Cerebral palsy: review of epidemiology, etiology, clinical features, classification and prevention

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ABSTRACT

Cerebral Palsy (CP) is a neurodevelopment disorder caused by improper brain development or harm to the developing brain and is the underlying cause of the most common motor disability in children. The clinical symptoms vary between subjects because the etiology is complex and can affect a variety of anatomical structures and each of these can lead to a different symptom. The motor dysfunction is often associated with sensory, perceptual, cognitive, communication and behaviour impairments as well as epilepsy and secondary musculoskeletal disorders which have a significant influence on the child’s quality of life, activity, and participation. The risk of developing CP is present in infants born preterm, but these children sum up less than 50% of cases. The factors that cause CP in children born at term are grouped in antenatal, perinatal, neonatal, some of them can be modified like alcohol consumption, maternal smoking, infections, but others like genetic factor cannot be modified. CP can be classified in different ways depending on the clinical manifestation. Throughout time classification was based on the type and distribution of motor anomalies, which often corresponded to the area of injury. Spastic subtypes, dyskinetic subtypes, and ataxic subtypes are the three basic forms of motor dysfunction. The most common conditions associated with CP are pain, intellectual disability, speech disorder, bladder control problems, epilepsy, and behaviour disorders. Early intervention is thought to be the most effective treatment for CP. As soon as the diagnosis is determined, rehabilitation treatment should begin. The earlier a rehabilitation intervention begins, the better the prospects of improving the child’s functional abilities and independence.

Keywords: cerebral palsy, risk factor, preterm infants, symptoms, dysfunction

INTRODUCTION

Cerebral palsy (CP) is a neurodevelopment disorder that appear in early childhood and encompasses a heterogeneous group of conditions with a different clinical pattern that have a common feature represented by permanent motor dysfunction characterized by modification in muscle tone, posture, and movement which have a significant influence on the child’s quality of life, activity, and participation [1]. CP is frequently accompanied by sensory, perceptual, cognition, communication, and behaviour impairments, as well as epilepsy and secondary musculoskeletal disorders. It is caused by improper brain development or harm to the developing brain. This normally occurs prior to labour, but it can also happen during delivery or early infancy. Even though CP is a non-progressive condition, clinical characteristics may change as the child’s central nervous system develops [2].
The overall prevalence is 2.11 per 1000 live birth, but is regarded to be the leading cause of substantial physical disability in children and has a significant impact on health, education, and social services, as well as on families and children [3]. In 2003, the estimated lifetime expenses in the United States for patients with this illness were $11.5 billion. Despite better survival of at-risk preterm newborns, the prevalence of CP has remained constant in recent years. Estimates of prevalence are required to guide health policy and aid resource allocation [4].

ETIOLOGY AND RISK FACTORS

The risk factors for CP may be antenatal, perinatal, or combined. Risk factors vary also by gestational age that is classified by the term of delivery in moderately preterm (MP) (32\textdegree – 33\textdegree weeks), late preterm (LP) (34\textdegree – 36\textdegree weeks), very preterm (VP) (32\textdegree weeks) and term infants (≥37 weeks). Incidence of CP is higher in children with MP, LP compared with term infants [5]. LP represents the largest proportion of preterm births with more than 70% of all prematurely born infants in the United States. LP and MP births account for more than 80% of all preterm births. It's vital to note that preterm babies' brains are more vulnerable to damage than full-term births. The weight of a 34-week-old baby is just 65% that of a full-term baby [6]. LPs are commonly referred to as “near term” infants, although this isn't the most accurate word because it downplays the risks of these preterm infants [7]. Even if the short-term and long-term mortality and morbidity in LP new-borns are minimal, they are significantly higher than in term infants [8]. The risk of developing CP is present in infants born preterm, but these children sum up less than 50% of cases with CP. There are numerous other prenatal and perinatal risk factors for developing CP.

The underlying causes of CP are not fully known, but several separate CP risk factors have been identified although little is known about how they interact and relate. The variance of CP is thought to be linked also with the social-economic status gradient and perinatal health [9]. Multiple risk factors coexist in the majority of cases with CP, but there are some factors that have a higher risk of developing CP: low birth weight, intrauterine infections, preeclampsia, placental abruption, multiple pregnancy, heavy maternal alcohol consumption, maternal smoking, maternal obesity (pre-pregnancy BMI ≥30), small size for gestational, Apgar <7 at 5 minutes, neonatal infection, respiratory distress syndrome, requiring mechanical ventilation after birth, requiring antibiotic therapy after birth, neonatal seizures [10]. The life of a newborn is divided into three important periods: antenatal – refers to the time between conception and the start of labour, perinatal – refers to the time between the start of labour and the seventh day of life, and neonatal – refers to the time between the seventh day of birth and four weeks of a child's life. In children born at term there are several factors that interact with these three periods that can lead to CP. The factors can be divided into maternal factors (reproductive history, medical history, pregnancy conditions), perinatal factors (cord complication, foetal distress, and Apgar scores), neonatal factors (neonatal infection, neonatal seizures, and meconium aspiration).

Antenatal risk factors for CP in children born at term are: infections (clinical diagnosis of chorioamnionitis, any maternal infection, neurotropic virus infection with any Herpes viruses group B for hemiplegia, first trimester cytomegalovirus maternal infection), social deprivation (mothers' exposure to extremely severe prenatal stress, children born at term with intra-uterine growth restriction), thrombophilic risk factors (factor V Leiden mutation, protein C and protein S mutation), genetic factors, (cytokine polymorphism – TNF-alpha in quadriplegia, mannose binding lecithin codon-54 in diplegia), malformations (congenital abnormalities, cerebral malformations), multiple gestation, maternal trauma.

Neonatal risk factors for CP in children born at term include: events during labour and delivery (Apgar score <7 at five minutes, Apgar 0-3 at five minutes, Apgar 4-6 at five minutes, severe foetal placental vascular lesions), perinatal stroke, parity, meconium aspiration, hyperbilirubinemia [11].

Some risk factors cannot be predicted, but some prenatal risk factors are modifiable like mother alcohol consumption, maternal smoking, maternal obesity, and infections during pregnancy.

CLINICAL FEATURES AND CLASSIFICATION

Motor symptoms

CP is mainly characterized by motor abnormalities and associated conditions that are linked to the disorder of cerebral function. Voluntary movement are complex actions that are preceded by brain activity aimed for preparing and executing the action. This brain activity can start up to 2 seconds before the execution of the movement [12]. In affected patients, these movements are uncoordinated, stereotypic, and limited. Actions that are considered simple by a normal, unaffected person can be challenging and realized with a big effort by a person with CP and in seriously impaired patients an attempt of voluntary movement evokes primitive reflexes [13]. For these patients isolated movements, such as those of a single finger, may be difficult to achieve and in most cases are impossible.

The classification of CP uses different schemas for subtype and functional status. CP can be classified in
control, spasticity may begin by 2 to 3 month of age, addition of the thighs results in typical scissoring of the leg. When babies are 9 to 10 months old, they are unable to stretch their legs and have poor truncal balance. Children with spastic quadriplegia over the age of 5 have all of their limbs affected, with the upper limbs being more affected than the lower ones. These children are frequently severely handicapped, with feeding difficulty, chronic respiratory insufficiency, and seizure disorder. Most commonly are affected term infants. In early infancy the symptoms are characterised by reduced spontaneous movement, hypotonia at rest, variable tone with movement or emotions, oro-motor incoordination, involuntary grimacing, drooling, delayed psychomotor development, head can be persistently turned [17]. From age 2 to 3 involuntary movement are apparent, abnormal posturing with extension pattern in the spine position, flexion with shoulder retraction in the prone position, head usually is persistently turned to one side [18,19].

**Dyskinetic subtypes** represent 12 to 14 percent of CP [14]. Most cases are caused by severe asphyxia resulting in injury to the thalamus, basal ganglia, hipocampus, reticular formation, and cerebellum. Severe hyperbilirubinemia can cause choreo-athetoid CP. Symptoms change after 5 years and the children have involuntary movements, contracture are not common but may evolve later in life, variable degree of dysarthria and intellectual disability. Choreo-athetotic CP consists of rapid, irregular, unpredictable contractions of the individual muscle or small muscle groups that involve the face, bulbar muscle proximal extremities, fingers, and toes. Athetosis consists of slow, smooth, writhing movements that involve distal muscle, movements may be induced or accentuated by emotion and change in posture. Athetosis is most apparent during reaching objects. Stress, excitement, or fever may exacerbate chorea. Oropharyngeal difficulties occur commonly, and primitive reflexes often are retained. Dystonic CP consists in repetitive patterned, twisting, and sustained movements of the trunk and limbs that may be either slow or rapid, pyramidal signs and anarthria may occur, tendon reflexes are normal or may be difficult to elicit, clonus and extensor plantar responses are absent [18].

**Ataxic CP** represents 4 to 13% of all CP. Most cases are caused by early prenatal events, etiologic in not fully known. Some cases have genetic causes (cerebellar hypoplasia, granule cell deficiency, Joubert syndrome), it is rarely associated with congenital hypoplasia of the cerebellum. The most affected infants are throws born at term. In infants and young children hypotonia and incoordination are the most commonly symptoms, motor milestones and languages typically are delayed. In children over 5 years appears ataxic movements that usually improves
with time, widespread disorder of motor function. Speech typically is slow, jerky, and explosive [18].

Discussions about how different manifestation of CP can be best classified continue to the present day.

Other forms of brain dysfunction are frequently associated with CP. Cognition, vision, hearing, language, cortical sensation, attention, alertness, and behaviour may all be affected by the disorders. It’s not uncommon for CP to be linked to seizures and gastrointestinal problems. The nutrients are not properly absorbed, which has a supplementary impact on the child’s growth and development. Regardless of the severity of the motor deficiency, dyspraxias and agnosia can cause problems with skilled activities. Activities of daily life such as dressing or managing buttons are usually affected. It is thought that the more severe the motor disability, the more likely the kid may acquire further disorders [13].

Conditions associated with CP are pain (3 out of 4 patients), intellectual disability (1 out of 2 patients), speech disorder, bladder control problems (1 out of 4 patients), visual impairment (1 out of 10 patients are blinded), epilepsy (1 out of 4 patients), behaviour disorder, hip displacement (1 out of 3 patients), sleep disorder (1 out of 5 patients), drooling, hearing impairment (1 out of 25 patients are deaf), gastrostomy tube dependence (1 out of 15 patients) [20].

PREVENTION

Preventive measures can be grouped according to the time of birth into prenatal measures, intrapartum measures, and postnatal measures.

To lower the risk of preterm delivery, antenatal methods include providing standard prenatal care [21].

Clinical trials show that giving magnesium sulphate to pregnant women at risk of preterm reduces the frequency and severity of CP in their kids without affecting mortality.

Intrapartum measures are considered to be effective in delaying umbilical cord clamping by at least 30 seconds after birth in energetic premature newborns, and may reduce the incidence of intraventricular haemorrhage [22].

Postnatal measures for preventing CP include supportive, neuroprotective measures for neonates at risk of neurologic injury. These measurements include maintaining adequate ventilation, sufficient cerebral perfusion maintaining normal metabolic status, controlling seizures and treating any underlying cause for encephalopathy.

At 18 months, therapeutic hypothermia improves survival and neurodevelopmental outcomes in newborns with hypoxia and/or encephalopathy [23].

CONCLUSIONS

Early intervention is thought to be the most effective treatment for CP. As soon as the diagnosis is determined, rehabilitation treatment should begin. The earlier a rehabilitation intervention begins, the better the prospects of improving the child’s functional abilities and independence. The intervention targets the child’s motor impairment (e.g., spasticity therapies, orthotics, physical and occupational therapy, orthopedic surgery), as well as the treatment of accompanying comorbidities. The child’s involvement, communication, education, community engagement, and social and emotional development should all be emphasized in the care plan. Supportive therapeutic and educational services, as well as family involvement, all play an important role in encouraging growth [24].

To be able to contribute with capital to the health system, it is necessary to understand epidemiology. The etiology is crucial to know since it may be used to take preventative steps to reduce the likelihood of acquiring CP. Interventions for cerebral palsy (CP) should begin as soon as a diagnosis is suspected to optimize the child’s independence in daily functioning tasks while also reducing the severity of the condition. Comorbidities are common in people with CP, and they can have an influence on their quality of life.

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REFERENCES


