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Letter to the Editor

Some marginal considerations about both preventive and therapeutic measures towards intestinal fibrosis in IBD

Dear Editors,

I have read, with highest interest, the article "Can we prevent, reduce or reverse intestinal fibrosis in IBD?", by G. Latella et al[1]. In such study, the Authors, with very significant and praiseworthy coherence with well explained pathogenetic factors,suggest both preventive and therapeutic measures against the bowell fibrotic complication in inflammatory bowel disease (IBD).

Apart from antinflammatory and immunosuppressive drugs (salicylates, corticosteroids, azatioprine, 6-SH-purine, methotrexate, cyclosporine, etc), whose effectiveness to treat the intestinal fibrosis in IBD remains questionable[1-3] – compared with their antifibrotic effects in several fibrotic disorders such as systemic fibrosis, retroperitoneal fibrosis (RPF), idiopathic lung fibrosis[4-6] – attention has been turned, some years ago, to various bioagents[1]. Anti-TNF-alpha antibodies (infliximab, adalimumab, etc), besides inducing at times, intestinal obstructive complications, that have been mentioned by the Authors[1], unfortunately may cause, in addition, other serious adverse events such as infliximab-related lupus-like syndrome development, reactivation of latent tubercolosis, onset of malignancies, particularly lymphoma and melanoma[7], what dissuading from their resort.

Looking to novel biotherapies, as well as mediators and molecular pathways which might be developed as antifibrogenic modalities that have been highlighted by the Authors[1] – from different agents able to block TGF-beta signaling or induce dual inhibition of c-Abl/PDGF receptors or even interfere with renin-angiotensin system, to statins because of their antinflammatory/antifibrotic properties (besides lipid lowering)that's why their use has been also suggested to prevent/treat RPF[8] and, furthermore, to miRNAs downregulating Smad-3 activity – other intriguing measures could represent a challenging prospect against the intestinal fibrotic complication of IBD.

There is, indeed, a growing interest in the potential use of phosphodiesterase-4 blockers as antinflammatory cAMP elevating agents, that are currently used to treat various autoimmune disorders such as particularly the rheumatoid arthritis[9] and have also been proposed preventing/mitigating the RPF[5], and, moreover, of Janus kinases-signal transducers/activators of transcription (Jak-STATs) inhibitors which have received considerable attention to treat some chronic autoimmune diseases[10-12] and have been perspectively suggested for the therapeutic management of RPF[8].

Conflict of Interest

The Authors declare that they have no conflict of interests.

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