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Summary

Barrier dysfunction in eosinophilic oesophagitis: What's the evidence and current treatment to improve it

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This tandem talk presented evidence that eosinophilic oesophagitis is fundamentally a disease of epithelial barrier disruption and reviewed how current therapies affect barrier restoration as measured by histological surrogates and mucosal impedance.

KEY POINTS

- The speakers stated that genetic defects, type 2 cytokines (IL-13, IL-33, TSLP), TGF-beta, hypoxia, acid, and detergents can all disrupt the oesophageal epithelial barrier and drive EOE pathogenesis through a feed-forward inflammatory loop.
- According to the speakers, dupilumab demonstrated the first prospective randomised placebo-controlled evidence of restoring mucosal impedance in EOE, with consistent increases not seen in placebo, and also improved histological surrogates of barrier integrity including dilated intercellular spaces and basal zone hyperplasia.
- The speakers reported that elimination diet and topical steroids improved impedance in trials, though impedance did not fully reach control levels, while IL-5 blockade improved eosinophil counts but did not favourably change barrier-related histological features or symptoms.
- The speakers stated that proton pump inhibitors can counteract IL-13 effects on impedance and alter the epithelial transcriptome beyond acid suppression, but are effective in only a fraction of patients.
- New data presented showed that two grams of SDS-containing toothpaste caused detectable decreases in oesophageal impedance, raising the speaker's concern that daily detergent ingestion may counteract therapeutic barrier restoration.
- The speakers proposed that mucosal impedance measurement and histological assessment beyond eosinophil counts (including dilated intercellular spaces and basal zone hyperplasia) may serve as treatment endpoints to define true epithelial healing in EOE.

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