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Iron deficiency anemia in IBD

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Abstract

Inflammatory bowel disease (IBD) is a relapsing chronic inflammation of the small and large gut with rising incidence and prevalence worldwide. Iron deficiency anemia is one of the most common extraintestinal manifestations of IBD which correlates with the disease activity and tendency to relapse even after successful management. Anemia affects various aspects of quality of life like physical, cognitive, emotional, ability to work, and healthcare costs. The anemia in IBD can be due to iron deficiency (ID) or anemia of chronic disease (ACD). The relative frequency of ID in IBD is 60 % according to some studies and only 14 % receive treatment. The evaluation of ID is also difficult as ferritin being an inflammatory marker also rises in chronic inflammatory diseases like IBD. The evaluation of anemia in IBD patients involves other investigations like transferrin saturation and exploration of other nutritional deficiencies to curb the marker asthenia with which these patients often present. This highlights the importance of timely investigation and treatment to prevent long-term sequelae. Oral iron therapy can be started in certain circumstances but as inflammation of the gut hampers the iron absorption hence the alternative route to bypass the inflamed gut is usually recommended to avoid the requirement for blood transfusions.

INTRODUCTION

Inflammatory bowel disease (IBD) is a relapsing chronic inflammation of the small and large gut with rising incidence and prevalence worldwide. Iron deficiency anemia is one of the most common extraintestinal manifestations of IBD which correlates with the disease activity and tendency to relapse even after successful management. Anemia affects various aspects of quality of life like physical, cognitive, emotional, ability to work, and healthcare costs. The anemia in IBD can be due to iron deficiency (ID) or anemia of chronic disease (ACD). The relative frequency of ID in IBD is 60 % according to some studies and only 14 % receive treatment. The evaluation of ID is also difficult as ferritin being an inflammatory marker also rises in chronic inflammatory diseases like IBD. The evaluation of anemia in IBD patients involves other investigations like transferrin saturation and exploration of other nutritional deficiencies to curb the marker asthenia with which these patients often present. This highlights the importance of timely investigation and treatment to prevent long-term sequelae. Oral iron therapy can be started in certain circumstances but as inflammation of the gut hampers the iron absorption hence the alternative route to bypass the inflamed gut is usually recommended to avoid the requirement for blood transfusions.

Iron is an essential component of hemoglobin found in erythrocytes and myoglobin muscle and enzymes, it forms around 80% of total body iron. Iron is essential for oxygen-carrying, cellular respiration, and cellular proliferation.[8-9] The daily iron loss is 1-2 mg/day and this ongoing loss gets exaggerated in IBD due to chronic inflammation. Iron deficiency leads to chronic fatigue, adverse effects on the growing brain, and a negative impact on quality of life[10] The ID in IBD is both absolute and functional iron deficiency. Absolute iron deficiency is due to impaired absorption through the inflamed mucosa and chronic blood loss dietary restriction due to disease, malabsorption, and the effect of drugs which is being used for the management of the disease.[11] The

anemia of chronic disease is due to an increase in hepcidin level which is a polypeptide synthesized in the liver and it is an acute phase reactant that regulates the plasma iron concentration at the systemic level. The increase in hepcidin is directly related to the release of proinflammatory cytokines like IL-6, IL-7, and tumor necrosis alpha(TNF-a). The hepcidin induces cellular degradation of the ferroportin transporter. This transporter regulates the iron transfer from intracellular to extracellular medium[12]. Hepcidin reduces the absorption of ferrous from the due to inhibition of divalent metal transport [13]. Transferrin is the main iron carrier protein during inflammation the inflammatory cytokine namely a-antitrypsin blocks the receptor of transferrin in erythroid progenitor cells thus inhibiting erythropoiesis.

CONCLUSION

The management of iron deficiency in IBD requires continuous monitoring both in terms of clinical signs and symptoms and biochemical investigations. Parenteral therapy should be instituted as soon as possible to avoid the deleterious effect of chronic anemia on quality of life. Parenteral therapy is more effective, faster, and better tolerated by patients with anemia.

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1	Katell Peoc'h, Hana Manceau, Francesca Joly, Xavier Treton. "Iron deficiency in chronic inflammatory bowel diseases: an update", Journal of Laboratory and Precision Medicine, 2021 Crossref	23 words — 3%

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