jankovic J.: Treatment of dystonia. Lancet Neurol 2006; 5: 864–872.
15. Benecke R., Dressler D.: Botulinum toxin treatment of axial and cervical dystonia. Disabil Rehabil 2007; 29: 1769–1777.
16. Koman L.A., Smith B.P., Shilt J.S.: Cerebral Palsy. Lancet 2004; 363: 1619–1631.
17. Sławek J.: Toksyna botulinowa typu A w leczeniu spastyczności w mózgowym porażeniu dziecięcym –podstawy teoretyczne i praktyczne skutecznej terapii. Ortop Traumatol Rehabil 2001; 4: 541–546.
18. Sławek J.: Rola toksyny botulinowej w terapii neurologicznej. Post Psych Neurol 1997; 6: 193-200.