Madam, the idiopathic inflammatory bowel diseases comprise two types of chronic intestinal disorders: Crohn's disease and ulcerative colitis. Accumulating evidence suggests that inflammatory bowel disease results from an inappropriate inflammatory response to intestinal microbes in a genetically susceptible host. Genetic studies highlight the importance of host-microbe interactions in the pathogenesis of these diseases.¹

Recent study has shown how the host microbe interaction leads to the inflammatory bowel diseases. Researchers at the University of Rochester, New York have found that Vitamin D Receptor located in the normal intestinal mucosa negatively regulates bacterial-stimulated NF-kB activity in Intestine.² NF-kB is a well known transcription factor and through its five proteins it plays a pivotal role in innate and adaptive immune responses. NF-kB requires nuclear localization for exerting its effect and normally this is inhibited by the inhibitor of kB (IκB). IκB is degraded by phosphorylation of IκB by IκB Kinase (IKK). The IKK complex is activated by growth factors, proinflammatory cytokines (such as IL-1 and TNF-α), and hormones through TNF receptor and Toll-like receptor superfamily which ultimately leads to the activation of the NF-kB pathway.³

Vitamin D has been well known for strengthening the immune system. Vitamin D exerts its effects through vitamin D receptor (VDR). VDR is expressed by monocytes and activated B and T lymphocytes and 1, 25(OH)2 vitamin D3 is synthesized by activated macrophages thus acting in a local paracrine manner similar to other cytokines. It activates monocytes and macrophages but suppresses lymphocyte proliferation and immunoglobulin production. It inhibits the action of the proinflammatory transcription factor NF-κB and the production of a variety of different cytokines, including interleukin (IL)-2, IL-12, and interferon-γ.⁴

Keeping in view the functional role of vitamin D and VDR in the inflammation soup and its association with autoimmune diseases further investigations should be done so that this wizard of mucosal cosmos can change the crippling life of patients.

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References