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ISSN: 2161-0509
Journal of Nutritional Disorders & Therapy

Opinion Article

Clinical Overview of Molybdenum Deficiency Syndrome

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DESCRIPTION

Molybdenum deficiency syndrome represents an uncommon metabolic disorder associated with insufficient functional activity of molybdenum-dependent enzymes. Molybdenum is a trace element required for proper function of several enzyme systems involved in oxidation reactions within human metabolism. While dietary intake of molybdenum is generally adequate in most populations due to its presence in plant-derived foods and water sources, deficiency-like conditions may still arise due to genetic defects, long-term nutritional imbalance, or impaired absorption. These disturbances can lead to abnormal accumulation of metabolic intermediates that influence neurological, renal, and systemic physiological processes.

The biological importance of molybdenum lies in its role as a cofactor for enzymes such as sulfite oxidase, xanthine oxidoreductase and aldehyde oxidase. These enzymes facilitate the conversion of reactive metabolic substances into excretable end products. When molybdenum-dependent activity is reduced, metabolic intermediates such as sulfite and xanthine may accumulate. This accumulation can interfere with normal cellular processes and contribute to biochemical instability across multiple organ systems.

Sulfite oxidase dysfunction is one of the most clinically significant consequences of molybdenum-related metabolic impairment. Under normal conditions, sulfite is converted into sulfate, a relatively inert compound that is safely eliminated. When this conversion is impaired, sulfite levels may rise and exert toxic effects on neural tissue. In severe cases, this can affect neurological development and function, leading to symptoms such as developmental delay, muscle tone abnormalities, and seizure activity. The central nervous system is particularly sensitive to fluctuations in sulfur compound concentrations, making it vulnerable to even minor disruptions in this pathway.

Purine metabolism is also affected in molybdenum-dependent conditions. Xanthine oxidoreductase participates in the breakdown of purine nucleotides into uric acid. Reduced enzyme activity may result in elevated levels of xanthine and hypoxanthine, along with altered uric acid production. These biochemical changes may influence renal excretion patterns and, in some instances, contribute to the formation of crystalline deposits in urinary pathways. Although not universally present, these findings provide useful biochemical indicators in diagnostic evaluation.

The causes of molybdenum deficiency syndrome can be grouped into nutritional insufficiency, medical treatment-related factors, and inherited metabolic defects. Nutritional deficiency is uncommon but may occur in populations with extremely limited dietary diversity or in individuals with long-term gastrointestinal absorption disorders. More frequently, deficiency states are observed in patients receiving prolonged parenteral nutrition without adequate trace element supplementation. In such situations, gradual depletion of molybdenum-dependent enzyme activity may occur over time.

Inherited forms of the disorder are linked to defects in molybdenum cofactor synthesis. These genetic alterations prevent activation of molybdenum-dependent enzymes even when dietary intake is sufficient. Such conditions often present in early infancy with severe neurological symptoms and biochemical abnormalities. Diagnosis typically relies on specialized laboratory assays that detect abnormal metabolite accumulation in biological fluids, along with molecular genetic testing to identify causative mutations.

CONCLUSION

Clinical manifestations vary depending on severity and underlying cause. Neurological involvement is often prominent in severe cases and may include developmental delay, seizures and abnormal neuromotor function. In less severe or partial deficiency states, symptoms may be subtle and include fatigue, mild neurological disturbances, or laboratory-detected biochemical irregularities without obvious clinical signs. Management approaches depend on the underlying etiology. Nutritional deficiency can often be addressed through dietary

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Received: 25-May-2026, Manuscript No. JNDT-26-32154; **Editor assigned:** 27-May-2026, PreQC No. JNDT-26-32154 (PQ); **Reviewed:** 10-Jun-2026, QC No. JNDT-26-32154; **Revised:** 14-Jun-2026, Manuscript No. JNDT-26-32154 (R); **Published:** 24-Jun-2026, DOI: 10.35248/2161-0509.26.16.372.

Citation: Nadeau J (2026). Clinical Overview of Molybdenum Deficiency Syndrome. J Nutr Disord Ther. 16:372.

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correction and appropriate trace element supplementation. In individuals receiving long-term intravenous nutrition, careful formulation of nutritional solutions is necessary to ensure adequate trace element provision. Regular monitoring of biochemical markers is essential in these settings to prevent metabolic imbalance.