Serum carnitine and selenium levels in children with celiac disease

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Background and Aim: Celiac disease (CD) is a gluten-induced enteropathy that results in malabsorption of nutrients. We studied the serum levels of carnitine and selenium in children with CD. Methods: Serum levels of free carnitine and selenium were studied in 30 children (mean age 8.1 [4.4] years) with CD and 30 age- and gender-matched healthy children. All patients had type 3 duodenal lesions. The mean (SD) serum levels of free carnitine and selenium were lower among patients with CD (24.5 [7.7] μmol/mL and 52.1 [12.9] μmol/mL, respectively) than among healthy controls (29.4 [9.2] and 65.1 [17.2] μmol/mL; p<0.05 each). Levels were similar in children with and without diarrhea. Conclusions: Serum carnitine and selenium levels are decreased in children with CD, probably due to malabsorption. [Indian J Gastroenterol 2004;23:87-88]

Key words: Malabsorption, nutrient, trace element

Celiac disease (CD) is characterized by permanent intestinal intolerance to wheat gliadin and similar proteins. In patients with CD, intestinal absorptive surface is decreased, leading to malabsorption of nutrients. The duodenum is the main site for absorption of selenium and carnitine. Carnitine, which is crucial for mitochondrial energy production, has a particular importance in skeletal and heart muscle. Its deficiency causes muscular hypotonia and weakness, which are common in CD.

Selenium is an integral part of the enzyme glutathione peroxidase, which forms a major cellular defense system against oxidative injury. Selenium deficiency has been incriminated in causation of many diseases including malignancies. Increased incidence of gastrointestinal malignancies in celiac patients is well known. We therefore estimated serum free carnitine and selenium levels in children with active CD.

Methods
The study included 30 consecutive children (18 girls) with CD diagnosed based on the revised ESPGHAN criteria between November 2000 and December 2001. Thirty age- and gender-matched healthy children (14 girls) formed the control group.

Serum free carnitine levels (normal range 22-50 μmol/mL) were measured using a tandem mass spectrometer.
long-chain fatty acids across the inner mitochondrial membrane for oxidation. Dietary intake and absorption are important for maintaining tissue stores of carnitine, besides its endogenous synthesis. It has been reported that carnitine concentrations do not correlate with degree of mucosal atrophy. In addition to poor absorption, poor dietary intake may also play a part in carnitine deficiency in patients with CD. Previous studies have shown normalization of serum carnitine concentrations after institution of gluten-free diet.

Carnitine deficiency is associated with signs and symptoms related to alterations of mitochondrial metabolism. Although none of our patients had symptoms of carnitine deficiency, recent reports suggest that decreased muscle tone, moderate muscle weakness and liver dysfunction in patients with CD may be related to low plasma carnitine concentration; gluten-free diet and carnitine supplementation resulted in improvement of muscle tone.

Selenium, an essential element, is deficient in many conditions such as total parenteral nutrition, cancer and muscular dystrophy. Low selenium levels in patients with CD appear to be related to malabsorption. In addition, these may be related to low selenium intake; this may be particularly important in countries like Turkey. Selenium intake is known to be dependent on the content of the soil and foodstuffs. Although serum levels of selenium were low in patients with CD, we found no clinical symptoms associated with this. A relative deficiency may be associated with an increased risk of developing cancer, in particular gastrointestinal cancers, cardiovascular diseases and immune deficiency states. Patients with CD have an increased frequency of gastrointestinal tumors; it is not clear whether selenium deficiency may play a role in this. In view of a narrow margin between the safe and toxic doses of selenium, its supplementation in therapeutic doses is not recommended in patients with CD; instead, choosing foodstuffs relatively high in selenium content may be advisable.

In conclusion, we found low serum carnitine and selenium concentrations in children with CD, though the patients had no clinical findings related to these deficiencies. The clinical importance of these findings and need for supplementation of these nutrients in patients with CD need further investigation.

References

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