Osseous metaplasia of the colon in an ulcerative proctosigmoiditis

A 77-year-old woman presented with acute abdominal pain and hematochezia. The patient reported a normal routine colonoscopy 2 years ago. Her long-term medication consisted of statins, allopurinol, and triazolam. Additionally she had a short-term analgesic medication (naproxen). At colonoscopy, a marginal macroscopic inflammation of the sigmoid colon and a 4-mm rectal polypoid lesion were visible (Fig. 1). During histological examination, a mixed inflammatory infiltrate was visible in the lamina propria, with multiple crypt abscesses (Fig. 2). The polypoid lesion showed hyperplastic crypts and foci of heterotopic bone formation (Fig. 3). Small regions with surface ulceration could be seen. Thus the histopathological diagnosis was ulcerative proctosigmoiditis with metaplastic bone formation. It seems that the bone formation had persisted for a long time and that the finding of ulcerative colitis was overlaid by an infectious component. Due to a normal number of leukocytes in the blood sample, and unremarkable stool samples, a parasitic infection was excluded. Heterotopic ossification in the gastrointestinal tract is described predominantly in mucin-producing carcinomas of the colon [1]. Descriptions of ossification within inflammatory gastrointestinal lesions are extremely rare, and the pathological mechanisms remain unclear. Sperling et al. assumed that bone-forming osteoblasts differentiate from immature fibroblasts [2]. Rifas et al. demonstrated that T-cell cytokines regulate the differentiation process of human mesenchymal stromal cells into osteoblasts by inducing bone morphogenetic protein-2 (BMP-2) [3]. Yu et al. reported that an active actin receptor-like kinase-2 (ALK2), activated by BMP receptor 1, leads to ectopic bone formation [4]. Finally, Shafritz et al. showed that overexpression of BMP-4 in lymphocytes is associated with ectopic osteogenesis in fibrodysplasia ossificans progressiva [5]. Overall, chronic inflammatory processes seem to play an important role in ectopic bone formation.

Competing interests: None

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Fig. 1 Endoscopic view of the lesion.

Fig. 2 Microscopic image showing colitis with crypt abscess (hematoxylin and eosin stain, ×400).

Fig. 3 Microscopic image showing colitis with heterotopic ossification, ulceration, and an osteoclastic giant cell (shown also in enlarged inset) (hematoxylin and eosin stain, ×40).
References

1 Haque S, Eisen RN, West AB. Heterotopic bone formation in the gastrointestinal tract. Arch Pathol Lab Med 1996; 120: 666–670
4 Yu PB, Deng DY, Lai CS et al. BMP type 1 receptor inhibition reduces heterotopic ossification. Nat Med 2008; 14: 1363–1369

Bibliography

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