v. 3, n. 60, 2023

International Journal of Health Science

RISK FACTORS AND CLINICAL FORMS OF CEREBRAL PALSY - LITERATURE REVIEW

André Costa Correia
Doctor by: Universidade Estadual de Ciências da Saúde de Alagoas, Maceió, AL, Brazil

Samanttha Cristina da Silva Chaves
Medical course student- Universidade Federal de Catalão, Catalão, GO, Brazil

Palloma Assis Alves Januário
Medical course student- Centro universitário de Maringá, Maringá, PR, Brazil

Noelle Freire Santana
Doctor by: Universidade do Estado do Pará, Belém, PA, Brazil

Victória Ferro da Silva
Medical course student- Centro universitário de Goiatuba, GO, Brazil

Carlos Wagner Leal Cordeiro Júnior
Medical course student- Universidade Nove de Julho, Mauá, SP, Brazil

Elisangela Calmon de Campos
Medical course student- Centro universitário de Maringá, Maringá, PR, Brazil

Deisiane de Almeida Agnelo
Physiotherapist by: Universidade Católica do Salvador, Salvador, BA, Brazil

Rafaela Scremin Frantz
Medical course student- Centro universitário de Maringá, Maringá, PR, Brazil

Bruno Vasconcelos Rossini
Medical course student- Centro universitário de Maringá, Maringá, PR, Brazil

Manuel Agnelo dos Santos Júnior
Orthopedist and traumatologist by: Hospital Espanhol, Salvador, BA, Brazil

Eduardo Barbosa Nunes
Orthopedist and traumatologist by: Hospital Cirurgia, Aracajú, SE, Brazil

Institution: ``Universidade Federal do Catalão``, Catalão, Goiás, Brazil

Conflict of interests: nothing to disclose.

Abstract: **Introduction**: Cerebral Palsy (CP) is one of the clinical forms of chronic non-progressive encephalopathy, with unknown prevalence in Brazil, but with 2:1,000 live births in the USA. **Objective:** To describe the risk factors and clinical forms of CP. **Results**: As prenatal risk factors, we have genetics and maternal and fetal factors, responsible for restricting fetal development. As perinatal, we have inadequate intrapartum conditions that cause suffering to the baby, causing neuronal injury. As postnatal, we have meningoencephalitis, traumatic brain injury, demyelinating diseases, strokes, epilepsies and hypoxic ischemic encephalopathy after cardiac arrest. The spastic form of CP comprises 75% of the cases and, in addition to it, there are also the athetotic, ataxic and flaccid forms. **Conclusion:** The vast majority of CP risk factors are reversible during pregnancy and childbirth. Decentralized prenatal care and adequate treatment are precordial factors in the detection of these risk conditions.
Keywords: Cerebral palsy; Risk factors; Clinical evolution.

INTRODUCTION

Cerebral Palsy (CP) is one of the clinical forms of non-progressive chronic encephalopathy, with predominantly motor-related clinical impairment. In Brazil, the rates are unknown, but in the USA, a prevalence of 2:1,000 live births is estimated (VITRIKAS K, et al., 2020).

Injury to the first motor neuron at any point of its course in the brain (motor cortex, corona radiata, internal capsule, cerebral peduncles) and whose evolution does not worsen or progress over time is its pathophysiology already established (SADOWSKA M, et al, 2020).

There are often other associated neurological dysfunctions in the areas: sensory (speech, vision, hearing), cognitive

(intellectual disability), behavioral (agitation, inattention, anxiety, aggressiveness) and epilepsy. However, motor deficiency is the most important abnormality for defining the term cerebral palsy (SADOWSKA M, et al., 2020).

CP can be caused by a series of factors, divided into pre, peri and post-natal, with predominance in 80% of prenatal cases. The different causes have in common the predilection for compromising the pyramidal motor bundle (PATEL DR, et al., 2020).

As prenatal risk factors, we can mention genetics, which manifests PC when the FBXO31 and RHOB genes, in particular, are mutated. In general, they found that many of the genes implicated in cerebral palsy have important roles in linking brain circuits during early stages of development (FAHEY MC, et al., 2017).

As maternal factors, hypertension, hypotension, hemorrhage and chronic anemia, diabetes mellitus, placental abruption, malposition of the umbilical cord, infection of the placenta, umbilical cord and amniotic fluid are important risk factors for CP, as they restrict the development of the fetus. (VITRIKAS K, et al., 2020).

Congenital infections such as toxoplasmosis, rubella, cytomegalovirus, HIV, syphilis, herpes simplex virus, listeriosis and use of drugs, alcohol or anticonvulsant medication are at risk because they are transmitted to the fetus and also often restrict the neural growth of the fetus (VITRIKAS K, et al., 2020).

Physical factors such as radiation (especially in the first trimester of pregnancy), twins and brain malformations are also considered prenatal risk factors for the development of CP (VITRIKAS K, et al., 2020).

Regarding perinatal risk factors, we have dystocia, asphyxia, intracranial hemorrhage, prematurity (mainly babies <1,000g, who are at high risk of developing periventricular leukomalacia), low birth weight, jaundice (bilirubin encephalopathy), intrapartum infections: listeria, streptococcus or herpes, which cause intrapartum distress to the baby, causing different degrees of neuronal injury. However, it is known that this degree of injury varies individually and there is no absolutely linear correlation between these clinical and laboratory parameters and the severity of the future condition (PATEL DR, et al., 2020).

Neurological abnormalities in the first 24 hours of life can manifest themselves, such as changes in the level of consciousness, presence of seizures, irritability, respiratory changes, feeding difficulties, pupil changes, absence of primitive reflexes and electroencephalographic abnormalities (PATEL DR, et al, 2020).

As postnatal risk factors, we have bacterial and viral meningoencephalitis, traumatic brain injury, demyelinating diseases, cerebrovascular accidents (for example: sickle cell anemia, thrombophilia), epileptic encephalopathies and hypoxic ischemic encephalopathy after cardiac arrest (VITRIKAS K, et al., 2020).

The clinical forms of CP are diverse, and the main ones are the spastic, athetotic or choreoathetotic form, the ataxic form and the flaccid form.

The spastic form comprises 75% of cases, and its clinical types are quadriplegic, hemiplegic and diplegic (SADOWSKA M, et al., 2020).

Spastic quadriplegia (Double Hemiplegia) is the most severe form of CP. There is weakness in all four limbs from the first days of life, which can be symmetrical or asymmetrical. There is axial hypotonia (head, neck and trunk) and appendicular hypertonia. There is microcephaly and one side of the head may become flattened, demonstrating a long stay in the same position (plagiocephaly) (SADOWSKA M, et al., 2020).

The patient does not acquire the neuropsychomotor development milestones such as not holding the head up, rolls over, sits or walks at the expected age. he has very impaired speech, is very irritable, cries frequently and has restless sleep. There may be persistence of primitive reflexes beyond the given time, for example, Moro reflex after six months of age. Seizures are frequent and the diagnosis of epilepsy is made in about 50% of cases. Intellectual disability is often severe but not mandatory. Computerized tomography or magnetic resonance imaging of the skull show typical cystic lesions of periventricular leukomalacia, with white and gray matter lesions (SADOWSKA M, et al., 2020).

In spastic hemiplegia, only one side of the body is compromised and usually the weakness of the upper limb is greater than that of the lower limb (SADOWSKA M, et al., 2020).

Computerized tomography or magnetic resonance imaging of the skull shows cortical atrophy on the side contralateral to the hemiplegia, enlargement of the cortical grooves and dilation of the lateral ventricle. The main cause related to this form is intrauterine or neonatal stroke. Conditions predisposing to thromboembolism, such as antiphospholipid antibody syndrome and coagulation disorders, must be investigated in the mother, as they may be related to the genesis (SADOWSKA M, et al., 2020).

In spastic diplegia, there is weakness in the lower limbs and relative normal strength in the upper limbs. The intellectual prognosis of these patients is excellent, and only a minority evolves with convulsive crises. presence of cysts affecting the pyramidal fibers located in the internal capsule (responsible for lower limb motricity). This clinical form is closely related to prematurity and ischemia (SARVINOZ T, et al., 2022).

The Athetotic or Choreoathetotic (Extrapyramidal) form is a rare form of CP. Affected infants are hypotonic, with poor head control, increased appendicular tone, with development of rigidity and dystonia. PA speech is very difficult and, when acquired, it becomes dysarthric and difficult to understand, usually due to impairment of the oropharyngeal muscles. Swallowing disorders and dysacusis may occur. Some patients manage to acquire gait, but it assumes a grotesque appearance. This form is related to cases of untreated severe neonatal jaundice (kernicterus) and cases of perinatal asphyxia (SARVINOZ T, et al., 2022).

The ataxic form is also a rare form of CP. It is characterized by static and kinetic incoordination. Children with this clinical form have a drunken gait with a wide base (ataxic), static instability, dysmetria, dysdiadochokinesia (inability to perform alternating movements), intention tremors, nystagmus, and dysarthric or scant speech. Usually has an average IQ of 60 (SARVINOZ T, et al., 2022).

Finally, the flaccid form, which is uncommon, has extremely serious motor and intellectual impairment. It must be distinguished from spinal amyotrophies, in which the cognitive is normal (SARVINOZ T, et al., 2022).

MATERIAL AND METHODS

The search was performed in the PubMed database and was limited to articles between 2019 and 2023 that met the criteria of being literature reviews and case reports.

Then, the keywords of the titles of the articles were analyzed and those whose theme best fits our objective were selected.

Six articles were selected for full reading.

DISCUSSION

The vast majority of CP risk factors are reversible during pregnancy and delivery. Therefore, public policies for prenatal care in a simpler and more decentralized way within reach of all women, as well as the treatment of cases that could not be avoided, are precordial factors in the detection of these risk conditions.

CONCLUSION

As prenatal risk factors, we can mention genetics, maternal factors such as hypertension, hypotension, hemorrhage and chronic anemia, diabetes mellitus, placental abruption, malposition of the umbilical cord, infection of the placenta, umbilical cord and amniotic fluid, responsible for restrict fetal development.

Regarding perinatal risk factors, we have dystocia, asphyxia, intracranial hemorrhage, prematurity, low birth weight, jaundice (bilirubin encephalopathy), intrapartum infections: listeria, streptococcus or herpes, which cause intrapartum suffering to the baby, causing different degrees of neuronal injury.

As postnatal risk factors, we have bacterial and viral meningoencephalitis, traumatic brain injury, demyelinating diseases, cerebrovascular accidents (eg sickle cell anemia, thrombophilia), epileptic encephalopathies and post-CA ischemic hypoxic encephalopathy.

The spastic form comprises 75% of cases, and its clinical types are tetraplegic, hemiplegic and diplegic. In addition to these, there are also the Atetotic or Choreoathetotic, ataxic and flaccid forms.

REFERENCES

1. FAHEY, Michael C. et al. The genetic basis of cerebral palsy. Developmental Medicine & Child Neurology, v. 59, n. 5, p. 462-469, 2017.

2. VITRIKAS, Kirsten; DALTON, Heather; BREISH, Dakota. Cerebral palsy: an overview. American family physician, v. 101, n. 4, p. 213-220, 2020.

3. SADOWSKA, Małgorzata; SARECKA-HUJAR, Beata; KOPYTA, Ilona. Cerebral palsy: current opinions on definition, epidemiology, risk factors, classification and treatment options. Neuropsychiatric disease and treatment, p. 1505-1518, 2020.

4. PATEL, Dilip R. et al. Cerebral palsy in children: a clinical overview. Translational pediatrics, v. 9, n. Suppl 1, p. S125, 2020.

5. JIN, Sheng Chih et al. Mutations disrupting neuritogenesis genes confer risk for cerebral palsy. Nature genetics, v. 52, n. 10, p. 1046-1056, 2020.

6. SARVINOZ, Tulanboeva; MUZAFFAR, Zokirov. Rehabilitation for childhood cerebral palsy. Uzbek Scholar Journal, v. 6, p. 97-101, 2022.