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Summary

THE DISAPPEARANCE OF CELL DEATH BRAKES: A NEW MOLECULAR DEFECT IN INFLAMMATORY BOWEL DISEASE (IBD)

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IBD

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A comprehensive assessment of programmed cell death in adult IBD patients reveals loss of cellular death brakes (cIAP-1, cIAP-2, cFLIPL) even in histologically non-inflamed tissue, unleashing both apoptosis and necroptosis.

KEY POINTS

- Interim analysis of intestinal biopsies from 28 healthy controls and 52 IBD patients (25 ulcerative colitis, 25 Crohn's disease, 2 pouchitis) shows suppression of cell death inhibitory proteins cIAP-1, cIAP-2 and cFLIPL even in non-inflamed tissue from IBD patients.
- Loss of these cellular death brakes is associated with statistically significant increases in cleaved caspase-3 (apoptosis marker) and phosphorylated RIPK-3 (necroptosis marker) in nearby tissue, occurring despite apparent mucosal and histologic healing.
- This is the first study to comprehensively assess programmed cell death in intestinal tissue of adult IBD patients using a novel toolbox of affinity reagents, employing immunoblotting, immunohistochemistry, immunofluorescence, RNA sequencing, and organoid development.
- The findings challenge current dogma that necroptosis occurs in the absence of apoptosis and that necroptosis is universally increased in all forms of IBD.

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