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Summary

[68GA]FAPI PET-MRI QUANTIFIES FIBROBLAST ACTIVITY BURDEN AND DIFFERENTIATES FIBROSTENOSIS FROM AN INFLAMMATORY PHENOTYPE IN CROHN'S DISEASE

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[68Ga]FAPI PET-MRI differentiated fibrostenosing from inflammatory Crohn's disease, with significantly higher radiotracer uptake in stricturing disease (median SUVpeak 3.0 vs 1.1, P=0.012).

KEY POINTS

- [68Ga]FAPI uptake was significantly elevated in fibrostenosing Crohn's disease compared to inflammatory CD (median SUVpeak 3.0 vs 1.1, P=0.012) and non-IBD controls (3.0 vs 1.3, P=0.008) in 24 participants from the FATE-CD prospective cohort.
- Molecular validation using single-cell transcriptomics from 61 intestinal samples showed FAP expression enriched 5.2-fold in fibroblast subpopulations within fibrotic niches versus non-fibrotic tissue (P<0.001), supporting biological specificity of the PET signal.
- FAPI uptake did not correlate with conventional biochemical markers of inflammation, and wide heterogeneity of uptake was observed across strictured regions classified by STAR consortium criteria.
- [68Ga]FAPI binds to fibroblast activation protein on activated fibroblasts, key drivers of extracellular matrix deposition, offering non-invasive visualisation and quantification of active fibrosis within the bowel wall.
- The technique is proposed as a promising biomarker for identifying stricturing phenotypes that warrants validation in larger cohorts; next steps include histopathology correlation and longitudinal imaging in newly diagnosed patients receiving anti-TNF therapy.

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