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Bile acid malabsorption in inflammatory bowel disease.

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Abstract

Bile acid malabsorption (BAM) is a common but an underestimated and often neglected sign of inflammatory bowel diseases (IBDs), especially those affecting the distal ileum. Clinically relevant BAM is most often present in patients with Crohn's ileitis and particularly in ileal-resected Crohn's disease patients. However, deterioration of bile acid (BA) metabolism occurs also in patients with IBD without ileal disease or in those in clinical remission, and the role of BAM in these patients is not well appreciated by clinicians. In a majority of cases, BAM in IBD is caused by impaired conjugated BA reabsorption, mediated by apical sodium/BA cotransporting polypeptide, localized at the luminal surface of the ileal enterocytes. As a consequence, numerous pathological sequelae may occur, including the malfunction of lipid digestion with clinical steatorrhea, impaired intestinal motility, and/or significant changes in the intestinal microflora environment. In this review, a detailed description of the pathophysiological mechanisms of BAM-related diarrhea is presented. Although BAM is present in a significant number of patients with Crohn's disease, its laboratory assessment is not routinely included in diagnostic workups, partially because of costs, logistical reasons, or the unavailability of the more sophisticated laboratory equipment needed. Simultaneously, novel findings related to the effects of the BA signaling pathways on immune functions (mediated through TGR5, cell membrane G protein-coupled BA receptor 1, nuclear farnesoid X receptor, nuclear pregnane X receptor, or nuclear vitamin D receptor) are discussed along with intestinal metabolism in its relationship to the pathogenesis of IBD.

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