

Early-onset adenocarcinoma of the ampullary rectum: a case report

Ziyali I. Rzayev

rzayevziyali264@gmail.com

III of Azerbaijan Medical University

Fariz H. Jamalov

III of Azerbaijan Medical University

Dilara A. Akyuz

III of Azerbaijan Medical University

Mazahir M. Mursalov

III of Azerbaijan Medical University

Imran A. Shafiev

III of Azerbaijan Medical University

Azer Sh. Gasimov

III of Azerbaijan Medical University

Terane P. Jamalov

III of Azerbaijan Medical University

Khatire S. Rahimova

III of Azerbaijan Medical University

Khaleddin N. Musayev

III of Azerbaijan Medical University

Sardar A. Asadov

III of Azerbaijan Medical University

Elchin A. Abdinov

III of Azerbaijan Medical University

Case Report

Keywords: Early-onset colorectal cancer, rectal adenocarcinoma, ampullary rectum, poorly differentiated adenocarcinoma, lymphovascular invasion, perineural invasion, case report

Posted Date: October 14th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10658319/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License.
[Read Full License](#)

Additional Declarations: No competing interests reported.

Abstract

Background: Early-onset colorectal cancer can present with nonspecific gastrointestinal symptoms, contributing to delayed diagnosis. Adenocarcinoma arising in the ampullary region of the rectum may pose additional diagnostic and surgical challenges because of its distal location.

Case presentation: A 33-year-old man presented with nonspecific gastrointestinal complaints. Colonoscopy demonstrated an exophytic tumor involving the lower and middle ampullary regions of the rectum, with luminal narrowing and contact bleeding. Computed tomography showed rectal wall thickening and mesorectal lymph nodes. Histopathological examination confirmed poorly differentiated adenocarcinoma (G3) with lymphovascular and perineural invasion. The final pathological stage was pT3N0M0, with no metastatic lymph nodes identified. The patient underwent abdominoperineal excision of the rectum with sigmoid colon pull-through and had an uncomplicated early postoperative course.

Conclusions: This case emphasizes the need to consider colorectal malignancy in younger adults with persistent or unexplained gastrointestinal symptoms. Prompt endoscopic assessment and histopathological confirmation are essential when symptoms are atypical or persistent. Histological risk features should be integrated with tumor stage when planning postoperative management.

Background

Colorectal cancer is increasingly recognized in younger adults, and early-onset disease may be diagnosed after a period of nonspecific or initially attributed symptoms. Contemporary epidemiological analyses have reported a rising incidence of colorectal cancer among people younger than 50 years [1, 16, 17], and molecular studies indicate that early-onset tumors can display distinct age-related mutational signatures compared with later-onset disease [2], increasing the importance of timely diagnostic assessment in symptomatic younger patients.

Rectal adenocarcinoma develops through interacting genetic, environmental, inflammatory, and lifestyle-related mechanisms. Established molecular pathways include alterations involving APC, KRAS, TP53, and the DNA mismatch-repair system, while microsatellite instability (MSI) represents an important molecular phenotype with diagnostic and therapeutic implications [3, 4, 12, 14, 15]. Chronic inflammatory bowel disease and dietary factors, including high intake of red or processed meat, have also been associated with colorectal cancer risk [6–10].

Distal rectal tumors may produce symptoms that overlap with common benign anorectal disorders, including altered bowel habits and rectal bleeding. Such overlap can contribute to delayed investigation. The present case is clinically relevant because it combines young age at presentation, nonspecific gastrointestinal symptoms, a tumor involving the lower and middle ampullary regions of the rectum, and unfavorable histopathological features despite the absence of nodal or distant metastases.

Case presentation

Patient information

A 33-year-old man (born in 1991) was admitted to the Department of Surgical Diseases, Azerbaijan Medical University, with nonspecific gastrointestinal complaints. The source clinical record provided for this manuscript does not contain sufficient information on family history, occupational history, previous medications, allergies, or relevant comorbidities; therefore, these details are not inferred here.

Clinical findings

On admission, the patient was described as being in moderate general condition. The skin and mucous membranes were pale. His pulse rate was 100 beats per minute and blood pressure was 100/60 mmHg. Abdominal tenderness and absent bowel sounds were documented.

Diagnostic assessment

Initial colonoscopy demonstrated a tumor in the lower ampullary region of the rectum with a tendency to bleed. Computed tomography showed rectal wall thickening of up to 13 mm and mesorectal lymph nodes.

Repeat colonoscopy was advanced from the anal canal to the splenic flexure. Exophytic tumor growths were identified in the lower and middle ampullary regions of the rectum. The lesions caused significant luminal narrowing, bled on contact, and were partially covered by necrotic deposits. Histopathological examination of biopsy specimens demonstrated extension of the tumor into the anal canal and confirmed rectal adenocarcinoma.

Final histopathological examination of the resection specimen demonstrated poorly differentiated adenocarcinoma (G3), measuring 30 mm, with lymphovascular and perineural invasion. No metastatic lymph nodes were identified. The pathological stage was pT3N0M0 according to the TNM classification reported in the source manuscript.

Therapeutic intervention

Treatment planning incorporated clinical, radiological, and morphological findings. The patient underwent abdominoperineal excision of the rectum with sigmoid colon pull-through. The operative description in the source manuscript included abdominal cavity inspection, sigmoid and rectal mobilization, tumor resection, sigmoid colon descent, anastomosis formation, drainage, and closure.

The source manuscript states that adjuvant chemotherapy was planned after multidisciplinary consultation. The specific chemotherapy regimen and subsequent oncological treatment were not provided and are therefore not added to this revision.

Follow-up and outcomes

The early postoperative course was reported as stable. Bowel function was restored by postoperative day 3, no postoperative complications were documented, and the patient was discharged on postoperative day 5. The available manuscript does not provide a sufficiently detailed long-term follow-up period or subsequent imaging/oncological outcomes.

Patient perspective

A patient-perspective statement was not included in the source manuscript. If available, a brief statement describing the patient's experience of the symptoms, diagnostic process, treatment, and recovery should be added before submission.

Discussion

This case illustrates the diagnostic challenge posed by early-onset rectal adenocarcinoma in a young adult with nonspecific gastrointestinal complaints. Symptoms such as altered bowel habits or rectal bleeding may initially be attributed to benign anorectal disorders, potentially delaying endoscopic assessment. Current epidemiological evidence supports increasing clinical awareness of colorectal cancer in younger adults [1, 16, 17].

The biological basis of colorectal carcinogenesis is heterogeneous. The classical adenoma–carcinoma model includes sequential alterations involving APC, KRAS, and TP53, while other tumors develop through mismatch-repair deficiency and MSI-related pathways [3, 4, 12, 14, 15]. Environmental and inflammatory factors may contribute to carcinogenesis, including inflammatory bowel disease and dietary exposure to red or processed meat [6–10]. These mechanisms provide important context for younger patients, although the available clinical information for this individual case does not establish a specific etiologic pathway.

The tumor in this case was poorly differentiated and demonstrated both lymphovascular and perineural invasion. Tumor-associated angiogenesis and the emergence of invasive tumor-cell subpopulations at the invasive front are recognized biological mechanisms that support local invasion and dissemination in colorectal cancer [5, 11]. Although the final pathological stage was pT3N0M0 and no metastatic lymph nodes were identified, these histopathological characteristics indicate biologically unfavorable features and are clinically relevant when assessing recurrence risk and postoperative management.

Tumor location was also important in the management of this patient. Lesions involving the lower rectal region may limit the feasibility of sphincter-preserving procedures and can require radical surgery depending on local extension and the relationship of the tumor to the anal canal and surrounding structures. In this case, the tumor extended into the anal canal and was associated with marked luminal narrowing, providing a clinical rationale for radical surgical management.

The case also supports a multidisciplinary approach involving surgery, radiology, pathology, and oncology. Molecular assessment, including mismatch-repair/MSI status and RAS mutation testing, can be relevant to treatment selection in colorectal cancer; however, the source manuscript does not report the results of molecular testing in this patient. Such results should not be assumed and should be added only if they are documented in the clinical record.

The principal clinical lesson is that young age should not by itself lower the threshold for diagnostic evaluation when gastrointestinal symptoms are persistent, unexplained, or associated with rectal bleeding. The combination of early-onset disease and unfavorable histopathological findings in this case reinforces the importance of timely colonoscopic evaluation and coordinated postoperative management.

Conclusions

Early-onset rectal adenocarcinoma can present with nonspecific symptoms and may be diagnosed only after locally advanced disease has developed. In young adults with persistent or unexplained gastrointestinal symptoms, timely endoscopic evaluation should be considered rather than attributing symptoms solely to benign anorectal conditions. Histopathological features such as poor differentiation, lymphovascular invasion, and perineural invasion should be integrated with TNM stage when assessing prognosis and planning postoperative care. Further molecular characterization may support individualized management when clinically indicated.

Abbreviations

AJCC: American Joint Committee on Cancer; CT: computed tomography; MSI: microsatellite instability; TNM: tumor, node, metastasis

Declarations

Ethics approval and consent to participate

The source manuscript states that the study was conducted in accordance with ethical standards and that patient confidentiality was maintained. It also states that informed consent was obtained for the use of clinical data for scientific publication. The exact institutional ethics committee/IRB name, approval number, or waiver status was not provided in the source manuscript and should be added if applicable.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this

journal.

Competing interests

The source manuscript does not provide a competing-interests statement. The authors should add the appropriate declaration before submission.

Funding

Funding information was not provided in the source manuscript. Add a statement such as “No specific funding was received for this work” only if this is factually correct.

Authors’ contributions

An authors’ contributions statement was not included in the source manuscript. This should be completed by the authors before submission, using each author’s actual contribution.

Acknowledgements

No acknowledgements were provided in the source manuscript.

Availability of data and materials

No additional datasets were identified in the source manuscript. If the journal requires a data-availability statement, the authors should provide one appropriate to the underlying clinical records and privacy requirements.

References

1. Araghi M, Soerjomataram I, Bray F, et al. Global trends in early-onset colorectal cancer incidence: an international analysis. *Lancet Oncol.* 2024;25(3):312–325. doi:10.1016/S1470-2045(24)00045-6.
2. Koveitypour Z, et al. Age and geographic variation in mutational signatures of colorectal cancer. *Nat Commun.* 2019;10:1–12.
3. Markowitz SD, Bertagnolli MM. Molecular basis of colorectal cancer. *N Engl J Med.* 2009;361(25):2449–2460. doi:10.1056/NEJMr0804588.
4. Fearon ER, Vogelstein B. A genetic model for colorectal tumorigenesis. *Cell.* 1990;61(5):759–767. doi:10.1016/0092-8674(90)90186-I.

5. Folkman J. Role of angiogenesis in tumor growth and metastasis. *Semin Oncol.* 2002;29(6 Suppl 16):15–18. doi:10.1053/sonc.2002.37263.
6. Nguyen LH, et al. Dietary red meat intake and a colorectal cancer somatic mutational signature. *Cancer Discov.* 2021;11(10):2488–2505. doi:10.1158/2159-8290.CD-20-1490.
7. Eaden JA, Abrams KR, Mayberry JF. The risk of colorectal cancer in ulcerative colitis: a meta-analysis. *Gut.* 2001;48(4):526–535. doi:10.1136/gut.48.4.526.
8. Jess T, Rungoe C, Peyrin-Biroulet L. Risk of colorectal cancer in inflammatory bowel disease: a meta-analysis of population-based cohort studies. *Clin Gastroenterol Hepatol.* 2012;10(6):639–645. doi:10.1016/j.cgh.2012.01.010.
9. Chan DSM, Lau R, Aune D, et al. Red and processed meat and colorectal cancer incidence: meta-analysis of prospective studies. *PLoS One.* 2011;6(6):e20456. doi:10.1371/journal.pone.0020456.
10. Alexander DD, Weed DL, Cushing CA. Red and processed meat and colorectal cancer: a review of published meta-analyses. *Nutr Res Rev.* 2015;28(1):1–23. doi:10.1017/S0954422415000035.
11. Brabletz T, Jung A, Spaderna S, Hlubek F, Kirchner T. Migrating cancer stem cells—an integrated concept of malignant tumour progression. *Nat Rev Cancer.* 2005;5(9):744–749. doi:10.1038/nrc1694.
12. Esteller M. Epigenetics in cancer. *N Engl J Med.* 2008;358(11):1148–1159. doi:10.1056/NEJMr072067.
13. Kraus S, Arber N. Inflammation and colorectal cancer. *Curr Opin Pharmacol.* 2009;9(4):405–410. doi:10.1016/j.coph.2009.06.006.
14. Baker SJ, Preisinger AC, Jessup JM, et al. p53 gene mutations occur in combination with 17p allelic deletions as late events in colorectal tumorigenesis. *Cancer Res.* 1990;50(23):7717–7722.
15. Boland CR, Goel A. Microsatellite instability in colorectal cancer. *Gastroenterology.* 2010;138(6):2073–2087.e3. doi:10.1053/j.gastro.2009.12.064.
16. Siegel RL, Giaquinto AN, Jemal A. Cancer statistics, 2024. *CA Cancer J Clin.* 2024;74(1):17–48. doi:10.3322/caac.21820.
17. Akimoto N, Ugai T, Zhong R, et al. Rising incidence of early-onset colorectal cancer—a call for action. *Nat Rev Clin Oncol.* 2023;20(4):230–243. doi:10.1038/s41571-023-00727-7.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [CAREchecklistcompleted.docx](#)