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Withdrawal of anti-TNFα therapy in inflammatory bowel disease: Is it feasible?

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Anti-tumour necrosis factor alpha (anti-TNFα) therapy is an established treatment in inflammatory bowel disease (IBD) namely Crohn's disease (CD) and ulcerative colitis (UC). However, this treatment is associated with high costs and the possibility of severe adverse events representing a true challenge for patients, clinicians and health care systems. Consequently, a crucial question is raised namely if therapy can be stopped once remission is achieved and if so, how and in whom. Additionally, in a real-life clinical setting, discontinuation may also be considered for other reasons such as the patient's preference, pregnancy, social reasons as moving to countries or continents with less access, or different local policy or reimbursement. In contrast to initiation of anti-TNFα therapy guidelines regarding stopping of this treatment are missing as supporting data is lacking. There is even less information regarding prognostic factors that could predict relapse or sustained remission after anti-TNFα therapy discontinuation. The only provided evidence regarding CD comes from the landmark STORI trial and a few retrospective observational or small prospective studies, while for UC there are even less data available. As a result, the decision of discontinuation is still a challenging aspect in the use of anti-TNFα therapy. Currently this is typically based on an estimated, case-by-case, benefit-risk ratio as the optimal withdrawal strategy is still debated. Another important issue when considering cessation of anti-TNFα therapy is whether the drug can safely be restarted when needed and whether efficacy will be similar. Possible lower response rates after re-initiation of biological therapy, limited alternative treatment options and/or immunogenicity concerns are all factors which constitute to the fear of stopping treatment.

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Changing perceptions of vitamin D requirements: Focus on the gastrointestinal tract

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Because of its' formation through the action of sun exposure, vitamin D is often referred to as "the sunshine vitamin". However, with this endogenous formation of vitamin D being significantly curtailed because of public awareness of skin cancer dangers, attention is turning to dietary sources. As well as it's long recognized role in bone health, increasing evidence has implicated vitamin D deficiency in susceptibility to various gastrointestinal disorders, including colorectal cancer, inflammatory bowel diseases, diverticulitis and irritable bowel syndrome. There is also reason to suggest that vitamin D might provide an adjunct to therapy for such diseases, as well as retarding disease progression. However, an excessive vitamin D intake has been associated with adverse cardiovascular events. However, the optimal vitamin D intake will vary among individuals, because of their genetic differences. Several hundred genes have now been associated with various actions of this vitamin. Randomized controlled trials have been used to justify vitamin D supplementation in different population groups. Nevertheless, these studies may be misleading in the absence of genetic stratification. Genomic technologies have revealed several hundreds of genes associated with vitamin D actions. The nature of these genes emphasizes the potentially negative implications of modulating vitamin D intakes in the absence of complementary human genetic and genomic data, including information on the gut microbiome. We suggest that there is an increasingly strong case for considering the more widespread use of vitamin D fortified foods and/or dietary supplements to optimize gastrointestinal health. However, intake levels should be informed by personalized genetic and genomic information, for the maintenance of disease prevention or disease remission.

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