BIOCHEMICAL AND MORPHOMETRIC PREDICTORS OF ORAL DISEASE IN PAEDIATRIC PATIENTS WITH CEREBRAL PALSY

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https://doi.org/10.5281/zenodo.17461652

ARTICLE INFO

Received: 25th October 2025
Accepted: 26th October 2025
Online: 27th October 2025

KEYWORDS

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ABSTRACT

Cerebral palsy (CP) represents one of the most clinically complex neurodevelopmental disorders in childhood, with long-term somatic, neurological and maxillofacial consequences. In recent years, the prevalence of CP and related secondary comorbidities has demonstrated a steady increase across many regions of the world. Among the most frequent associated manifestations are disorders of the oral cavity, where the prevalence of dental and periodontal pathologies in children with CP ranges from 42% to 95.3%, significantly exceeding rates observed in neurologically healthy pediatric populations.

Introduction

Cerebral palsy (CP) represents one of the most clinically complex neurodevelopmental disorders in childhood, with long-term somatic, neurological and maxillofacial consequences. In recent years, the prevalence of CP and related secondary comorbidities has demonstrated a steady increase across many regions of the world. Among the most frequent associated manifestations are disorders of the oral cavity, where the prevalence of dental and periodontal pathologies in children with CP ranges from 42% to 95.3%, significantly exceeding rates observed in neurologically healthy pediatric populations.

This unfavorable epidemiological trend is explained not only by motor dysfunction and impaired neuromuscular control, but also by underlying pathogenetic alterations affecting salivary composition, mineral exchange, craniofacial skeletal development, mastication biomechanics and orofacial musculature maturity. The oral pathologies in CP often present as complex, combined forms, which progress rapidly and are frequently resistant to conventional approaches of pediatric dental care.

Moreover, early clinical manifestations are frequently masked or misinterpreted, especially in the absence of standardized pathogenetic diagnostic criteria. Traditional dental screening based solely on visual examination and caries indices is not sufficient for complex disorders such as CP. Today, evidence increasingly shows that biochemical markers of salivary homeostasis, immune imbalance, and disturbances of oxidative–reductive reactions may serve as earlier and more reliable indicators of pathological change compared with clinical symptoms alone.

Keywords

Cerebral palsy; pediatric dentistry; pathogenetic mechanisms; craniofacial development; oral fluid biochemistry; oxidative stress; multidisciplinary diagnosis.

Main Part

Children with CP exhibit distinctive pathogenetic mechanisms that differentiate them sharply from healthy peers. The chronic neuromotor impairment leads to reduced oral self-cleaning, altered muscular tone of the orbicularis oris and masticatory muscles, dysphagia, drooling and mouth breathing. These functional disturbances accelerate plaque accumulation and increase the susceptibility of both hard tissues and periodontal structures to inflammation and demineralization.

Beyond functional impairment, modern studies confirm a close correlation between anthropometric asymmetry of the craniofacial complex and biochemical dysregulation of oral fluid in CP. Morphometric evaluations have documented delayed maxillo-mandibular growth, micrognathia or narrowed upper arch forms in up to 63% of examined children, as well as altered occlusal development linked with neurogenic muscular hypotonia or spasticity. These skeletal deviations not only impair aesthetics but contribute directly to traumatic occlusion, abnormal masticatory loading, and progressive periodontal destruction.

Biochemical assessment of oral fluid in children with CP demonstrates a persistent imbalance between pro-oxidant and antioxidant systems, which reflects systemic metabolic stress and impaired local homeostasis. Elevation of inflammatory cytokines, reduction of calcium–phosphate ions, lowered buffering capacity and increased markers of oxidative stress collectively form an unfavorable oral microenvironment that accelerates enamel demineralization and soft-tissue breakdown. These mechanisms explain why dental caries and periodontal disease in children with CP are not merely behavior-dependent, but pathogenetically conditioned.

Additionally, oral breathing — observed in more than 55% of pediatric neurogenic cohorts — aggravates mucosal dryness, alters pH levels, and disrupts normal microbiota composition, further predisposing to gingival inflammation and enamel erosion. The pathogenetic model is therefore not linear, but multilevel: neuromuscular impairment → craniofacial dysmorphogenetic → salivary biochemical dysregulation → combined dental pathology.

A clinically important implication of these findings is that early diagnosis should not be restricted to routine clinical examination alone. Instead, it requires integration of morphometric, biochemical and immunological diagnostic criteria in order to detect pathological progression before irreversible tissue damage occurs.

Results and Discussion

Recent clinical evaluations of children with cerebral palsy (n=299) have shown that:

Clinical indicator	Prevalence
Combined dental pathology	60%
Malocclusion	up to 63%

Periodontal inflammation	78–95%
Mouth breathing	>55%

These data confirm that the oral health burden in CP is not secondary, but directly rooted in the underlying pathophysiology of the disorder. Therefore, early predictive markers — such as local oxidative stress indicators and morphometric skeletal deviations — can be used as screening tools for primary prevention.

International evidence now supports a multidisciplinary care model that integrates pediatric dentists, neurologists, physiotherapists and psychologists. Such an approach surpasses symptomatic treatment, addressing both causative and compensatory mechanisms. In Uzbekistan, the introduction of an individualized "medical-social card" for CP children presents a viable mechanism for systematic follow-up and tailored intervention.

Conclusion

Oral disease in children with cerebral palsy is driven by complex pathogenetic mechanisms that combine neuromotor impairment, craniofacial dysmorphogenetic and biochemical disruption of local homeostasis. The high prevalence and combined forms of pathology justify a revision of traditional diagnostic strategies. Predictive screening based on morphometric and salivary biochemical indicators enables earlier detection, improved treatment planning and greater clinical effectiveness. Consequently, a personalized, interdisciplinary diagnostic model should be regarded as a standard of care for this vulnerable patient group.

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