Biomarkers and gene signatures identified by transcriptomics

Ahmed Hegazy

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The speaker reviewed transcriptomic studies identifying gene expression modules associated with therapy resistance in IBD, focusing on IL-1, IL-22, and oncostatin M pathways that appear to drive neutrophil recruitment and treatment failure in a subset of patients.

KEY POINTS

- The speaker reported that approximately 20% of highly inflamed ulcerative colitis patients in the IBIDOM cohort express an oncostatin M module associated with non-response to infliximab and adalimumab, with a trend for vedolizumab.
- Analysis of around 100 UC patients identified 17 gene expression modules; one module enriched in oncostatin M, IL-1, neutrophils, and stromal cells correlated strongly with histological inflammation and predicted anti-TNF failure.
- The speaker described a proposed mechanism in which IL-1 and oncostatin M act on stromal and epithelial cells to induce chemokine expression, recruiting neutrophils and driving therapy-refractory inflammation.
- Studies reviewed showed IL-22 signature predicted poor response to ustekinumab, with higher IL-22 enrichment correlating with neutrophil infiltration; IL-7 pathway signature distinguished anti-TNF non-responders.
- Comparison of IBIDOM with Oxford and Mount Sinai cohorts revealed overlapping but not identical modules, with differences potentially attributable to treatment history, geography, microbiota, or technical factors according to the speaker.
- The speaker stated that a clinical trial of anti-oncostatin M receptor therapy was stopped due to lack of efficacy, and noted these findings are not yet ready for clinical application.

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