Rectal carcinoid tumor: a delayed localized recurrence 23 years after endoscopic resection

Rectal carcinoid tumors are rare representing approximately 2% of rectal malignancies [1]. While treatment approaches vary and can include endoscopic or surgical resection, small (<1 cm) carcinoid tumors have extremely low rates of local recurrence and metastasis [1–3]. Our case is the first describing a delayed recurrence of rectal carcinoid tumor more than 20 years after initial endoscopic resection. A 67-year-old man presented for surveillance colonoscopy with a history of rectal carcinoid tumor. He had undergone endoscopic resection of a 4 mm rectal carcinoid tumor 5 cm from the anal verge 23 years earlier (Fig. 1). Three surveillance colonoscopies since resection were negative for residual carcinoid tissue. Recent colonoscopy showed a scar in the rectum with a 5-mm raised area (Fig. 2), which was resected via snare cautery polypectomy. Pathology was consistent with rectal carcinoid tumor (Fig. 3 and Fig. 4). Immunohistochemical stain for synaptophysin was positive; chromogranin was negative (Fig. 5). Less than 1% of cells stained positive for Ki-67, indicative of a low proliferative rate and metastatic potential (Fig. 6) [4]. Flexible sigmoidoscopy 3 months later showed no recurrence.

Rectal carcinoids are well-differentiated hindgut neuroendocrine tumors and are the most common location for colorectal carcinoids [1–3, 5]. Hindgut tumors typically display a trabecular growth pattern whereas midgut tumors usually show a nested pattern [5]. As in our case, the majority are found incidentally during endoscopic screening [1–3]. Lesions less
than 1 cm have very low rates of metastasis and local recurrence, and endoscopic resection via polypectomy or endoscopic mucosal resection is appropriate [1–3]. Our case demonstrates that delayed recurrence of rectal carcinoid tumors can occur decades later in lesions with low mitotic rates and also highlights the importance of vigilantly assessing and sampling the scar site for suspicious tissue via biopsy or polypectomy, even years after resection.

Endoscopy_UCTN_Code_CCL_1AD_2AB

Competing interests: None

Fig. 5 Immunohistochemical staining for synaptophysin was positive, demonstrated by diffuse red staining in cytoplasm (original magnification: × 100).

Fig. 6 Staining with Ki-67 showing that less than 1% of tumor cells stained positive (red/brown dots represent nuclear staining for proliferation marker Ki-67) (H&E; original magnification: × 1).

Acknowledgments
This material is the result of work supported with resources and the use of facilities at the John D. Dingell VAMC, Detroit, Michigan. The content does not represent the views of the Department of Veterans Affairs or the United States Government.

References
5. Odze RD, Goldblum JR. Surgical pathology of the GI tract, liver, biliary tract, and pancreas. 2nd ed. Philadelphia: Elsevier; 2009

Stephanie Judd1,2, Sharad Nangia1,2, Edi Levi3, Fadi Antaki1
1 Division of Gastroenterology, Department of Internal Medicine, John D. Dingell VA Medical Center and Wayne State University School of Medicine, Detroit, United States
2 Detroit Medical Center, Detroit, United States
3 Department of Pathology, John D. Dingell VA Medical Center and Wayne State University School of Medicine, Detroit, United States

Corresponding author
Stephanie Judd, MD
John D. Dingell VA Medical Center – Gastroenterology
4646 John K. Road
11M-GAS
Detroit
Michigan 48201
United States
Fax: +1-313-576-1237
sjudd@med.wayne.edu

Fig. 5

Fig. 6

Bibliography
DOI http://dx.doi.org/10.1055/s-0034-1377950
Endoscopy 2014; 46: E555–E556
© Georg Thieme Verlag KG
Stuttgart · New York
ISSN 0013-726X