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.

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L. Veits1,2, A. Perathoner3, C. Profanter3, C. Falkeis1,2, G. Mikuz1, C. Ensinger1
1 Institute of Pathology, Medical University of Innsbruck, Innsbruck, Austria
2 Institute of Pathology, Klinikum Bayreuth, Bayreuth, Germany
3 Department of Visceral, Transplant and Thoracic Surgery, Medical University of Innsbruck, Innsbruck, Austria
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Corresponding author
L. Veits, MD
Institute of Pathology
Klinikum Bayreuth
Preuschwitzerstrasse 101
95445 Bayreuth
Germany
Fax: +49-921-4005609
lothar.veits@klinikum-bayreuth.de