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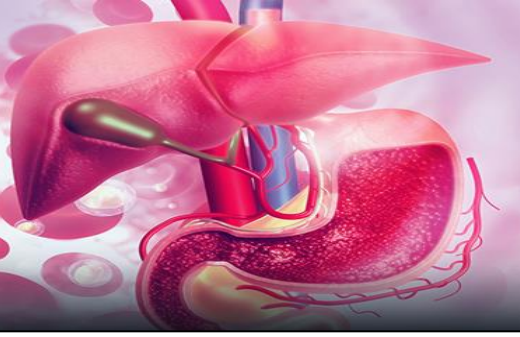

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Dear Doctor,

Greetings from Micro Labs limited. We are happy to bring you "ENTERON" which contains latest and interesting news in the field of Gastroenterology.

Inside This Issue:

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Looking forward to your valuable feedback

Best Regards,

Medical Team, Micro Labs Ltd

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A study on outcome of Infliximab vs. cyclosporine A for steroid-refractory acute severe ulcerative colitis

Background

Introduction of biologic agents has remarkably improved outcomes among patients with active ulcerative colitis (UC). But treatment of patients with acute severe ulcerative colitis (ASUC) is still challenging. Among patients with ASUC, induction therapy with intravenous (IV) steroids can be used as the first-line therapy to achieve clinical remission. However, 30–40% of patients with ASUC have shown refractoriness to induction therapy with IV steroids. In such patients, rescue therapy with cyclosporin A (CsA, a calcineurin inhibitor) or with infliximab (IFX, an anti-tumor necrosis factor [TNF] agent) has been recommended. In a randomized controlled trial, the overall rates of treatment failure, colectomy, and adverse events were similar between the two treatment groups. In a follow-up report, the investigators reported that the long-term colectomy-free survival was also similar between the two groups. An Australian prospective study reported that rates of colectomy and adverse events were significantly lower in the IFX group than in the CsA group during 12 months of follow-up. Most studies evaluating treatment outcomes of CsA and IFX have been performed in Western populations. Real-life data comparing the outcomes of CsA and IFX rescue therapy among Asian patients with SR-ASUC are scarce.

Objective:

Aim: To evaluate the outcomes of both cyclosporine A and infliximab among Asian patients with steroid refractory acute severe ulcerative colitis (SR-ASUC).

Methods:

A retrospective, cohort and single-cantered with 121 patients who received CsA; n = 23 and IFX ; n=96 for the treatment of SR-ASUC. Induction and maintenance regimen with CsA and IFX was done. Cyclosporin A was administrated intravenously at 2 mg/kg and adjusted according to the blood concentration and IFX was administrated intravenously at 5mg/kg at 0, 2 and 6 weeks. Once the patients achieved clinical response, intravenous CsA was stopped and oral CsA was started. The primary end point was to evaluate the efficacy of CsA and IFX for 3 months in terms of treatment failure among the patients. The treatment was define by 1)

colectomy, 2) switch to other anti-TNF drugs, 3) acute flare up events and 4) drug interruption due to adverse events. The secondary end point was to assess the efficacy of CsA and TFX for 3 months in terms of colectomy rate among the patients.

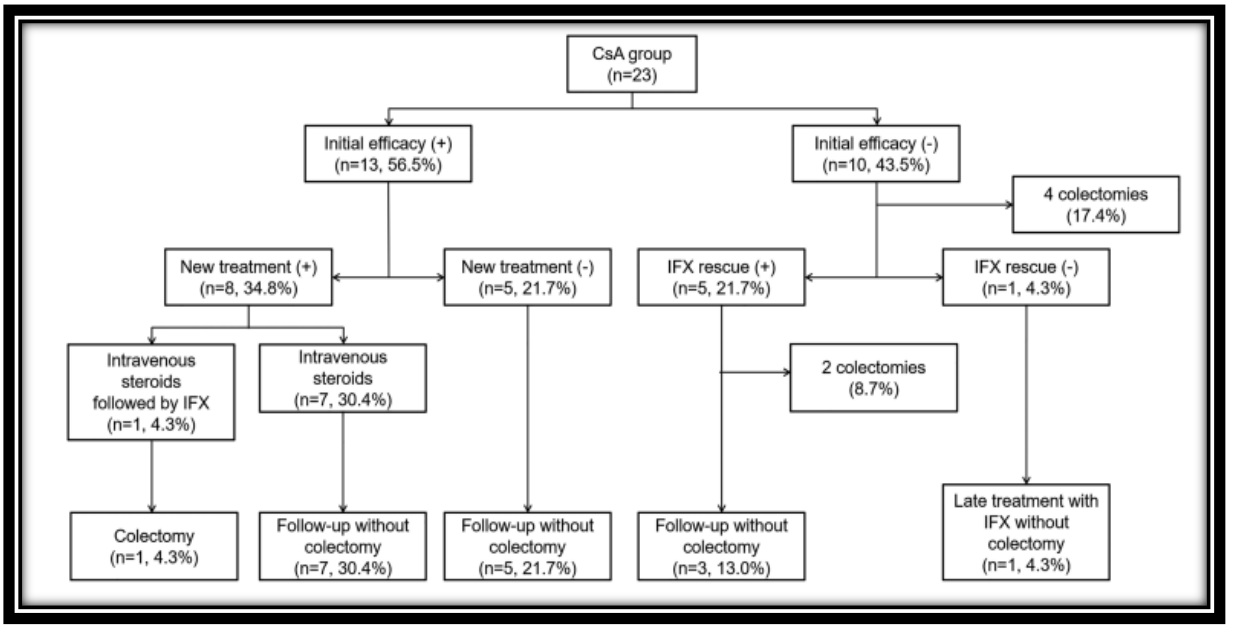

Figure 1: Outcomes of cyclosporine A rescue therapy among patients with steroid-refractory acute severe ulcerative colitis. CsA, cyclosporine A; IFX, infliximab.

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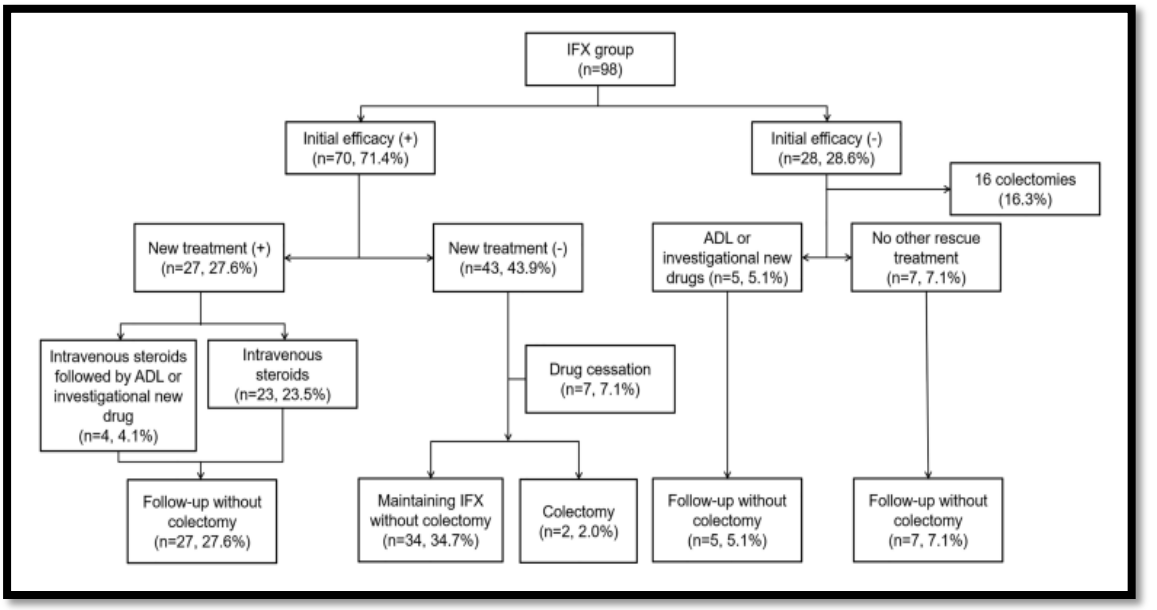

Figure 2: Outcomes of infliximab rescue therapy among patients with steroid-refractory acute severe ulcerative colitis. ADL, adalimumab; IFX, infliximab.

Results:

The two groups had similar baseline characteristics (age at diagnosis, sex, disease duration, disease extent at rescue therapy, and Mayo score at treatment commencement). 84 patients (69.4%) reported treatment failure during follow-up (median 45 months; interquartile range 29.3–61.8), and 25 patients (20.7%) required colectomy. At 3 months, there were no significant differences between the CsA and IFX groups in the cumulative rates of treatment failure (39.1 percent vs 34.7 percent, P = 0.714) or colectomy (26.1 percent vs 13.3 percent, P = 0.198). Treatment failure was linked to previous use of azathioprine (odds ratio [OR] = 2.309, 95 percent confidence interval [CI] = 1.076–4.951, P = 0.032). Colectomy was substantially linked with a Mayo score of 10 or above at the time of rescue therapy (OR = 8.444, 95 percent CI = 2.592–27.506, P 0.001).

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Duration	Total (*N* = 121), *n* (%)	CsA group (*N* = 23), *n* (%)	IFX group (*N* = 98), *n* (%)	*P*†
15 days	10 (8.3)	6 (26.1)	4 (4.1)	0.001
1 month	18 (14.9)	8 (34.8)	10 (10.2)	0.007
2 months	27 (22.3)	9 (39.1)	18 (18.4)	0.049
3 months	43 (35.5)	9 (39.1)	34 (34.7)	0.714
6 months	50 (41.3)	11 (47.8)	39 (39.8)	—
12 months	61 (50.4)	13 (56.5)	48 (49.0)	—
24 months	72 (59.5)	17 (73.9)	55 (56.1)	—
36 months	76 (62.8)	18 (78.3)	58 (59.2)	—
48 months	79 (65.3)	19 (82.6)	60 (61.2)	—
60 months	82 (67.8)	19 (82.6)	63 (64.3)	—

Table 1; Cumulative treatment failure rates at 15 days, 1, 2, 3, 6, 12, 24, 36, 48, and 60 months in the CsA group and in the IFX group

Duration	Total (*N* = 121), *n* (%)	CsA group (*N* = 23), *n* (%)	IFX group (*N* = 98), *n* (%)	*P*†
15 days	5 (4.1)	3 (13.0)	2 (2.0)	0.047
1 month	12 (9.9)	5 (21.7)	7 (7.1)	0.050
2 months	17 (14.0)	5 (21.7)	12 (12.2)	0.314
3 months	19 (15.7)	6 (26.1)	13 (13.3)	0.198
6 months	19 (15.7)	6 (26.1)	13 (13.3)	—
12 months	20 (16.5)	6 (26.1)	14 (14.3)	—
24 months	21 (17.4)	6 (26.1)	15 (15.3)	—
36 months	23 (19.0)	6 (26.1)	17 (17.3)	—
48 months	23 (19.0)	6 (26.1)	17 (17.3)	—
60 months	24 (19.8)	6 (26.1)	18 (18.4)	—

Table 2: Cumulative colectomy rates at 15 days, 1, 2, 3, 6, 12, 24, 36, 48, and 60 months in the CsA group and the IFX group

Conclusion:

Treatment failure and colectomy rates were not significantly different between the CsA and IFX groups at 3 months in patients with SR-ASUC. Use of azathioprine in the past was found to be a strong predictor of treatment

failure. A higher incidence of short-term colectomy was linked to more severe illness when rescue therapy was initiated.

Reference:

Dai C, Jiang M, Huang YH. Comparison of outcomes of cyclosporine A and infliximab for steroid-refractory acute severe ulcerative colitis. J Gastroenterol Hepatol. 2021 Jul;36(7):2024-2025.

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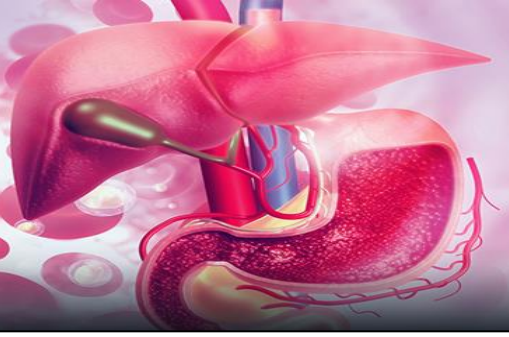
AGA Clinical Practice Update on the Diagnosis and Management of Atrophic Gastritis: Expert Review

Best practice advice statements

- Atrophic gastritis is defined as the loss of gastric glands, with or without metaplasia, in the setting of chronic inflammation mainly due to Helicobacter pylori infection or autoimmunity. Regardless of the etiology, the diagnosis of atrophic gastritis should be confirmed by histopathology.
- Providers should be aware that the presence of intestinal metaplasia on gastric histology almost invariably implies the diagnosis of atrophic gastritis. There should be a coordinated effort between gastroenterologists and pathologists to improve the consistency of documenting the extent and severity of atrophic gastritis, particularly if marked atrophy is present.
- Providers should recognize typical endoscopic features of atrophic gastritis, which include pale appearance of gastric mucosa, increased visibility of vasculature due to thinning of the gastric mucosa, and loss of gastric folds, and, if with concomitant intestinal metaplasia, light blue crests and white opaque fields. Because these mucosal changes are often subtle, techniques to optimize evaluation of the gastric mucosa should be performed.
- When endoscopic features of atrophic gastritis are present, providers should assess the extent endoscopically. Providers should obtain biopsies from the suspected atrophic/metaplastic areas for histopathological confirmation and risk stratification; at a minimum, biopsies from the body and antrum/incisura should be obtained and placed in separately labeled jars. Targeted biopsies should additionally be obtained from any other mucosal abnormalities.
- In patients with histology compatible with autoimmune gastritis, providers should consider checking antiparietal cell antibodies and anti-intrinsic factor antibodies to assist with the diagnosis. Providers should also evaluate for anemia due to vitamin B-12 and iron deficiencies.
- All individuals with atrophic gastritis should be assessed for H pylori infection. If positive, treatment of H pylori should be administered and successful eradication should be confirmed using non - serological testing modalities.



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- The optimal endoscopic surveillance interval for patients with atrophic gastritis is not well-defined and should be decided based on individual risk assessment and shared decision making. A surveillance endoscopy every 3 years should be considered in individuals with advanced atrophic gastritis, defined based on anatomic extent and histologic grade.
- The optimal surveillance interval for individuals with autoimmune gastritis is unclear. Interval endoscopic surveillance should be considered based on individualized assessment and shared decision making.
- Providers should recognize pernicious anemia as a late-stage manifestation of autoimmune gastritis that is characterized by vitamin B-12 deficiency and macrocytic anemia. Patients with a new diagnosis of pernicious anemia who have not had a recent endoscopy should undergo endoscopy with topographical biopsies to confirm corpus-predominant atrophic gastritis for risk stratification and to rule out prevalent gastric neoplasia, including neuroendocrine tumors.
- Individuals with autoimmune gastritis should be screened for type 1 gastric neuroendocrine tumors with upper endoscopy. Small neuroendocrine tumors should be removed endoscopically, followed by surveillance endoscopy every 1–2 years, depending on the burden of neuroendocrine tumors.
- Providers should evaluate for iron and vitamin B-12 deficiencies in patients with atrophic gastritis irrespective of etiology, especially if corpuspredominant. Likewise, in patients with unexplained iron or vitamin B-12 deficiency, atrophic gastritis should be considered in the differential diagnosis and appropriate diagnostic evaluation pursued.
- In patients with autoimmune gastritis, providers should recognize that concomitant autoimmune disorders, particularly autoimmune thyroid disease, are common. Screening for autoimmune thyroid disease should be performed

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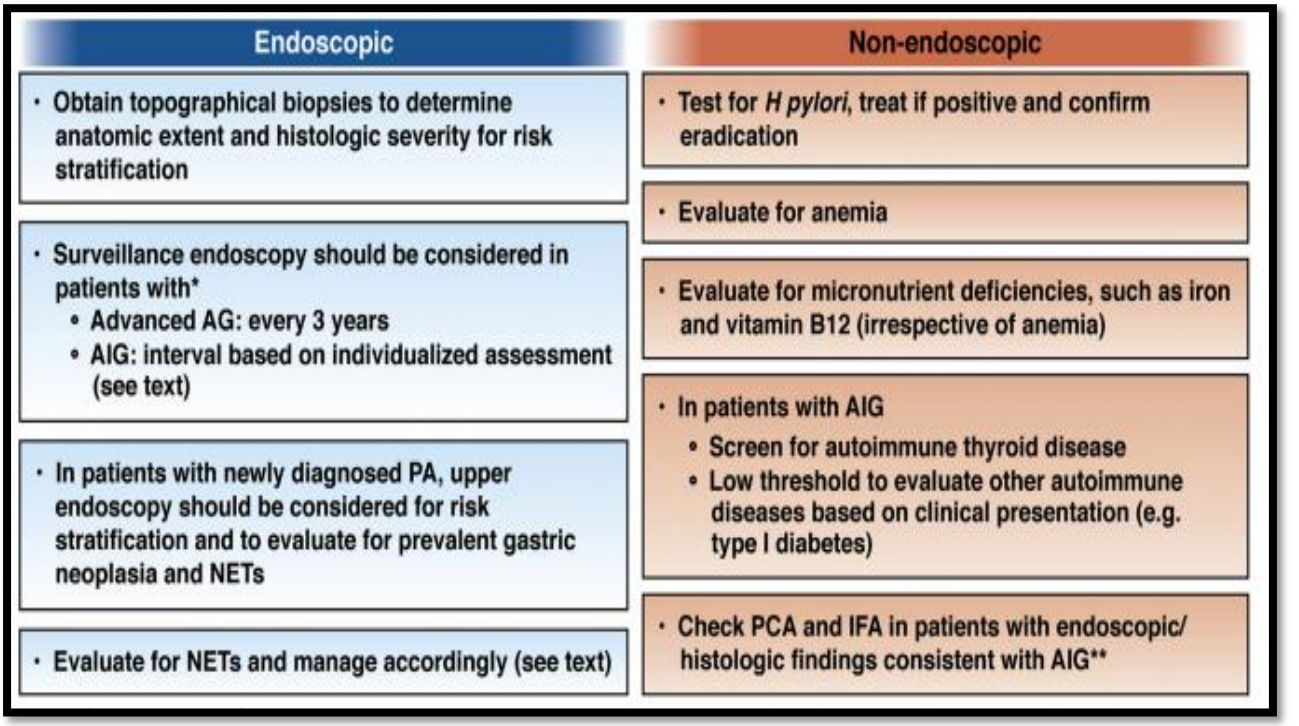
Algorithm for clinical management of AG



Conclusion:

Endoscopic surveillance should be considered in patients with severe AG for the purpose of early gastric cancer detection and require additional management considerations, including attention to micronutrient deficiencies, particularly iron and vitamin B-12 deficiencies. To improve the diagnosis and characterization of AG, co-ordinated efforts between gastroenterologists and pathologists is required.

Reference:

Shah SC, Piazuelo MB, Kuipers EJ, Li D. AGA Clinical Practice Update on the Diagnosis and Management of Atrophic Gastritis: Expert Review. Gastroenterology. 2021 Oct;161(4):1325-1332.e7.

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A case report on intestinal ulcers in a patient with myelodysplastic syndrome

Background:

Myelodysplastic syndrome (MDS) is a group of heterogeneous acquired clonal malignant diseases. Isolated trisomy 8 is one of the most frequent cytogenetic abnormalities in MDS which presents in 10–15% of MDS/ myeloproliferative neoplasm (MPN) cases. Has a short median survival when compared to normal karyotype. But these characteristics of trisomy 8 positivity MDS are poorly reported. Behçet's disease (BD) an autoimmune vasculitis which is characterized by recurrent involvement of the mouth, eyes, genitals and skin. A number of cases have developed gastrointestinal ulcers which called gastrointestinal (GI) BD. Nowadays, some evidence shown an association between MDS, trisomy 8 and Behçet's-like disease.

Aim: To provide a new initiative for the diagnosis and treatment for trisomy 8 positive MDS with BD.

Case presentation:

A 63-year-old man with fever, stomach pain, and hematochezia was presented in this study. The terminal ileum and cecum had mural hyperenhancement and gut wall thickening, according to imaging investigations. Multiple round ulcers in the terminal ileum, the ileocecal valve, and numerous yellow dots pseudomembranous attachments throughout the colon were discovered during a colonoscopy. Multiple irregular ulcers in the lower ileum were discovered during capsule endoscopy. C-reactive protein in the blood and calprotectin in the faeces were both excessively high. The toxin A and B from Clostridium difcile were positive.

The patient's intestinal ulcers, on the other hand, did not heal after a two-week course of vancomycin. The patient was diagnosed with MDS-RAEB2 and has a karyotype of 47 XX,+8. 2 months before to admission, a thorough medical history investigation revealed epifolliculitis and recurrent mouth ulcers. A trisomy 8 positive MDS with BD diagnosis was made. The patient was then given glucocorticoids and the fifth round of azacytidine. Two months after treatment, a follow-up endoscopy revealed a much better intestinal ulcer.

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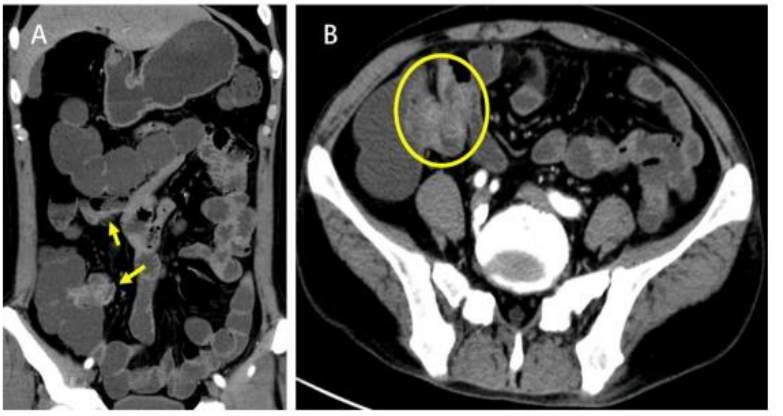

Figure 3: The coronal images (A) and cross-sectional images (B) of computed tomography enterography showed bowel wall thickening and mural hyperenhancement of terminal ileum and cecum (the arrows)

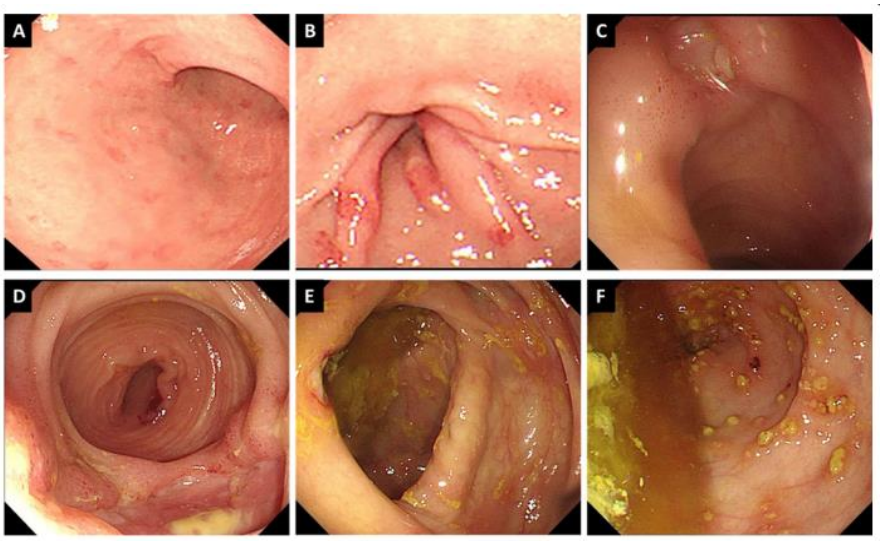

Figure 4: Gastroscopy (A, B) revealed multiple erosions and telangiectasias in the antrum of stomach. Colonoscopy showed multiple round ulcers in terminal ileum and ileocecal valve and multiple yellow (C–E) dotted pseudomembranous attachments throughout colon (F)

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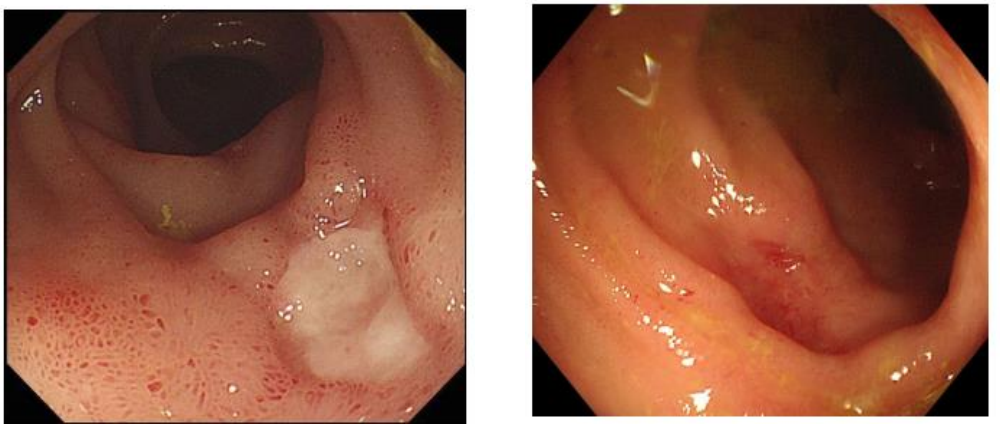

Figure 5: A) Colonoscopy showed that the intestinal ulcers have no tendency to shrink following successful treatment of clostridium difcile infection and B) Endoscopy showed significantly improved intestinal ulcer 2 months after treatment

Conclusion:

The study emphasises the procedure of considering the diagnosis. This could lead to a novel approach to diagnosing intestinal ulcers.

Reference:

Zhang X, et al. Intestinal ulcers in a patient with myelodysplastic syndrome: A case report. BMC Gastroenterol. 2021 Oct 11;21(1):372.

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