

Fistulising Crohn's disease: Underlying mechanism

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Presentation

IBD

Primary Care

Surgery

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This presentation explored cellular and genetic mechanisms underlying fistulating Crohn's disease, emphasizing transmural inflammation, myeloid cell involvement, and spatial transcriptomics findings that implicate epithelial-mesenchymal transition, matrix degradation, and specific genetic loci including PTGER4.

KEY POINTS

- The speaker stated that fistula pathogenesis starts with mucosal failure to heal and requires transmural inflammation, with spatial transcriptomics showing profusion of myeloid cells in ulcers and marked thickening of the muscularis mucosa in chronic disease.
- Anti-TNF therapy has the most data for treating fistulae, with the speaker noting that early treatment with effective biologics is valuable; phase 3 results for anti-p19 agents are pending, and post-hoc analysis reported upadacitinib effective in acute and maintenance phases.
- African Americans have approximately two-fold higher rate of perianal fistula (40% versus 20% as stated by the speaker) compared to other populations, reported consistently in published and unpublished data.
- The speaker's lab reported that fine mapping of the chromosome 5p13 region implicates PTGER4 with regulated T cell expression, and gene regulatory analysis showed JUN occupancy and FOS expression implicating the AP-1 transcription factor family in disease chronicity.
- Comparing African versus European ancestry individuals, the speaker's lab found increased chitinase 3-like 1 secretion in cultured CD14 monocytes from African Crohn's patients, and reported that CD14-positive cells cultured long-term can become collagen-high fibroblast-like cells.
- The speaker emphasized that ileal and perianal fistulae are fundamentally different, with NOD2 mutations linked to ileal stricturing disease, and stated that curettage is beneficial in stem cell trials for perianal fistula.

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