

New Insights into Celiac Disease and Micronutrient Deficiencies in Pediatrics

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Abstract This editorial highlights the role of micronutrient deficiencies in the clinical presentation of celiac disease. The clinical presentation of celiac disease in children is apparently changing from forms with evident gastrointestinal signs and symptoms to frequent silent clinics. Manifestations of micronutrient deficiencies are frequently found now, especially that of Fe and Zn, but also many other micronutrients, sometimes observed after many years of diagnosis. Celiac disease can be suspected when an unexplained micronutrient deficiency is found and it is recommended to assess micronutrient status periodically during the course of disease.

Keywords: *celiac disease, children, micronutrient deficiencies*

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1. Introduction

During the past decades, the management of celiac disease (CD) has been challenged with respect to changes in its clinical course, with a more silent clinical presentation as more frequent rather than the chronic gastrointestinal disease and also diagnosed primarily in older age groups.

Micronutrient deficiencies are being diagnosed more frequently both in the usual clinical presentation, in which malabsorption is evident, and in those with silent clinical presentation. Decreased micronutrient intake, increased loss due to enteropathy and malabsorption, metabolic disturbances and increased renal loss have been proposed as contributing factors to micronutrient deficiencies.

Published micronutrient deficiencies in CD have included: Fe, Zn, Cu, Ca, Se, folate, vitamin E, D, B12 and B6. Every micronutrient requires complex mechanisms for intestinal absorption, transportation through the enterocyte and exocytosis to the blood stream.

Ferropenic anemia has been described in CD as an exclusive sign, being the most common extraintestinal sign. Iron absorption requires specific mechanisms, specific chaperones and transporters including: the heme and folate transporter for HEM iron, FTR1-endocytosis for non-HEM ferritin iron (provided mainly by legumes), and DMT1 for iron salts or chelates (as pharmaceutical supplements or as food fortification). Ferroportin is an iron efflux protein, regulated by the hepcidin, a specific hormone. These complex mechanisms allowing human iron absorption are well regulated by several genes, and interacting with environmental factors (e.g. diseases, dietary iron intakes, gliadin intake) resulting in normal iron homeostasis. Research providing insight into how all these processes adapt to the celiac condition is scarce.

Zn deficiency is frequent in CD and is associated with growth delay and immune alterations. Its absorption also requires the use of specific transporters: ZnT and SLC-ZIP. Within Zn absorption regulation by these transporters, Zn absorption presents a partial homeostatic regulation, with increased absorption in some clinical conditions with decreased zinc body stores, and a decreased absorption rate in conditions of normal Zn status. Finally, the relationship of intestinal excretion of endogenous zinc to zinc absorption is also important in zinc homeostasis and requirements.

Even though the main basis for vitamin D metabolic status is the activation of subdermal vitamin precursors by sun-UVB rays, compromise to the small bowel may affect activity and vitamin D absorption. Pathophysiology of vitamin B12 deficiency in CD is unknown; it must be suspected in CD patients presenting neurological and haematological alterations, mainly after many years of CD. Copper deficiency has been described mainly in adult CD patients.

Along with the physiological processes for the absorption of every micronutrient, children presenting celiac disease are frequently living in different environmental conditions which affect the risk for micronutrient deficiencies: diet components vary considerably between communities, intake of foods containing specific micronutrient is frequently decreased, other diet components may affect micronutrient bioavailability, and infectious or parasitic diseases frequently decrease its absorption.

2. Conclusions

Micronutrient deficiencies should be periodically studied through CD follow-up; celiac disease must be

suspected if clinical signs of micronutrient deficiencies are diagnosed.

Future research into this issue may include: an assessment of the mechanisms underlying the effects of micronutrient deficiencies on the clinical course of celiac disease; the role of intestinal micronutrient transporters in micronutrient malabsorption in the celiac disease and the effect of micronutrient supplementation on the clinical course of disease.

References

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