

Management and Treatment for Cerebral Palsy in Children

Padmakar S¹, K Sujan Kumar¹, S Parveen²

¹Pharm-D, P. Rami Reddy Memorial College of Pharmacy [PRRMCP], Kadapa, Andhra Pradesh, INDIA.

²Department of Pharmacy Assistant Professor, P. Rami Reddy Memorial College of Pharmacy [PRRMCP], Kadapa, Andhra Pradesh, INDIA.

ABSTRACT

'Cerebral' – refers to the brain. 'Palsy' – can mean weakness or paralysis or lack of muscle control. Therefore, cerebral palsy is a disorder of muscle control which results from some damage to part of the brain. Cerebral palsy (CP) is a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing foetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbances of sensation, perception, cognition, communication, and behaviour, by epilepsy, and by secondary musculoskeletal problems. Approximately 80% to 90% of children with cerebral palsy have spastic cerebral palsy. The diagnosis of spasticity in children with CP requires a complete physical examination, with ancillary testing as needed. The aim of treatment is to encourage the child to learn to be as independent as possible. Some children who have mild cerebral palsy will not have any problems in achieving independence. For others, it will be a slow process. In some with severe difficulties, considerable assistance from others will always be needed. Specific treatment varies by individual and changes as needed if new issues develop. In general, treatment focuses on ways to maintain or improve a person's quality of life and overall health. The goal of management of cerebral palsy is not to cure or to achieve normalcy but to increase functionality, improve capabilities, and sustain health in terms of locomotion, cognitive development, social interaction, and independence.

Key words: Cerebral palsy, Management, Treatment, Children.

INTRODUCTION

DEFINITION

Cerebral Palsy is a group of permanent, but not unchanging, disorders of movement and/or posture and of motor function, which are due to a non-progressive interference, lesion, or abnormality of the developing/immature brain.¹

Cerebral palsy is primarily a disorder of movement and posture. It is defined as an "umbrella term covering a group of non-progressive, but often changing, motor impairment syndromes secondary to lesions or anomalies of the brain arising in the early stages of its development".²

EPIDEMIOLOGY

Unfortunately, it is difficult to access and clarify the prevalence and incidence rate of

disabilities in poor-resource settings (Gladstone, 2010). Not only the prevalence of childhood disability is on the rise and Cerebral Palsy is one of the costliest chronic conditions, but also life expectancies are improving, which increases the burden of Cerebral Palsy (Papavasiliou, 2009). For comparison, in the USA, there are approximately 700'000 children with Cerebral Palsy, 2-5/ 1000 born.

Cerebral Palsy is the most common motor disability in childhood. The aetiology of Cerebral Palsy is very diverse and multifactorial. The causes are congenital, genetic, inflammatory, infectious, anoxic, traumatic and metabolic. Population-based studies from around the world report prevalence estimates of Cerebral Palsy ranging from 1.5 to more than 4 per 1,000 live births or

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Address for correspondence:
Padmakar S,

Pharm.D Internship, P. Rami Reddy Memorial College of Pharmacy [PRRMCP], Kadapa, Andhra Pradesh, INDIA.

Phone no: 9603656446
Email Id: spadmakar71@gmail.com



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children of a defined age range. Most of the children identified with Cerebral Palsy have Spastic Cerebral Palsy (77, 4%). Over half of the children identified with Cerebral Palsy (58, 2%) can walk independently, 11, 3% walks using a handheld mobility device and 30, 6% has limited or no walking ability. Many children with Cerebral Palsy also do have at least one co-occurring condition (e.g. 41% Epilepsy).³

TYPES OF CEREBRAL PALSY

There are three types of cerebral palsy that can be distinguished by their symptoms and management approaches. The main types of CP are Spastic, Ataxic and Athetoid cerebral palsy.⁴

A. Spastic Cerebral Palsy: This is the most common type of CP. Spastic CP is characterized by unique muscle tightness, patients have muscle spasticity as the main impairment characteristic.⁵ This type of CP occurs in at least 70% of all CP cases in the world. In cases of spastic CP, the disorder is more easily manageable as compared to other types since treatment through medication can be pursued in several neurological and orthopaedic approaches. The spasticity of muscles leads to other muscle stress symptoms that may include tendinitis and arthritis in individuals who are 20-30 years old. This type of CP can be managed using occupational and physical therapy where strengthening, stretching, exercise and other physical activities are used to manage the disorder on a daily basis. The disorder can also be managed using medications that eliminate spasticity by killing the very nerves that cause the disorder.⁵

B. Ataxic Cerebral Palsy: This type of CP is less common as compared to spasticity, it may occur in 6-10% of all cases of CP. Ataxic CP is characterized by “ataxia-type” symptoms that inflict some cerebellum damage. The child may exhibit symptoms of unsteady posture. One may also shake while attempting to hold objects with the hand. Such symptoms are part of the motor degraded motor skills experienced by the child. One may have difficulties in their control of motor skills, which include typing, writing and holding small objects. The child may also show some disorientation and poor control while walking. Visual and auditory processing may also be affected in ataxic CP.

C. Athetoid Cerebral Palsy: This is also called Dyskinetic CP; it occurs in at least 10% of all CP cases. As compared to spasticity, the occurrence of this type of CP is relatively low. Patients with this type of disorder may have challenges in maintaining steady positioning. Steady sitting and walking is quite problematic; individuals may show some unintended

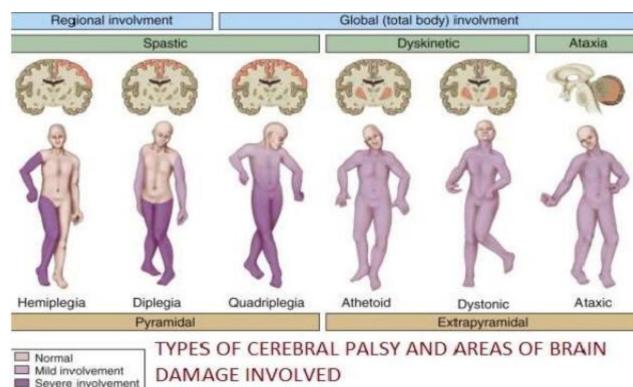


Figure 1: TYPES OF CEREBRAL PLASY

motions. In addition, patients may lose their ability to hold objects especially small objects that require some fine or advanced motor control. Such patients may not be able to hold small objects such as pens, coins and other small objects.⁵

CLINICAL RISK FACTORS FOR CP DURING PREGNANCY

There is increasing scientific evidence that CP is usually associated with longstanding intrauterine pathology like genetic mutations and probable environmental triggers such as bacterial and viral intrauterine infection, intrauterine growth restriction (IUGR), antepartum hemorrhage, tight nuchal cord, and threatened miscarriage.⁶ It can be difficult to pinpoint adverse pregnancy factors in retrospect, many years after birth, that individually or together might have triggered the pathways to the neuropathology.

Preterm delivery

Preterm delivery is a major risk factor for CP and is seen in approximately 35% of all cases, and the risk increases the lower the viable gestational age.^{6,7} The risk of subsequent CP <33 weeks' gestation is 30 times higher than among those born at term and is approximately 70/1000 deliveries.⁷ The prevalence of CP is highest in children born <28 weeks' gestational age (111.8/1000 neonatal survivors; 82.25/1000 live births) and declines with increasing gestational age, being 43.15/1000 live births between 28-31 weeks, 6.75/1000 between 32-36 weeks, and 1.35/1000 for those born >36 weeks.⁸ The mechanisms and pathways to the neuropathology of CP may differ from term babies, although associated risk factors such as infection, genetic variations, and growth restriction are likely to contribute.

Coexisting congenital anomalies

The prevalence of congenital anomalies in children with CP is much higher than in the general population

and most are cerebral, such as schizencephaly and hydrocephaly.⁹ Non-cerebral malformations are also increased, such as cardiac, musculoskeletal, and urinary.¹⁰ In a case-control study of 494 singleton infants with CP born >35 weeks' gestation included on the Western Australian Register of Developmental Anomalies and 508 matched controls, birth defects (42.3%) and fetal growth restriction (16.5%) were more strongly associated with CP than potentially asphyxial birth events (8.5%) and inflammation (4.8%).¹¹ Birth defects had the largest association with CP in that study in both term and preterm babies. Growth restricted babies with birth defects were at special risk of CP. The strong association with congenital abnormalities suggests possible genetic factors although congenital infections, nutritional disorders, and teratogenic influences all contribute to maldevelopment.

INTRAUTERINE INFECTION

There are many probable antenatal causes of white-matter damage and risk factors for CP. Some of these causes include damage acquired following perinatal infection (i.e., maternal infection that affects the foetus and its brain during pregnancy and/or labour or in the neonatal period).¹² Viral or bacterial infections may be relatively silent during pregnancy and not recognized clinically at the time and the placenta is often discarded without histological examination for inflammatory pathology. Maternal reports of fever or infection during pregnancy are significantly associated with an increased risk of CP in our recent large Australian case-control study.¹³ Evidence of intrauterine infection, evidenced by histological chorioamnionitis in the placenta and membranes or intrapartum pyrexia, is associated with a 4-fold increase in CP (odds ratio 3.8; 95% confidence interval, 1.5e10.1) in term infants.¹⁴

Intrauterine growth restriction

IUGR is associated with up to a 10 to 30 fold increase in the risk of CP at term.¹⁵ In particular, spastic CP increases with the degree of fetal growth restriction. A growth-restricted foetus may show signs of possible fatal compromise during labour. This can reflect reduced capacity/reserves to withstand the normal stresses of labour, established neurological and ongoing fatal compromise, or both. It is not possible to distinguish between these timings.

Multiple pregnancy

Multiple pregnancy increases CP risk 2-fold in each twin. *In vitro* fertilization twins each have >4-fold risk (9.5/1000), giving another reason to encourage single embryo transfer in fertility programs.¹⁶

Viral infection in pregnancy

Studies using polymerase chain reaction techniques on neonatal blood spots from CP cases and controls show increased CP risk after both Cytomegalovirus and Epstein-Barr virus infections during pregnancy.¹⁷ Epidemiological studies do not associate upper respiratory infections during pregnancy with CP, but some studies have associated increased risk with bacterial urinary tract infections.^{12,18}

Genetic causes of CP

Genetic causes have long been suspected because of the link with congenital malformations, and increased risk in consanguineous families and monozygotic twins.⁴³ Although initially candidate gene association studies suggested that several genes may be linked to CP, the power of these studies was low and multiple comparisons weakened their validity.^{19,20}

DIAGNOSIS

Observation of slow motor development, abnormal muscle tone, and unusual posture are common initial clues to the diagnosis of cerebral palsy. Assessment of persistent infantile reflexes is important. In infants who do not have cerebral palsy, the Moro reflex is rarely present after six months of age, and hand preference rarely develops earlier than 12 months of age. Hand preference may occur before 12 months of age if spastic hemiplegia is present.²¹ Progressive hereditary neurologic or metabolic disorders must be eliminated as the cause of observed abnormalities. The testing strategy is based on the clinical picture, pattern of development of symptoms, family history, and other factors influencing the probability of specific diagnoses. Targeted laboratory tests and cerebral imaging using computed tomography, magnetic resonance imaging, and ultrasound are useful physical diagnostic tools. Surveillance for associated disabilities such as hearing and vision impairment, seizures, perception problems with touch or pain, and cognitive dysfunction can help complete the clinical assessment and determine the diagnosis.²²

MANAGEMENT

The goal of management of cerebral palsy is not to cure or to achieve normalcy but to increase functionality, improve capabilities, and sustain health in terms of locomotion, cognitive development, social interaction, and independence. The best clinical outcomes result from early, intensive management. Optimal treatment in children requires a team approach.²³ A modern team approach focuses on total patient development, not just on improvement of a single symptom. Treatment

programs encompass physical and behavioural therapy, pharmacologic and surgical treatments, mechanical aids, and management of associated medical conditions. In physical, occupational, speech, and behavioural therapies, the goals include enhancing patient and caregiver interactions while providing family support.²³

Management of spasticity is a major challenge to treatment team. Various forms of therapy are available to people living with cerebral palsy as well as caregivers and parents caring for someone with this disability. They can all be useful at all stages of this disability and are vital in a CP person's ability to function and live more effectively.²⁴

Oral Medications

Oral medications are a systemic, rather than focal, treatment for spasticity in children with cerebral palsy. Oral medications commonly used in children are baclofen, diazepam, clonazepam, dantrolene and tizanidine.²⁵

Intrathecal Baclofen

Intrathecal baclofen (ITB) was approved for the treatment of spasticity of cerebral origin in 1996. ITB is a surgically implanted system used to control spasticity by infusing baclofen directly into the spinal canal and around the spinal cord.²⁶ Baclofen inhibits spasticity by blocking excitatory neurotransmitters in the spinal dorsal horn. ITB maximizes the dose delivered to spinal receptors and minimizes the side effects associated with oral baclofen.²⁷

Botulinum Toxin

Botulinum toxin (BT) injection is now an established first-line treatment for focal spasticity. Botulinum toxin type A produces dose-related weakness of skeletal muscle by impairing the release of acetylcholine at the neuromuscular junction. This partially interrupts muscle contraction making the muscle temporarily weaker. Muscles commonly treated with BT include the gastrocnemius-soleus complex, hamstrings, hip adductors and flexor synergy muscles of the upper extremity. Intramuscular injections can be localized by surface landmarks, electromyography stimulation, and/or ultrasound. Following injection, muscle relaxation is evident within 48 to 72 hr and persists for a period of 3 to 6 months. Botox injection can help improve a child's ability to walk or use hands and allow for a better fitting orthotics by reducing spasticity. Therapists can take advantage of the time when an overly powerful muscle is weakened to work on strengthening the muscle on the opposite side of the joint (antagonist). Sometimes, casting of the involved extremity is done after the injection to increase the stretch of the tight muscle.²⁸⁻³²

SURGERY

Orthopedic surgery

Surgery is mainly undertaken on the lower limb, but occasionally in the upper limb. Some children require surgery for scoliosis. Physiotherapy is an essential part of post-operative management. Gait laboratories are useful in planning the surgical program for children who can walk independently or with sticks or walking frames.

- The hip: soft tissue surgery is often effective for children when the hip problems are detected at an early stage (hence the importance of regular X-rays). Lengthening of the adductor muscles may be all that is required in younger children. However, if the problem progresses, and especially if it is neglected, more extensive surgery to the hip bones is required for a significant number of children. For most children surgery to keep the hips in joint, or to put the hips back in joint, is preferable to leaving the child with a dislocated hip which is frequently painful in later life.
- The knee: lengthening of the hamstrings can help the knee straighten and so improve the walking pattern. Sometimes transferring a muscle from the front to the back of the knee can also help by reducing stiffness around the knee.
- The ankle and foot: This is the commonest area where orthopedic surgery is required. Sometimes children require orthopedic surgery in several different areas (for example, hip, knee and ankle). Frequently this now involves a single hospitalization and is called '**multilevel surgery**'. Multilevel surgery is of most benefit to children who walk independently or with the assistance of crutches.

The best age is usually between 8 and 12 years old although it can occasionally be helpful for older or younger children.³³

TREATMENT FOR THE ASSOCIATED MEDICAL PROBLEMS

1. Epilepsy

Knowledge of epilepsy has increased substantially in the past few years. There are many types of epilepsy, and medication is often prescribed following a careful diagnosis of the type of seizures and their cause. The most commonly used anticonvulsants are: Carbamazepine, Sodium valproate, Lamotrigine, Phenytoin etc.

2. Saliva control

The speech pathologist plays a central role and can provide strategies to improve dribbling problems. When these strategies are not effective, medication is

occasionally used, particularly in children over the age of six years. These medications are as follows:

- Benhexol hydrochloride ('Artane') reduces salivary secretions. Occasionally irritability may occur. Blurring of vision, constipation and difficulty with urination are also potential side effects.
- lycopyrrolate ('Robinul') is like benhexol hydrochloride but seems to produce fewer side effects.

3. Constipation

Children with cerebral palsy often have problems with constipation. A high fiber diet and increased fluid intake can help with this problem. This may not be easily achieved in some children with cerebral palsy. Careful use of laxatives is preferable to severe constipation.

4. Nutrition

A dietitian can provide useful advice about adequate nutrition. Excessive weight gain can be very disadvantageous for children learning to walk. Under nutrition and failure to make adequate weight gains may be related to feeding difficulties. In a small proportion of children, the use of tube feeding can be helpful.³³

CONCLUSION

Cerebral palsy is a disorder of muscle control which results from some damage to part of the brain. Children with cerebral palsy can have problems such as muscle weakness, stiffness, awkwardness, slowness, shakiness, and difficulty with balance which remain throughout the lifetime of a person. The goal of management of cerebral palsy is not to cure or to achieve normalcy but to increase functionality, improve capabilities, and sustain health in terms of locomotion, cognitive development, social interaction, and independence. The management often requires a variety of different approaches including oral medications, botulinum toxin, intrathecal baclofen, occupational and physical therapy and often surgical interventions.

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ABBREVIATIONS

CP-Cerebral palsy; IUGR- intrauterine growth restriction.

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