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Summary

B CELLS PROMOTE FIBROBLAST ACTIVATION THROUGH A BIDIRECTIONAL STROMAL FEEDBACK LOOP IN FIBROSTENOTIC CROHN'S DISEASE

Stefania Vetrano

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B cells drive intestinal fibrosis in Crohn's disease through direct contact-dependent bidirectional crosstalk with fibroblasts, inducing myofibroblast differentiation and collagen deposition comparable to TGF-β stimulation.

KEY POINTS

- Activated B cells induced myofibroblast differentiation (ACTA2, FAP) and collagen deposition at levels comparable to TGF-β stimulation in co-cultures of primary human intestinal fibroblasts and donor-derived B cells (n=5).
- Fibrosis developed in wild-type but not NSG immunodeficient mice in a TNBS model (n=5), indicating a requirement for adaptive immunity.
- Fibrostenotic Crohn's disease tissue (N=14) showed marked enrichment of $CD20^+$ B cells in dense lymphoid aggregates within fibrotic areas, with germinal center B cells closely apposed to $\alpha\text{-SMA}^+$ fibroblasts.
- Stromal fibroblasts activated resting B cells, inducing MYC and FOS expression; this effect required direct cell contact and was absent with healthy fibroblasts.
- IgA was increased both locally and systemically ($p<0.01$) and circulating naïve B cells were expanded in Crohn's disease ($p<0.01$), suggesting active recruitment to inflamed tissue.
- The bidirectional B cell–fibroblast circuit may maintain fibrosis independently of inflammatory triggers, and targeting this immune–stromal feedback loop may represent a therapeutic strategy to prevent or reverse intestinal strictures.

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