

Inflammatory Bowel Disease and Mineral Malnutrition Crohn's Disease and Ulcerative Colitis Present Problems

It has been clearly observed that inflammatory bowel disease (IBD) commonly leads to malnutrition. What is IBD? Inflammatory bowel disease is an idiopathic (unknown cause) chronic inflammatory disorder of the gastrointestinal system. There are two major forms of IBD. They are Crohn's disease (CD) and ulcerative colitis (UC). Crohn's disease, also called

regional enteritis, is a nonspecific granulomatous inflammatory disease usually affecting the lower ileum, but often involving the colon and occasionally other parts of the gastrointestinal tract. Ulcerative Colitis is a chronic, nonspecific inflammatory and ulcerative disease of the colon, characterized most often by bloody diarrhea¹. What is it about IBD that leads to malnutrition? The causes of malnutrition in people with IBD are multifactorial and have been listed in the Table 1 below².

while ulcerative colitis can cause acute reduction in nutrient levels during disease flare ups. It is very common for people with IBD to have protein-energy malnutrition. Food intolerances are twice as common among IBD patients. Combine food intolerance, malabsorption problems, decreased appetite, and less pleasure associated with food intake, and it becomes obvious that IBD can lead to malnutrition. Patients with IBD commonly have many micronutrient deficiencies, as seen in Table 2 below².

Crohn's disease normally leads to chronic malnutrition, which develops insidiously over long periods of time,

Patients suffering from IBD have been seen to have low levels of the

Table 1.

Causes of Malnutrition in IBD Patients

REDUCED DIETARY INTAKE

- Anorexia to avoid symptoms
- Restricted diets
- Drug-induced taste alterations

MALDIGESTION AND MALABSORPTION

- Inadequate mucosal surface
- Bile salt malabsorption from ileal disease
- Bacterial overgrowth
- Drug induced
- Increased requirements
- Inflammatory catabolism
- Drug-induced nutrient wasting

EXUDATIVE PROTEIN LOSSES FROM INFLAMED INTESTINE OR FISTULAE

Table 2.

| MICRONUTRIENT DEFICIENCIES IN IBD | | |
|-----------------------------------|-----------------|-------|
| MICRONUTRIENT | % PREVALENCE IN | |
| | UC | CD |
| Iron | 81 | 39 |
| Folic Acid | 35 | 54-67 |
| Vitamin B-12 | 5 | 48 |
| Potassium | | 6-20 |
| Calcium | | 13 |
| Magnesium | | 14-33 |
| Vitamin A | 26-93 | 11-50 |
| Vitamin D | 35 | 75 |
| Zinc | | 40-50 |
| Selenium | | 35-40 |

minerals zinc and selenium, which are cofactors for the oxidant protective enzyme systems, as well as low levels of the antioxidant vitamins A, C, and E). The lowering of these protective nutrients has been shown to worsen the course of IBD and contributes to a higher rate of carcinogenesis. The pediatric study on children with IBD below solidifies this point.

The serum concentration of zinc, copper and selenium in children with inflammatory bowel disease.

Ojuawo A and Keith L.

Cent Afr J Med Serp-Oct;48(9-10):116-9

The objective of this study was to estimate the levels of trace elements in children with inflammatory bowel disease (IBD). This prospective cross sectional study was done in a children's hospital in London, UK. The study involved 74 children (38 ulcerative colitis and 36 Crohn's disease) suffering from IBD and 40 matched controls. Serum zinc, copper and selenium were assayed at presentation. Assay results showed the following results. The serum selenium was significantly lower in both forms of IBD as compared to the controls. Serum copper was significantly higher in Crohn's sufferers than in the ones with ulcerative colitis and the controls. Children with Crohn's had lower serum zinc than the controls and ulcerative colitis.

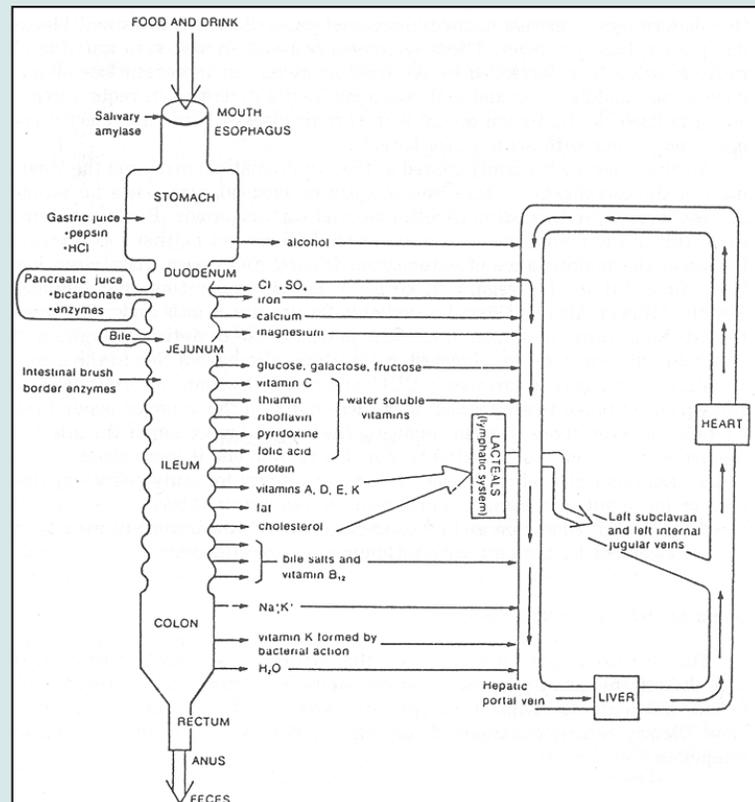
Researchers concluded that children with IBD have abnormalities in the area of trace elements probably a result of inadequate intake, reduced absorption, and increased intestinal loss due to the impairment

of absorption resulting from the inflammatory process. The decrease in free radical scavenging action of the zinc and selenium as a result of the deficiency could contribute to the continued inflammatory process of IBD. This supports the use of trace element supplementation for children with IBD.

Additionally, bone problems, like osteopenia and osteoporosis are a common finding in IBD. The nutritional problems of patients suffering from IBD are many, and the mineral demands for the IBD patient are a critical area. There is a need to take a rational approach to properly manage the nutritional problems of the IBD patient. The approach

must take into account the intestinal function and the site and extent of the disease process. Figure 1, can be used as a guide for assessing the potential for deficiencies. By knowing the site and severity of the symptoms for the IBD patient, along with the knowledge of the main absorption sites for each key nutrient, nutritional deficiencies for an IBD patient can be anticipated. The fact that they suffer from a variety of malabsorption causes, from disease limited absorptive surfaces to drug-induced nutrient wasting makes the selection of a diet rich in mineral content a key. One of the problems with minerals, in supplementation form, for the IBD patient rests in the usual poor tolerability characteristics of a typical mineral

Figure 1. The site and the extent of the disease process and the effect on nutrient absorption. Mayo Clinic, Diet Manual, 6th Edition.



salt. The study abstracted below is a point to consider.

Ferrous fumarate deteriorated plasma antioxidant status in patients with Crohn's disease
Erichsen, K, et al.
Scand J Gastroenterol 2003
May;38(5):543-8

Iron deficiency anemia is a frequent complication of Crohn's disease. Treatment with ferrous iron (Fe⁺²) compounds is often unsuccessful and is associated with gastrointestinal side effects. In theory, oral iron supplementation may even be harmful, since the iron may reinforce intestinal inflammation by catalyzing production of reactive oxygen species. This study was done to investigate the effect of ferrous ion on disease activity and plasma antioxidant status in people with active Crohn's disease. Ten patients with Crohn's and iron deficiency and 10 healthy controls were put on 120mg ferrous fumarate for 7 days. Researchers measured the Crohn's Activity Index, gastrointestinal complaints, and blood samples for antioxidant status, anemia, inflammation, and iron absorption on day 1 and day 8. During the week on ferrous fumarate, the Crohn's Activity Index increased, patients suffered aggravation of diarrhea, abdominal pain and nausea. Plasma-reduced cysteine was lower in the Crohn's sufferers than in the controls. Enzymes that are indicative of antioxidant capacity were also decreased in the Crohn's group.. Researchers concluded that the treatment of iron deficiency with ferrous fumarate deteriorated

the plasma antioxidant status and increased many clinical symptoms in patients with active Crohn's. Plasma reduced cysteine is a sensitive indicator of oxidative stress in the intestine.

It is of note that the ferrous (Fe⁺²) cation is the culprit in the above study. The ionized ferrous cation is what was shown to be the cause for the gastrointestinal (GI) aggravation experienced by the Crohn's patients, which could also lead to the decreased antioxidant status, in the chain of things. Ferrous fumarate, like ferrous sulfate and other iron salt forms all ionize in the gut, prior to absorption. This charged ferrous cation is known to interact with the mucosal lining of the gastrointestinal tract, causing an increase in the irritation of the lining. In the face of IBD, this could be a truly severe situation. The use of an iron form, such as Ferrochel® (ferrous bisglycinate chelate) might be a better choice for people with IBD. Studies have shown that Ferrochel has better GI tolerance characteristic than these salt forms of iron. Ferrous bisglycinate chelate stays intact while in the lumen of the GI tract, thus eliminating the irritation found with forms of iron that ionize in the gut. The needs of the IBD patient are such that one needs not only to supplement minerals, but also to consider the form of the nutrients, as well.

Review article: nutrition and adult inflammatory bowel disease
Goh, H, and O'Morain, CA
Aliment Pharmacol Ther 2003
Feb;17(3):307-20

There have been major advances in the understanding of the etiology, pathogenesis, and genetics of IBD. However the basic triple therapy (5-aminosalicylates, corticosteroids, and azathioprine) and nutrition have kept their central role. This attempts to give an overview of the supportive and therapeutic perspectives of nutrition in the adult with IBD. The objective of supportive nutrition is to correct malnutrition - calorie intake, macro and micronutrients. Of particular clinical relevance is deficiency of calcium, vitamin D, folate, Vitamin B-12, and zinc. There is ever increasing concern over the use of long term corticosteroids for IBD. There is a need to revisit the use of enteral nutrition as a primary treatment for Crohn's treatment failure is most commonly related to failure to comply to the enteral nutrition program. The factors that lead to failed compliance are: feed palatability, inability to stay on solid food for weeks, social inconvenience and transient feed-related adverse reactions. However, successful nutrition programs are key to IBD therapy. There is evidence to support a gradual return to a normal diet through exclusion reintroduction or other dietary regimens following the completion of enteral nutrition to increase remission rates. Emerging therapies are listed, as well, such as glutamine, growth factors, and short chain fatty acids. The future maybe the evolution of enteral nutrition into an important therapeutic strategy with the design of Crohn's disease specific formulation that is individually tailored, acceptable to patients, cost effective, free from adverse side effects and combining

enteral nutrition with novel pre- and probiotics.

It should be noted that this review article does not list iron as a nutrient that needs to be used in the enteral therapy for IBD. As seen in Table 1, iron has a 39% incidence of deficiency in Crohn's disease. The anemia of iron deficiency and the anemia of chronic disease are often encountered in the IBD patient. The anemia of iron deficiency is due to inadequate intake, poor absorption or loss of iron. The anemia of chronic disease is due to decreased erythropoiesis, secondary to increased levels of proinflammatory cytokines, reactive oxygen metabolites and nitric oxide. Despite the need for iron supplementation to improve the anemia in IBD, many shy away from this, since it has been seen that the iron salts used can cause an increased inflammatory activity through the generation of reactive oxygen species. As we know, Ferrochel® (ferrous bisglycinate chelate) does not ionize in the GI tract the way salt forms, like ferrous sulfate and ferrous

fumarate do. Theoretically, Ferrochel should be a form of iron that could be safe and effective for patients with IBD. Future research into this could be very important, and prove to be of critical therapeutic value.

As shown in Table 2, iron, potassium calcium, magnesium, zinc, and selenium have been observed to be deficient in a significant number of patients with IBD, and they are a chronic problem for the Crohn's disease sufferers. The very core of the symptoms of IBD involves GI pain and inflammation, along with bouts of nausea and diarrhea. Iron, potassium calcium, magnesium, and zinc, when given in the form of a salt have all been demonstrated to impart different degrees of GI intolerance. This tolerance problem is usually due to the ionized metal form. It may be that the use of a totally reacted mineral amino acid chelate, whenever possible, as part of the enteral feeding or dietary supplement could solve this problem. In addition, it has been shown that Albion's mineral amino acid chelates can be absorbed over a

longer segment of the GI tract, thus they could present the IBD patient with a chance for better absorption with less tolerance problems.

References

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