Symposium | All

Sat. Nov 21, 2026 10:25 AM - 11:40 AM JST | Sat. Nov 21, 2026 1:25 AM - 2:40 AM UTC | Room 5 (1F 102)

Symposium 7 Gut Microbiota and Colorectal Diseases: A Future Unlocked by the Microbiome

Moderator: Hiroto Miwa (Kawanishi City Medical Center, Japan), Tomohisa Takagi (Molecular Gastroenterology and Hepatology, Graduate School of Medical Science, Kyoto Prefectural University of Medicine, Japan)

[SY7-7] Efficacy of Colchicine in Mild-to-Moderate Ulcerative Colitis and Identification of Treatment Response–Associated Molecular Profiles

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Background

Inflammasome activation is a central driver of innate immune signaling and can amplify intestinal inflammation by releasing IL-1β. Our previous studies suggest that aberrant inflammasome activation contributes to the pathophysiology of ulcerative colitis (UC) in a subset of patients. This study aimed to evaluate the efficacy of colchicine, an inflammasome inhibitor, in patients with mild-to-moderate UC and to identify molecular profiles associated with treatment response using omics analyses.

Methods

Fifteen patients with mild-to-moderate UC (treatment-naïve or 5-ASA–refractory/intolerant) received colchicine 0.5 mg/day, escalated to 1.0 mg at day 14 if non-responding; non-responders at day 28 discontinued, while responders continued through day 84. The primary endpoint was clinical response at day 14. Secondary endpoints included clinical and endoscopic remission at day 84 and safety (CTCAE v5.0). Omics analyses comprised genomic (blood), transcriptomic (colonic mucosa), and gut microbiome analyses (fecal).

Results

Clinical response at day 14 was achieved in 53.3% (8/15), clinical remission at day 84 in 33.3% (5/15), and endoscopic remission in 13.3% (2/15). Adverse events were mild: one patient experienced grade 1 diarrhea, and one grade 1 liver dysfunction. Responders (R) carried MEFV variants in 87.5% (7/8), significantly higher than non-responders (NR) (28.6%, 2/7; p=0.041). Transcriptomic analysis showed enrichment of adaptive immune–related pathways in NR, with significantly elevated KLRG1 expression ($p<0.001$). Gut microbiome analysis revealed no difference in α-diversity between R and NR, whereas β-diversity differed significantly (PERMANOVA, p=0.049). In R, potentially inflammasome-activating bacteria such as Enterobacter were significantly enriched, whereas NR showed increases in anti-inflammatory taxa (e.g., Bifidobacterium longum), suggesting a compensatory expansion.

Conclusion

Colchicine may benefit a subset of patients with UC who show linked inflammasome signatures across genetics, microbiota, and mucosa.

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