

REVIEW ARTICLE OPEN ACCESS

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TREATMENT MODALITIES OF INFLAMMATORY BOWEL DISEASE (IBD)

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A chronic disease of the intestine in genetically susceptible individuals, Inflammatory bowel disease (IBD) is caused by contributing elements of both innate and adaptive immune system. IBD is a group of inflammatory conditions of the bowel that is chronic and uncontrolled in nature. The triggers that start and sustain this constant irritation are the subject of much hypothesis and exploration, in spite of the fact that the focal function of the microbiota (intestinal) is perceived, and is even an objective for treatment in certain conditions. The modern day IBD treatment is targeted at concealing safe reaction towards so far unknown antigens, and customary treatment that includes 5-aminosalicylic acid (5-ASA), thiopurines, corticosteroids and methotrexate. Although a complete cure for IBD is not yet available but ongoing revelations in the field of neurosciences and immunology have uncovered that signals in the fringe sensory system direct irritation, including levels of TNF-α show promising results. Clinical examinations utilizing vagus nerve embedded triggers for IBD treatment show empowering results. As needs be, the reflex control (neural) of aggravation is developing as a possible remedial objective in treating IBD. Here, we audit available and current treatment choices. These include the use 5-ASA, Glucocorticosteroids, Methotrexate and Thiopurines.

Keywords: Inflammatory Bowel Disease; Ulcerative Colitis; Crohn's Disease; Methotrexate

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INTRODUCTION

IBD has two divisions, namely: Crohn's infection (CD) and ulcerative colitis (UC). CD is the profound mucosal irritation or inflammation that can influence the whole length of gastrointestinal (GI) tract. UC on the contrary constitutes persistent shallow aggravation restricted to the digestive organ. Normal side effects are free stools with blood and bodily fluid, stomach torment, and weakness. CD has strong association with the development of perianal fistulas and intestinal injuries in the colonic region. Severe chronic infection in IBD are refractory to traditional therapy. For instance, the use 5-aminosalicylate and corticosteroids.

Antibiotics use regularly lowers or lightens the infection. This highlights the vital function of luminal microbiota in turning off the constant inflammation or irritation. In this article, we audit the available and current treatment options and modalities of.¹

- 1. Amino Salicylate
- 2. Glucocorticosteroids
- 3. Methotrexate
- 4. Thiopurines

REVIEW

Given that advancements to know the pathogenesis of IBD have not yet recognized a curative helpful objective. IBD treatments are aimed at decreasing incendiary movement by lessening multiplication and pro-inflammatory action of the safe cells. The cutting-edge restorative stockpile includes 5-ASA, Glucocorticosteroids, methotrexate, methotrexate and monoclonal biologic operators. These various medications balance different cell capacities, and by consolidating distinctive remedial modalities, an adequate illness control might be accomplished while keeping up the danger of unfavourable occasions at a satisfactory level.

Amino Salicylate

5-Aminosalicylate medications (5-ASA) were presented very nearly seventy years prior, as sulfasalazine. The first medication comprised of a blend of an anti-toxin, sulphapyridine and 5-ASA. 35 years after the fact, that the anti-inflammatory impact

of 5-ASA prompted the advancement of elective plans and improved side-effect profile.

High doses of 5-ASA have been associated with an increased or better efficacy in cases of acute UC. However, the exact dosing schedule for each ASA formulation is still unknown and needs further validation and research. The use of Topical ASA has also been warranted as is almost more effective than the use of steroids, especially in cases of distal UC. Maintenance doses of ASA are found useful in decreasing risk of clinical recurrence.^{2,3}

Glucocorticosteroids

Glucocorticosteroids (GCS) aims its antiinflammatory function through the intracellular
glucocorticoid receptor. This interaction leads to
movement of the cell core within 20 minutes of the
ligand/receptor interaction. This
glucocorticoid/receptor complex attached itself to the
positive glucocorticoid-response elements within the
DNA and leads to the production of anti-inflammatory
proteins. This process is called transactivation.

The Glucocorticosteroids/receptor complex also attaches to the negative glucocorticoid-responsive elements within the DNA, hindering quality articulation of cytokines, for example, IL-1 and IL-2. Budesonide is found effective in the treatment of CD, especially for the induction of remission phase. 4.5

Methotrexate

Methotrexate is used in the treatment of vide range of medical disorders including: IBD, Rheumatoid disorders and Skin. The primary component of methotrexate-an anti-cancer drug, Initially, has an element of folic acid digestion. This prompts a decreased change of homocysteine to methionine, along these lines stifling DNA-replication. The danger of antagonistic impacts, for example, leukopenia, paleness and stomatitis is moderated by supplemental folic acid. Methotrexate has clinically established drug efficacy in steroid dependant CD. Recently, Methotrexate has been used in paediatric population of CD as a monotherapy.^{6,7}

Thiopurines:

Azathioprine, thioguanine and 6-mercaptopurine are the major Thiopurines we 'll take into account while discussing the different treatment modalities in IBD.

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Thioguanine is under trial for the treatment of IBD but the rest two are approved drugs for treating IBD. Thiopurine methyltransferase (TPMT) methylates thiopurines into methyl mercaptopurine ribonucleotides that apply a quick hindrance of the purine combination in leucocytes. Thiopurines are immunosuppressive agents. They alter vital process that lead to inflammation via T lymphocyte.8,9

CONCLUSION

Although a complete cure for IBD is not yet available but ongoing revelations in immunology and neuroscience have uncovered that signals in the fringe sensory system direct irritation, including levels of TNF- α show promising results. Clinical examinations utilizing embedded vagus nerve triggers for treatment of IBD show empowering results. As needs be, the neural reflex control of aggravation is developing as a possible remedial objective in treatment of IBD.

REFERENCES

- Burisch J, Jess T, Martinato M, Lakatos PL, EpiCom E. The burden of inflammatory bowel disease in Europe. J Crohns Colitis. 2013;7:322-337.
- Rousseaux C, Lefebvre B, Dubuquoy L, et al. Intestinal antiinflammatory effect of 5-aminosalicylic acid is dependent on peroxisome proliferator-activated receptor-gamma. J Exp Med. 2005;201:1205-1215.
- Gisbert JP, Gomollon F, Mate J, et al. Role of 5-aminosalicylic acid (5-ASA) in treatment of inflammatory bowel disease: a systematic review. 2002
- Stahn C, Lowenberg M, Hommes DW, Buttgereit F. Molecular mechanisms of glucocorticoid action and selective glucocorticoid receptor agonists. Mol Cell Endocrinol. 2007;275:71-78.
- Buttgereit F, Straub RH, Wehling M, Burmester GR. Glucocorticoids in the treatment of rheumatic diseases: an update on the mechanisms of action. Arthritis Rheum. 2004;50:3408-3417.
- Cronstein B. How does methotrexate suppress inflammation? Clin Exp Rheumatol. 2010;28:S21-S23.
- Herfarth HH, Kappelman MD, Long MD, Isaacs KL. Use of Methotrexate in the Treatment of Inflammatory Bowel Diseases. Inflamm Bowel Dis. 2016;22(1):224-233.
- Derijks LJ, Gilissen LP, Hooymans PM, Hommes DW. Review article: thiopurines in inflammatory bowel disease. Aliment Pharmacol Ther. 2006;24:715-729.
- Neurath M. Thiopurines in IBD: What Is Their Mechanism of Action?. Gastroenterol Hepatol (N Y). 2010;6(7):435-4

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