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Summary

FAMILIAL COLORECTAL CANCER HISTORY IS ASSOCIATED WITH A DISTINCT IMMUNE–GENOMIC PHENOTYPE IN MICROSATELLITE-STABLE RECTAL CANCER: A PROSPECTIVE MULTICENTRE COHORT STUDY

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Family history of colorectal cancer, particularly an affected first-degree relative, may identify a biologically distinct subgroup of microsatellite-stable rectal cancer characterized by altered epithelial-immune coordination and impaired coupling between genomic stress and immune surveillance.

KEY POINTS

- Among 374 MSS rectal cancer patients (25% FH^+, 17% FDR^+), those with familial aggregation showed distinct immune patterns: in neoadjuvant-naive disease, FDR^+ cases had higher epithelial $CD80^+$ cell density in normal mucosa.
- After neoadjuvant chemoradiotherapy, FH^+ patients exhibited increased activated $CD8^+CD28^+$ T cells and reduced epithelial HLA-ABC expression, while FDR^+ cases showed lower $CD3^+$ T-cell density.
- Transcriptomic analyses revealed a relatively quiescent familial mucosal phenotype, with attenuation of DNA damage repair, proliferative, metabolic, angiogenic, and immune pathways.
- In sporadic MSS rectal cancer, higher mutational ratio was associated with improved disease-free survival and with immune features including $CD8^+$ T-cell density and epithelial CD80 expression, whereas in FH^+ cancer, mutational ratio was not associated with outcome and appeared uncoupled from these immune parameters.
- Family history may help refine biological subclassification within MSS rectal cancer and inform design of trials evaluating immunotherapy-based strategies in this traditionally immune-resistant disease.

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