

Heterotopic Osseous Metaplasia in a Juvenile Colorectal Polyp: An Exotic Finding Mediated by Inflammatory Microenvironment and Mechanical Stress.

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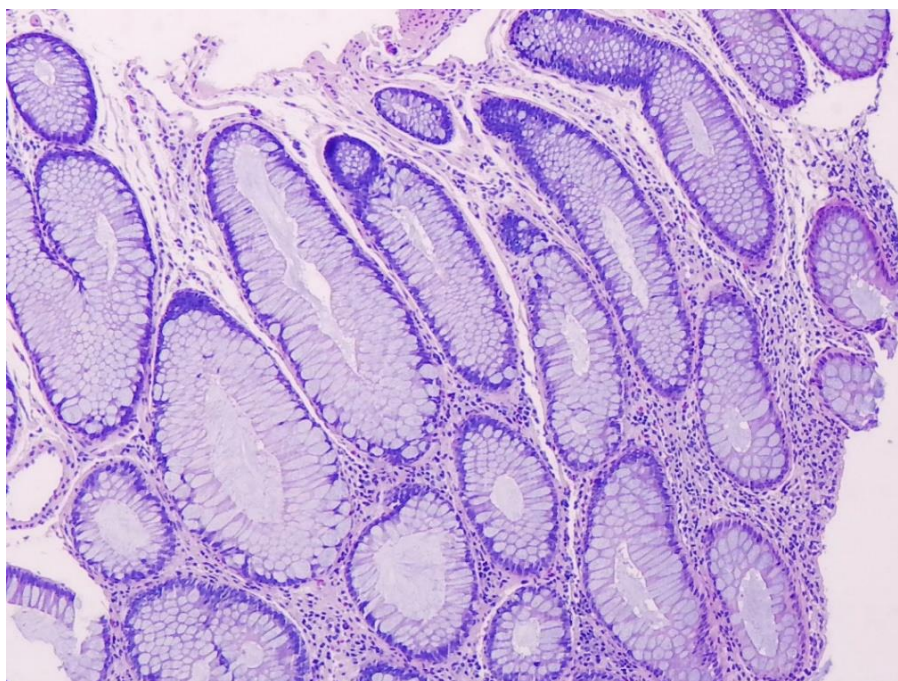
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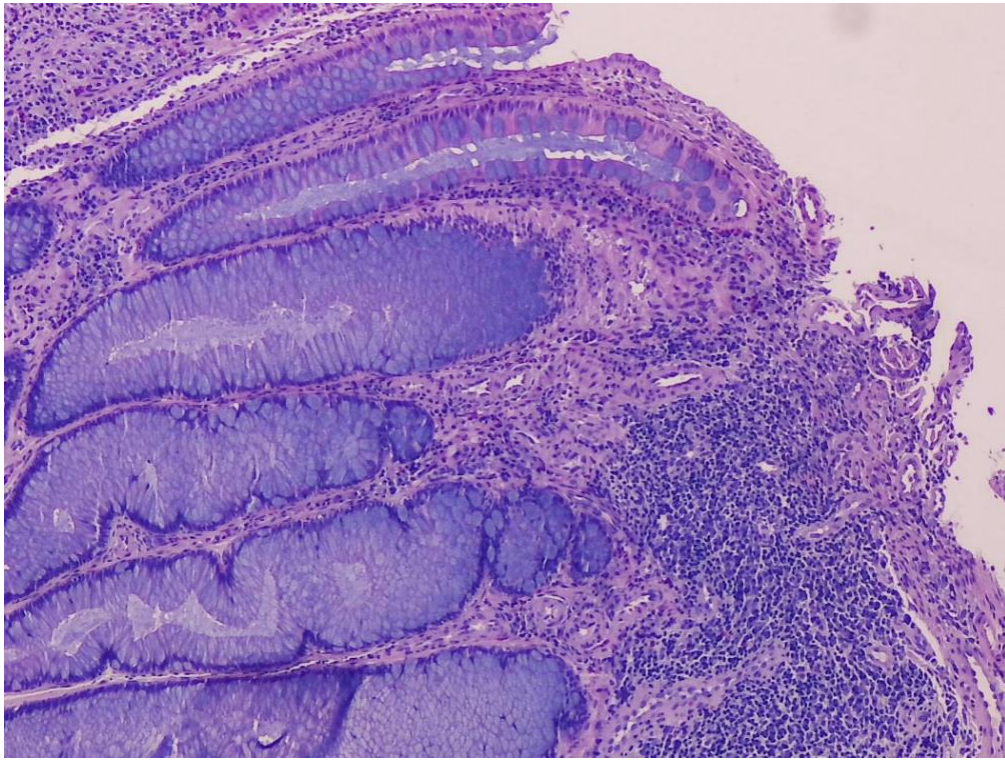
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To the Editor:

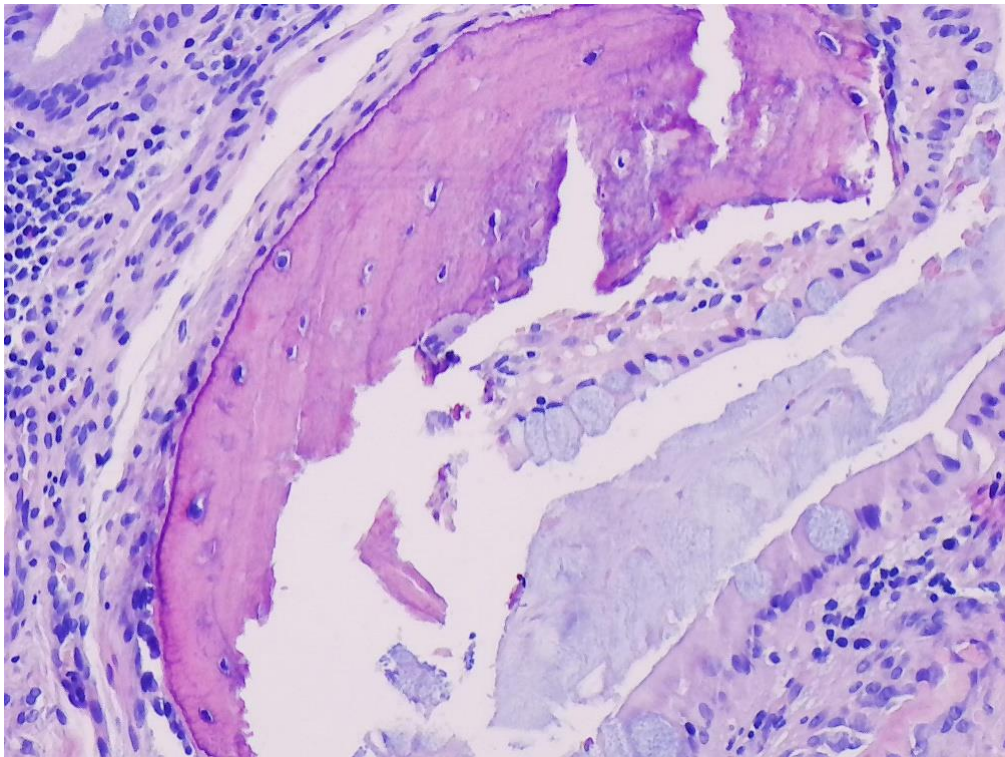
The presence of heterotopic bone tissue within the gastrointestinal tract is an extremely uncommon phenomenon that challenges the traditional understanding of mesenchymal cell plasticity [1]. While it has been occasionally described in association with gastric and colorectal mucinous adenocarcinomas, its finding within benign polypoid lesions in pediatric or adolescent patients represents an authentic diagnostic rarity [2]. We present the case of a 15-year-old patient in whom this exotic entity was incidentally identified. The patient, a 15-year-old male, presented

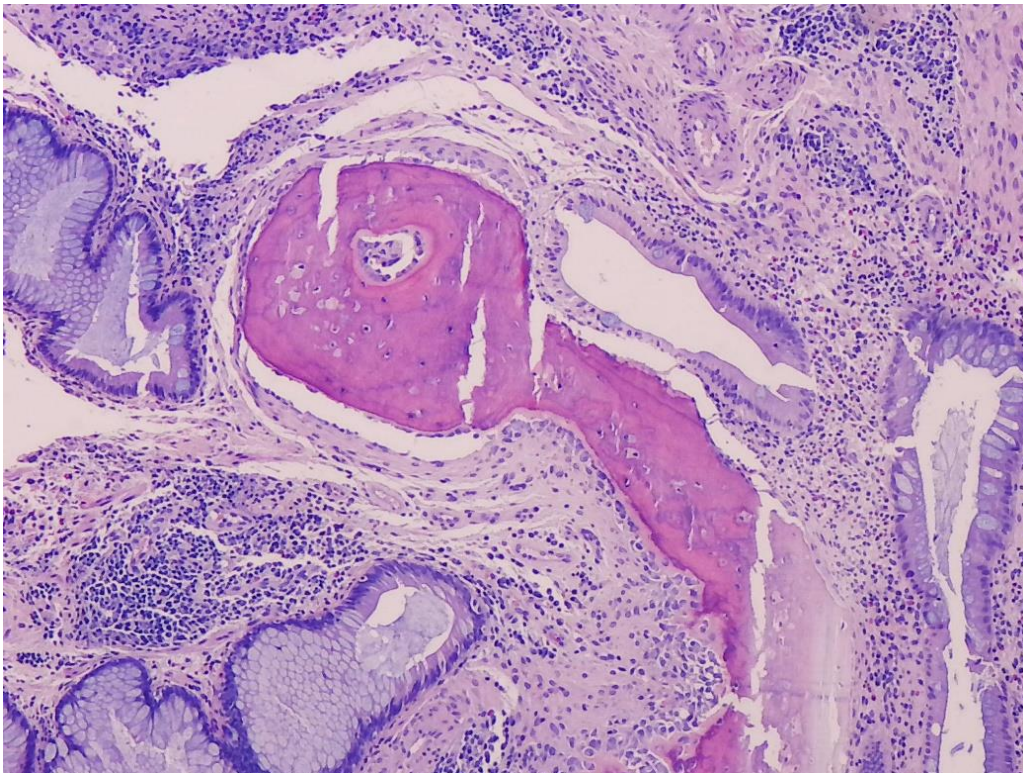
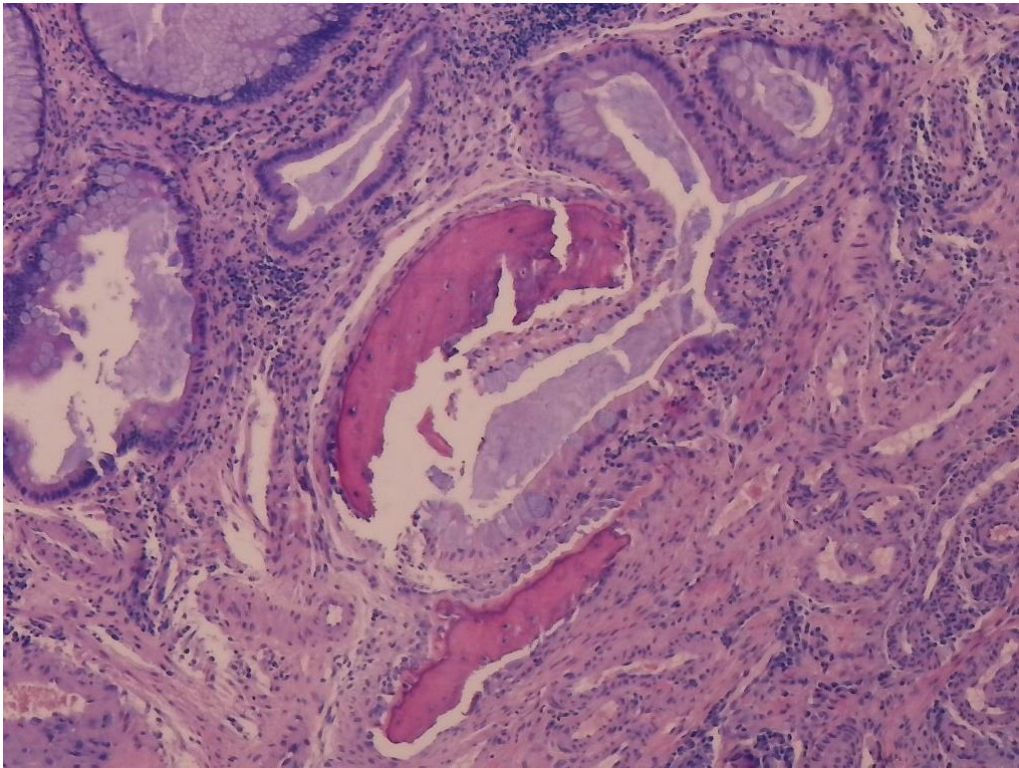
with isolated episodes of rectal bleeding. Video colonoscopy revealed a sessile polypoid lesion measuring 0.8 cm in maximum dimension, located in the colon at 40 cm from the anal margin, exhibiting marked superficial ulceration. Endoscopic polypectomy was successfully performed to achieve complete excision. The histopathological evaluation revealed a tissue architecture compatible with a mucous polyp of the hamartomatous/juvenile spectrum, characterized by dilated, cystic colorectal crypts embedded in an edematous stroma with a dense chronic lymphoplasmacytic inflammatory infiltrate (Figures 1 and 2).



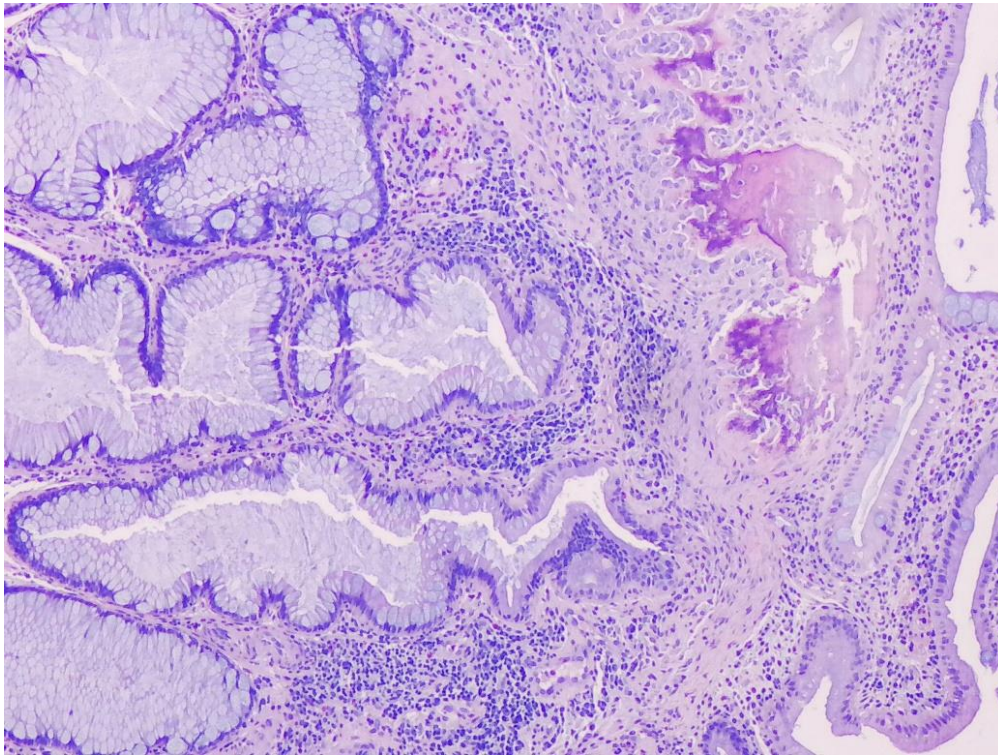


The most surprising and disruptive finding was located in the deep lamina propria, where multiple well-defined trabeculae of mature lamellar bone were identified, featuring viable osteocytes residing within their lacunae (Figures 3, 4, and 5).





These bone trabeculae were rimmed by a thin layer of cells with osteoblastic morphology, completely lacking cytological atypia or mitotic activity. This osseous tissue demonstrated close proximity to areas of granulation tissue and superficial epithelial ulceration (Figure 6).



From a pathogenic perspective, osseous metaplasia occurring within non-neoplastic benign lesions poses a fascinating biological scenario [3]. Traditionally, chronic inflammation and tissue necrosis have been postulated as the primary inductive stimuli. The local release of pro-inflammatory and reparative cytokines promotes the trans differentiation of local stromal fibroblasts or mesenchymal stem cells toward an osteoblastic lineage [3]. Furthermore, it has been shown that molecular paracrine factors and bone morphogenetic proteins (BMPs) belonging to the TGF-beta superfamily modulate this microenvironment, thereby facilitating ectopic osteogenesis [4]. However, to provide a distinctive and highly original perspective on this case, we suggest that a crucial determinant factor in young patients might be mechanotransduction. The constant mechanical stress and shearing forces exerted by intestinal peristalsis upon a sessile polyp—particularly one that is already ulcerated and structurally weakened—alter the cytoskeleton of the pluripotential stromal cells within the lamina propria. This biophysical signaling acts synergistically with the hyperemic and inflammatory microenvironment, triggering an aberrant yet strictly regulated and completely benign osteogenic transcriptional cascade [4]. Unlike the osseous metaplasia observed in colonic malignancies, where it has historically correlated with mucinous variants or advanced tumor stages and aggressive clinical behavior [5], in the context of a benign polyp this finding is purely anecdotal and lacks unfavorable clinical implications following complete

excision. We consider that documenting these cases is fundamental to avoiding diagnostic pitfalls—such as confusion with impacted exogenous bone fragments or choristomas—and to expanding our comprehension regarding the mechanisms of in vivo cellular reprogramming under combined mechanical and inflammatory stimuli.

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