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Summary

Understanding the mechanisms of extraintestinal manifestations of IBD

Daniel Kotlarz

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Extraintestinal manifestations in IBD, affecting up to 50% of patients, arise through four interconnected mechanisms including shared genetic susceptibility, epithelial barrier dysfunction, systemic immune activation, and aberrant lymphocyte trafficking, with important therapeutic implications for pathway-specific targeting.

KEY POINTS

- The speaker stated that up to 50% of IBD patients develop extraintestinal manifestations during disease course, with 26% manifesting before IBD diagnosis and 38% presenting with two or more extraintestinal manifestations.
- Four interconnected mechanisms underlie extraintestinal manifestations: shared genetic susceptibility involving pathways such as NOD2, IL-23 receptor, TNF, and JAK-STAT; epithelial barrier dysfunction leading to microbial antigen translocation and molecular mimicry; systemic immune activation; and aberrant lymphocyte homing to extraintestinal sites.
- In monogenic IBD, the speaker reported that 76% of patients develop at least one extraintestinal manifestation, with specific examples including IL-10 receptor deficiency causing nearly 100% intestinal disease with folliculitis and arthritis, and CTLA-4 haploinsufficiency causing multi-organ autoimmune phenotypes.
- TNF and JAK-STAT pathways operate across multiple organs and can target both intestinal and extraintestinal inflammation, while IL-23 inhibition benefits gut and skin but is less effective for peripheral joints, and IL-17 blockade is effective for axial spondyloarthritis and psoriasis but detrimental for Crohn's disease.
- The speaker envisions future deep phenotyping integrating multi-omics approaches to identify molecular endotypes, improve patient stratification, and enable mechanism-based precision therapies for extraintestinal manifestations.

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