Date Received : 17 AUGUST 2023 I Accepted: 05 OCTOBER 2023
Indian Journal of Developmental & Behavioral Pediatrics
(Official Journal of IAP Chapter of Neurodevelopmental Pediatrics)
REVIEW ARTICLE

Access this article online :
Quick Response Code:

Website :
https://ijdbp.in
DOI :
doi.org/10.5281/zenodo.10441309

Impact of the nutritional status on cerebral palsy and neurodevelopment– a scoping review.

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Source(s) of support in the form of grants, equipment, drugs or all of these –nil

Introduction

Cerebral palsy (CP) is primarily a non-progressive neuromotor disorder that affects the development of movement, muscle tone and posture resulting from injury to the developing brain. [1] The impact of Cerebral Palsy on the life of a child is not limited to functional impairment, but is multidimensional. The motor impairment also affects nutritional status, quality of life, developmental status and survival probability. [2] The prevalence of CP for all live births range from 1.5 to 3 per 1,000 live births, with variation between high-income and low to middle-income countries and geographic region. [3,4] Prevalence is as high as 59.18 per 1,000 live births among neonates weighing less than 1,500 Grams and it has remained constant over the recent decade, despite the increased survival of at-risk preterm infants. [5]

Malnutrition includes a group of conditions that refers to deficiencies, excesses, or imbalances in a child's intake, energy, or nutrients.Impaired nutrition, either under or over has devastating impact on overall health and quality of life in children with CP, resulting in increased hospital visits and reduced participation in educational and social activities. Malnutrition is frequently associated with impairment of linear growth, reduced peripheral circulation and wound healing, increased spasticity and irritability. Aetiology of malnutrition is multifactorial including both nutritional and non-nutritional factors. Among the nutritional factors, inadequate dietary intake as a consequence of oral motor dysfunction, gastroesophageal reflux and poor feeding. Among non-nutritional factors, the type and severity of underlying neurological disability, influencing ambulatory and cognitive status, and antiepileptic use are crucial factors involved in determination of the nutritional status.

Henceforth, maintenance of adequate nutrition is critical and malnutrition is common among this vulnerable population. [6–8]

Causes of nutritional problems in CP children

The causal pathway to malnutrition among children with CP is yet not clearly defined particularly in LMIC (low and middle income countries) settings, though several studies found a significant association between malnutrition, severity of motor impairment (e.g., Gross Motor Function Classification System (GMFCS) level III–V, tri/quadriplegia) and associated impairments (e.g., intellectual, speech, hearing)

Table:1 Causes of malnutrition in children with Cerebral palsy

❖ Feeding difficulties • Oropharyngeal Dysphagia • Recurrent aspirations • Oromotor dysfunction • Inadequate intake • Gastroesophageal reflux	❖ Altered metabolism ❖ Constipation ❖ Micronutrient deficiency ❖ Non nutritional factors • Cognitive impairment • Antiepileptic therapy

among children with CP. The prevalence of undernutrition increases with older age, lower intelligence quotients, and more severe neurological impairment.[9–11] The causes of malnutrition in CP may be broadly divided into nutritional and non-nutritional.(Table:1) Nutritional causes are further divided into-

1. Feeding Difficulties

The life expectancy of children with CP has gradually improved and thus, the prevalence and consequences of feeding difficulties are on the rise.

A recent data from the North American Growth in CP Project showed that 58% of children with moderate to severe CP had feeding difficulties, of which 23% were severe.[12] Feeding disorders play an important role in the development of malnutrition, documented in 29%–46% of CP children.

A. Oropharyngeal Dysphasia

Oropharyngeal dysphagia characterized by problems in any or all phases of swallowing often result in CP children having reduced lip closure, poor tongue function, tongue thrust, exaggerated bite reflex, tactile hypersensitivity, delayed swallow initiation, reduced pharyngeal motility and drooling. Impaired oral sensorimotor function can result in drooling which in turn results in impaired hydration. Problems with swallowing liquids are common and are usually related to a timing deficit with delayed pharyngeal swallow initiation. Residues in the pharynx due to reduced pharyngeal motility results in problems with thick smooth, lumpy or mashed foods. Children may appear to handle thicker food and liquid more easily, as they have more time to initiate a swallow. A clinical feeding/ swallowing evaluation or simple observation of children while they are eating and drinking, may not define the pharyngeal physiology of swallowing in the CP children. Though providing more time to complete feeding tasks may reduce their difficulty, one should be cautious as fatigue may set-in and reduced attention to the task also may become a factor. Children requiring meal time > 30 min, regularly, often point to a feeding/ swallowing problem.

B. Aspiration of food contents

Chronic aspiration is a pressing issue in this patient group with absence of cough response during aspiration events making it difficult to delineate it resulting in hypoxic events during oral feeding. Over time, as developmental gains are made, this risk of aspiration may decrease in children with CP, although it is not unusual

for them to have increasing dysphagia as they reach puberty. At this point of time changes in nutritional needs along with possible increased scoliosis or kyphosis sets in making it difficult. Further, the risks of aspiration complications are dependent partially on the initial condition of the child. [13]

C. Oromotor dysfunction

This dysfunction affects up to 90% of CP and is a major contributor to malnutrition in children with cerebral palsy. [14,15] Daily intake of nutrient depends on getting adequate food, as well as on the ability to adequately chew and swallow the food. These factors are associated with the functional status of a child with CP. Parents often report poor suck, breastfeeding difficulties, problems with the introduction of solid food and choking, prior to the making of diagnosis of CP.

D. Inadequate intake

The caloric intake of children with CP is lower than that of age-matched controls. [16] Though some patients can self-feed independently, lack of hand-mouth coordination may result in them spilling an excessive amount of food. These children may also eat more slowly than other children of same age group in the school and hence require more time to eat than is allowed by the school schedule. Hence, regular family or school mealtime may be insufficient for them to ingest a sufficient amount of food.

Severely affected children are dependent on a caregiver at mealtime and are often unable to communicate hunger and satiety. The caregiver regulated food intake may result in underfeeding because the caregiver often overestimates the time spent feeding the child or will overestimate the child's caloric intake. The caloric intake could be improved by thickening consistency and making the food calorie-dense.

Gastro esophageal reflux

Gastro esophageal reflux affecting a significant proportion of children with CP, frequently result in emesis and regurgitation, acting as a cause of caloric loss. Reflux esophagitis may be lead to food refusal, further decreasing food intake.

2. Altered metabolism

The hypotonic and non-ambulatory child requires few calories above the resting energy expenditure to thrive. But, children with hypertonic or athetoid CP may require more calories. So also are the ambulating children with mild to moderate diplegic or hemiplegic CP, often requiring more calories to perform daily activities than their normal counterparts. [17]

Most recent European Society for Paediatric Gastroenterology Hepatology and Nutrition (ESPGHAN) guidelines for the evaluation and treatment of gastrointestinal and nutritional complications in children with neurological impairment supports these findings. [18]

3. Constipation

Chronic constipation (CC), frequently seen in children with cerebral palsy, is estimated to have a prevalence ranging from 26% to 74% in children with severe disabilities. Contributory factors include abnormal bowel mobility, prolonged immobility, skeletal abnormalities, generalized hypotonia and reduced fluid and fiber intake. If chronic constipation is not catered to, it gives rise to several gastrointestinal complications such as, chronic nausea, recurrent vomiting, abdominal pain, early satiety with food refusal and poor dietary intake.

4. Micronutrient deficiencies in Cerebral Palsy

Micronutrients are important for many metabolic pathways with their deficiencies causing symptoms, which are often difficult to distinguish from the general neurologic impairment of CP children. Low micronutrient levels may indeed affect cognition, behaviour, social interaction, developmental outcomes and hence quality of life. It was shown in clinical trials that biochemical deficiency of micronutrients is common in children with CP, indicating that dietary intakes of vitamins and minerals are often too low to balance needs in this population.

A study conducted by Kalra et al.(2015) comparing the micronutrient levels in 50 children with CP (aged 2–12 years) and neurologically normal age and sex matched controls found that the serum levels of iron, copper and magnesium were significantly lower in children with CP. Furthermore, the exclusively tube-fed children, may develop nutrient deficiencies, because enteral formulas may provide adequate amounts of micronutrients only when volumes consumed meet their age-related daily recommended intakes for energy. Many CP children require lower energy intakes posing them at risk for low micronutrient intake. Papadopoulos et al.(2008) found a high incidence of anemia in patients with CP , with 87% having anemia and 95.7% iron deficiency when on liquid diet.[19]

Selenium deficiency is seen in children with long-term enteral nutrition, as many medical nutrition products do not contain adequate content of selenium. Selenium is an essential trace element and a component of selenoproteins. Carnitine deficiency is relatively common in children with epilepsy. Approximately 75% of carnitine is obtained from the diet and its deficiency can cause muscle weakness, cardiomyopathy and in severe cases also hypoglycaemia, abdominal pain, vomiting and hepatomegaly. Those children on multiple antiepileptic drug therapy (especially valproic acid), young age (<10 years), neurological disability (intellectual disability, cerebral palsy and microcephaly), a diet deficient in meat and dairy products, tube feeding or parenteral nutrition are at risk of carnitine deficiency.

5. Other nutritional factors that may result in inadequate energy and nutrient intake

- Sensory factors related to the texture and taste of foods can result in the consumption of a limited repertoire of foods that may be nutritionally incomplete.
- Negative feeding behaviours related to mealtime stress or discomfort
- Disturbances in the sensation of hunger and satiety
- Dental caries and dental malocclusion affect the quantity of food consumed

Non-Nutritional Factors

Among the non-nutritional factors impacting on dietary intake and nutritional status in CP children, cognitive impairment and prolonged use of antiepileptic medication are involved.

A. Cognitive Impairment

Intellectual Disability/ Mental retardation as well as hearing, language, visual, and behavioural disorders are often associated to CP. Cognitive impairment may result in inability to communicate hunger or satiety, inability to request food and drink and to communicate symptoms. It is known through

literature that the prevalence of malnutrition increases with lower intelligence quotients (IQs). Sánchez- Lastres et al.(2003) [20] showed that malnourished children had significantly lower mean IQs than those with normal nutrition; with severity of IQ deficit increasing with increasing malnutrition levels. Similar findings were reported by others also. [21]

B. Severeity of disability

Many studies in past have [11,22,23] analysed the risk of undernutrition among CP children based on their Gross Motor Function Classification System (GMFCS) level. The odds of having moderate and severe undernutrition was 4 and 14 times more respectively, in GMFCS level IV-V when compared with GMFCS level I-III.

C. <u>Antiepileptic Therapy</u>

CP children with gastrointestinal disturbances and bone disease are often affected by epilepsy requiring a long-term antiepileptic management (AEDs).

- ***Gastrointestinal disturbances:*** These are mainly feeding difficulties including anorexia and food refusal. Additionally, nausea, vomiting and dyspepsia secondary to gastric intolerance may contribute. Rarely, diarrhoea, weight loss, abdominal cramps and constipation may also occur. The above-described gastrointestinal side effects may contribute to poor nutritional status in these children. [24]

-Osteopenia: CP children with seizures on long-term AEDs, are at increased risk for metabolic bone disease resulting in bone turnover, osteoporosis, alterations in bone quality, and fractures. Pediatric age group being a critical period for bone mineralization, this issue is particularly important. The bone mineral density is achieved by the end of adolescence and this determines the risk for pathological fractures and osteoporosis later in life. Seizure-related falls, in addition to antiepileptic treatment, pose an increased risk for fractures. Fractures are reported to be two to six times more common in patients with epilepsy than in the general population. Some AEDs like phenytoin, phenobarbital and carbamazepine are inducers of hepatic enzyme cytochrome P-450 (CYP- 450); increasing the catabolism of vitamin D and inducing a state of hypovitaminosis D with subsequent hyperparathyroidism, increased bone turnover thereby reducing the bone density. But, use of non-enzyme inducing AEDs and polytherapy have also been implicated with vitamin D deficiency and osteopenia. It is known that long-term use of VPA is associated with bone metabolism abnormalities, including reduction in BMD and changes in bone turnover, in a dose dependent way. However, the effects of the newer AEDs such as gabapentin, lamotrigine, levetiracetam, oxcarbazepine, topiramate and zonisamide on bone and calcium metabolism need further evaluation due to paucity of published evidences.

Effect of malnutrition in Children with CP

The damaging effects of malnutrition on physiology, motor function, neurological and psychological function may be particularly detrimental during early child development. These effects are due to different factors like hormonal problems, deviant motor functionality and neurological functional limitation contributing to the same.[14] In addition to growth failure,

decreased cerebral function, reduced potential for development, impaired immune function, impaired circulation with poor wound healing, diminished respiratory muscle strength, all may be seen in these children.

In addition to decreased strength of cough, the decreased muscle strength resulting from malnourishment cause recurrent respiratory tract infection in CP children. Moreover, malnutrition may hamper the resolution of these infections subsequent to lower immune function. Impaired wound healing and immunity, increases the risk of postoperative complications following surgery for fundoplication [25] and scoliosis repair. [26] It also leads to diminished immune function, causing increased susceptibility to infection.

Brooks and colleagues (2007) [27] concluded that CP children with malnutrition have a greater number of secondary and chronic health conditions than children who are better nourished. Similarly, Rempel researchers in the North American Growth in Cerebral Palsy Research Collaborative (2006) [12] noted that undernourished children with CP with low muscle mass have poorer general health; but those with low fat reserves have increased health care utilization and decreased participation in school and family activities.

Children with CP who had increased fat mass, specially fed via gastrostomy tubes were noted to have increased risk of osteopenia because of rapid accrual of body fat than bone minerals, direct effect of the excess weight on the bone and impact of the fat mass itself on the bone mineral density.

Good nutrition being a powerful prognosticator of survival in CP children at all levels of motor involvement, those who are poorly nourished are at increased risk of mortality. Hence, periodic measurement of nutritional indicators should be an important aspect of routine health care for all CP children. In addition, these parameters should be compared against reference standards, or norms to serve as a screening tool for health problems. [28]

Impact of malnutrition on developmental status of otherwise healthy children

A child's nutritional status is highly correlated with its development.[29] While the entire period of childhood is important for development, the first 1000 days (conception to age 2) are critically important for brain development; with the most rapid and prolific development of neural pathways happening then, first in sensory development, then language skills and then higher cognitive functioning.[30] Stressors during this period can substantially impact the architecture of the developing brain. Adverse events that results in malnutrition like illness (anaemia, diarrhoea, poor feeding, micronutrient deficiencies, comorbidities) can all negatively impact cognitive development.[31]

Malnutrition and Developmental status of CP children

Children with CP specially in settings of poverty are often faced with many of the above mentioned stressors. Some of these factors, if identified early enough, are potentially modifiable through intervention, such as managing feeding difficulties, treating or preventing malnutrition.[32] Nutritional rehabilitation has been associated with improved overall health, improved peripheral circulation, healing of decubitus ulcers, decreased spasticity, and decreased irritability in patients with cerebral

palsy.[33] As it is in typically developing children, development is also affected in children with cerebral palsy and malnourished CP patients may have poorer DQ scores as compared to well-nourished CP children. [34,35,36] Considering the paucity of quality data on the developmental implications of malnutrition on children with cerebral palsy, more detailed studies in this regard is required to address it.

CONCLUSION

Cerebral palsy children are at increased risk of malnutrition due to several nutritional and non-nutritional factors. Among the nutritional factors, insufficient dietary intake as a consequence of feeding difficulties is one of the main issues. Feeding problems are frequently secondary to oropharyngeal dysphagia, which usually correlates with the severity of motor impairment and presents in around 90% of preschool children with cerebral palsy (CP) during the first year of life. Other nutritional factors are represented by excessive nutrient losses, often subsequent to gastroesophageal reflux and altered energy metabolism. Among the non-nutritional factors, the type and severity of neurological impairment, ambulatory status, the degree of cognitive impairment, and use of antiepileptic medication altogether concur with determination of nutritional status.

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