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Immunosuppression aggravates the gut microbial dysbiosis in patients with inflammatory bowel disease

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Objective: The microbial characterization among patients undergoing aminosalicylic acid (ASA) antiinflammation and immunosuppression was unclear. The aim of our study was to clarify the impact of two treatment strategies on gut microbial composition in IBD patients.

Methods: 73 IBD patients were divided into three groups, untreated, ASA antiinflammation and immunosuppression. 16S and ITS sequencing were performed to investigate the bacterial and fungal composition. Linear regression analysis was used to explore correlation between microbial abundance and IBD severity, and Spearman's test was used to investigate bacteria-fungi and microbiota-biomarkers correlation.

Results: For bacterial microbiota, immunosuppression increased Escherichia and decrease *Dorea* and Ruminococcus in inflamed mucosa, while ASA antiinflammation increased *Dorea* and *Bifidobacterium* and decreased Streptococcus in inflamed mucosa. The decrease of Firmicutes (*Faecalibacterium and Roseburia*) and Actinobacteria (*Bifidobacterium*), and increase of Proteobacteria (Escherichia) in inflamed mucosa were correlated to IBD severity. Although fungal abundance wasn't correlated to IBD severity, several fungi underwent abundant significant increase in inflamed mucosa, and immunosuppression increased scytalidium and verticillium in inflamed mucosa. IBD-associated bacteria were correlated to fungi that significantly increased in inflamed mucosa, and bacteria-fungi correlation showed a treatment-specific pattern. Additionally, a potent bacteria-biomarkers correlation and a weak fungi-biomarkers correlation were observed, and in IBD-associated biomarkers, IL-17A was extensively correlated with several bacteria.

Conclusion: Rather than ASA antiinflammation, immunosuppression aggravates gut microbial dysbiosis in IBD patients. The bacteria-fungi and microbiota-biomarkers correlation display a treatment-specific pattern. Regulation of IL-17A expression by bacterial microbiota tends to be a key pathway to induce inflammation in IBD.

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