PROTEIN AND MICRONUTRIENT STATUS (CA, MG, ZN, CU) AS MODIFIABLE FACTORS IN THE CORRECTION OF CONNECTIVE TISSUE DYSPLASIA IN CHILDREN

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Introduction: Connective tissue dysplasia (CTD) is among the most common multidisciplinary pathologies in pediatric practice. It serves as a biological background for the development of chronic diseases, ranging from orthopaedic disorders to cardiovascular complications. While the genetic nature of CTD is well-established, the search for modifiable environmental factors that can influence its progression remains a high priority for clinical research.

The pathophysiology of CTD is rooted in defective collagen fibrillogenesis and impaired extracellular matrix metabolism. From a biochemical standpoint, collagen is a unique protein whose synthesis requires a precise supply of amino acids—primarily glycine, proline, and lysine. Consequently, adequate protein nutrition is not merely a general health recommendation but a specific therapeutic necessity for children with CTD. Hypoalbuminemia or a low dietary protein intake can limit the substrate availability for collagen renewal, potentially worsening tissue laxity.

Simultaneously, the enzymatic cascades driving collagen maturation are metal-dependent. Zinc (Zn) is essential for the activity of collagenases and tissue repair mechanisms, while Copper (Cu) is non-replaceable in the final stages of extracellular matrix stabilization. Magnesium (Mg) and Calcium (Ca) further modulate the mechanical properties of bone and cartilage. Critically, these nutrients do not act in isolation; there is a known metabolic interplay. For instance, zinc can competitively inhibit copper absorption in the gut, and protein status influences the transport (albumin) of these metals in the bloodstream.

Currently, there is a significant gap in the literature regarding the combined assessment of dietary intake (protein and micronutrients) and actual serum levels in children with CTD. Most therapeutic strategies focus on symptomatic pharmacological treatment, often overlooking the foundational role of nutritional status.

This study aims to fill this gap by conducting a comprehensive evaluation of the nutritional status (protein intake, serum albumin) and micronutrient levels (Ca, Mg, Zn, Cu) in children with varying degrees of CTD severity. We seek to determine whether a combined deficiency in these parameters constitutes a distinct clinical-biochemical phenotype that requires targeted nutritional intervention.

Methods: A cross-sectional study will be conducted on 80 children with CTD. Dietary intake will be assessed using 24-hour recall and food frequency questionnaires to calculate daily protein, Calcium, Magnesium, Zinc, and Copper consumption. Biochemical analysis

will include serum total protein, albumin, and the aforementioned trace elements. Clinical manifestations (joint hypermobility, asthenic syndrome, scoliosis) will be recorded.

Expected Results: We anticipate detecting a high prevalence of combined protein and micronutrient deficiencies. We expect that low protein intake will correlate with low serum albumin and reduced levels of Zn and Cu (due to impaired transport). This combined deficiency is expected to correlate directly with the severity of asthenic syndrome and poor posture in the studied children.

Conclusion: The effectiveness of CTD correction in children depends not only on pharmacological support but also on the complete nutritional supply of building materials (protein) and cofactors (Zn, Cu, Mg). The data supports the need for comprehensive nutritional support as a fundamental part of CTD management.