

BOWEL OBSTRUCTION CAUSED BY AN UNUSUALLY LATE CASE OF METACHRONOUS COLORECTAL CANCER AFTER 22 YEARSTudor Negru², Dragoş Molnar^{2,*}, Cătălin Cosma^{1,2}, Călin Molnar^{1,2}¹George Emil Palade University of Medicine, Pharmacy, Science and Technology, Târgu Mureş, Romania²General Surgery I, Târgu-Mureş County Emergency Hospital, Târgu-Mureş, Romania;**REVIEW**Doi: 10.33695/rojes.v7i1.96
Accepted: 02.07.2025**Abstract**

Metachronous colorectal cancer (CRC) refers to a new primary colorectal malignancy developing at least six months after the initial diagnosis and treatment of a primary CRC. While most cases occur within the first 5 years postoperatively, delayed presentations beyond a decade are rare and poorly characterized. We report the case of a 62-year-old male with a history of transverse colon cancer resected in 2003 and treated with adjuvant chemotherapy. In 2025, after experiencing multiple episodes of bowel obstruction and initially refusing surgery at another clinic, he presented to our surgical department with diffuse abdominal pain, nausea, vomiting, altered bowel habits, and bloating. Imaging revealed a thickened segment of the descending colon with near-complete luminal narrowing and upstream distension. Surgery confirmed two synchronous tumors: (A) a low-grade adenocarcinoma of the descending colon infiltrating to the subserosa without nodal metastasis (pT3N0R0) and loss of MSH-6 expression; and (B) a mucinous adenocarcinoma of the rectum infiltrating the muscularis propria without nodal metastasis (pT2N0R0) and also with loss of MSH-6 expression. The patient underwent total colectomy with ileorectal anastomosis and segmental jejunal resection. This case highlights the possibility of extremely delayed metachronous CRC, occurring over two decades after initial treatment. It underscores the importance of individualized long-term surveillance strategies and the role of colonoscopy in detecting asymptomatic lesions. Recognition of atypical recurrence patterns is essential for timely diagnosis and intervention.

Corresponding author:
Dragoş Molnar
dragosmolnar2000@yahoo
.com

Keywords: metachronous colorectal cancer, colonoscopy surveillance, delayed recurrence, total colectomy

Introduction

Colorectal cancer (CRC) is a significant global health issue, with around two million new cases identified annually. Patients who

have curative-intent surgical resection for colorectal cancer (CRC) face a persistent risk of developing new tumors in the colon or rectum, referred to as metachronous colorectal tumors. Metachronous colorectal tumors are

characterized as new primary colorectal malignancies that arise at a minimum of six months following the initial excision of a colorectal carcinoma [1]. Their reported incidence varies from 0.5% to 9%, with a mean of approximately 1.6% [1].

Individuals having a history of colorectal cancer exhibit an elevated risk of acquiring future neoplasms in the large intestine. In one study, the occurrence of metachronous cancer was 4.6%, while more than 20% of individuals acquired benign adenomas with malignant potential [2]. Nonetheless, these statistics may be somewhat inflated, as certain metachronous lesions could potentially represent synchronous cancers overlooked during the initial diagnosis, especially when colonoscopy was not conducted at the time of the original surgery [2].

Metachronous colorectal cancer (CRC) is frequently linked to local recurrence or new primary tumors, but it may also present as metastatic illness resulting from micrometastases that were missed during the first treatment. Metachronous metastases typically arise within two years following surgery and are observed in up to 23% of patients post-treatment [3,4]. Notwithstanding progress in surveillance and treatment methods, the prognosis is unfavorable; over 70% of patients with metachronous metastases will die within five years [4].

Case presentation

We report the case of a 62-year-old male patient with a significant medical history, including arterial hypertension (HTN), ischemic heart disease (IHD), pulmonary hypertension (PH), atrioventricular block (AVB), chronic hepatitis C infection, and active tobacco use. The patient also had a history of colon cancer, for which he underwent surgical treatment in 2003 followed by four cycles of adjuvant chemotherapy. At that time, he was diagnosed with a stenosing transverse colon tumor that penetrated into the

mesentery and abdominal wall. A segmental resection of the transverse colon was performed, with a side-to-side anastomosis, Douglas pouch drainage, left paracolic drainage, and subhepatic drainage.

In 2025, 22 years after the initial surgery, after experiencing multiple episodes of occlusive syndrome and refusing surgical treatment at another clinic, the patient presented to the General Surgery Clinic I of the County Emergency Clinical Hospital in Târgu Mureș for scheduled evaluation. He reported a five-day history of diffuse abdominal pain, nausea, vomiting, altered bowel habits (alternating diarrhea and constipation), and marked abdominal bloating.

On admission, the patient's general condition was mildly affected by symptoms. He was conscious and cooperative (Glasgow Coma Scale 15), hemodynamically and respiratory stable, with normal spontaneous urination and bowel transit. His vital signs were within normal limits: blood pressure 146/90 mmHg, oxygen saturation 98%, heart rate 90 bpm, and temperature 36.1°C.

Local clinical examination revealed a supple, elastic abdomen located above the xiphoid-pubic plane, which moved symmetrically with respiration. There was a well-healed median surgical scar, normal in conformation and position. Palpation revealed diffuse tenderness on both superficial and deep palpation, while percussion identified alternating areas of dullness and tympany. Auscultation detected present and active bowel sounds. Digital rectal examination showed a rectal ampulla with normal relations and a sphincter with preserved tone.

Laboratory investigations at admission revealed elevated hemoglobin (17.7 g/dL) and hematocrit (52.4%), leukocytosis ($12.15 \times 10^3/\mu\text{L}$), hypercholesterolemia (total cholesterol 256 mg/dL, LDL cholesterol 173 mg/dL), hypertriglyceridemia (269 mg/dL), mild hyperglycemia (120 mg/dL), and markedly elevated fecal occult blood (quantitative hemoglobin >200.0 $\mu\text{g/g}$).

A native CT scan of the thorax, abdomen, and pelvis revealed circumferential wall thickening of the descending colon up to 13 mm over a 75 mm segment, with adjacent fat stranding, several small fluid collections and lymph nodes, and nearly complete luminal narrowing over a 10 mm segment. The ascending and transverse colon were distended, measuring up to 64 mm and 62 mm, respectively, filled with fecal material. An abdominal X-ray demonstrated multiple small-to-medium hydro-aerial levels projected in the mid and lower abdominal quadrants (Figure 1).

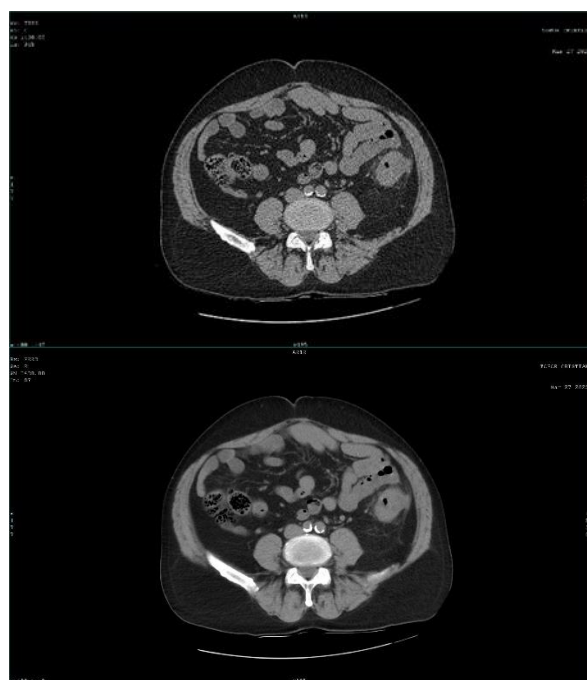


Figure 1 – Preoperative ct scan

After appropriate preoperative preparation, the patient underwent surgery on June 4, 2025. Intraoperative findings confirmed a stenosing metachronous neoplasm of the descending colon with invasion into the sigmoid colon, peritoneum, a jejunal loop, and the anterior capsule of the left kidney. Additionally, a metachronous tumor of the mid-rectum was identified, along with extensive adhesive disease and evidence of previous transverse colon adenocarcinoma surgery (Figure 2 and 3).

The surgical procedure included exploratory laparotomy, adhesiolysis, segmental resection of the jejunum with hand-sewn end-to-end jejuno-jejunal anastomosis, total colectomy with stapled side-to-end ileorectal anastomosis, abdominal lavage and toileting, drainage of the left and right paracolic gutters, double drainage of the sacral excavation, single-layer laparorrhaphy with interrupted sutures, skin closure, and sterile dressing.



Figure 2 – Terminal ileum + colon with penetrating jejunal segment + middle rectum

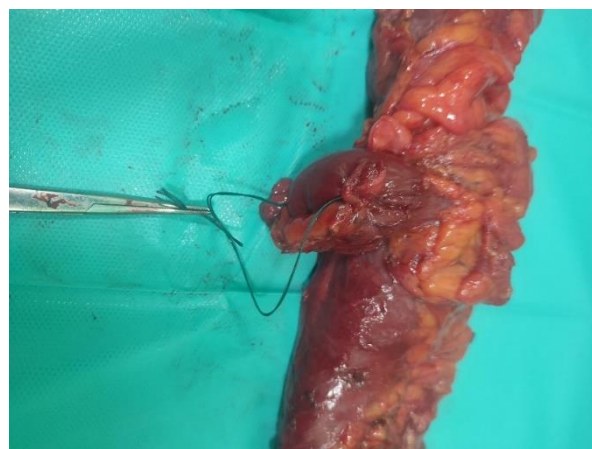


Figure 3 – Terminal ileum + colon with penetrating jejunal segment + middle rectum

The postoperative course was favorable. The patient was discharged 15 days after surgery in good general condition, afebrile, alert, cooperative, with restored bowel transit and spontaneous micturition. All drains were removed prior to discharge.

Histopathological analysis revealed two synchronous colorectal tumors:

A. Low-grade adenocarcinoma of the descending colon with loss of MSH-6 protein expression, infiltrating the colonic wall up to the subserosa, with no metastasis in 34 examined regional lymph nodes, clear resection margins, staged as pT3N0R0.

B. Mucinous adenocarcinoma of the mid-rectum with loss of MSH-6 protein expression, infiltrating up to the muscularis propria without extension beyond it, with no nodal metastasis, staged as pT2N0R0.

Discussions

Metachronous colorectal cancer (CRC) is a recognized risk subsequent to curative treatment for primary colorectal malignancy, characterized by the emergence of a new main tumor at least six months post-initial resection. This case exemplifies an unusually delayed metachronous presentation occurring 22 years after the first operation, confounding existing paradigms of follow-up and surveillance, despite most instances arising during the first 2–5 years postoperatively. Notably, Lazarus and Fazekas reported that up to 67% of metachronous cancers diagnosed in their series occurred more than 11 years after initial surgery, suggesting that extremely delayed cases like ours, though rare, are documented [6].

The documented prevalence of metachronous colorectal cancer (CRC) fluctuates in the literature, often between 0.5% and 9%, with certain studies approximating it at 1.6% to 4.6% [1,2]. In the cohort of 240 patients with a history of colorectal cancer surgery studied by Nava et al., the incidence was 4.6%, and more than 21% acquired benign

yet possibly malignant adenomas [2]. The actual frequency may be underestimated due to misclassification, because of the absence of baseline colonoscopy at the initial surgery could lead to synchronous lesions being incorrectly identified as metachronous [2]. Additionally, Pramateftakis et al. observed that synchronous and metachronous cancers often display mucinous histology and microsatellite instability, findings which align with our patient's final pathology [5]. Cavallaro et al. further emphasized that mucinous histology is more frequent in synchronous tumors and may be associated with distinct molecular pathways, including MSI and KRAS mutations, highlighting the importance of molecular profiling in guiding postoperative surveillance and adjuvant therapy decisions [10].

The patient underwent first surgery in 2003 for a stenosing tumor of the transverse colon, subsequently receiving four cycles of chemotherapy, with no recorded long-term colonoscopic follow-up. This feature is concerning, as colonoscopy is the definitive method for the early detection of metachronous lesions, even in asymptomatic individuals. Rao and Jayaraman highlighted that numerous metachronous cancers are asymptomatic and potentially treatable when detected using surveillance colonoscopy, with an average identification period of 18.85 months postoperatively [1]. Moreover, Parry et al. found that patients with MSH6 mutations have a significant lifetime risk of metachronous CRC and may benefit from more extensive surgeries such as total colectomy to reduce future cancer risk [7].

The 2023 systematic review by Zhang et al. indicates that the presence of synchronous advanced adenomas or synchronous cancers at initial diagnosis markedly elevates the risk of subsequent metachronous colorectal cancer, with risk ratios (RR) of 3.61 and 2.77, respectively [4]. The investigators discovered no significant correlations between lifestyle factors (such as

smoking, alcohol consumption, and BMI) and metachronous colorectal cancer; however, age consistently emerged as a risk factor for advanced metachronous neoplasia (relative risk per annual rise = 1.07) [4]. Liu et al. similarly demonstrated that first-degree relatives of patients with metachronous CRC have an increased risk of colorectal neoplasia themselves, emphasizing the importance of family counseling and possible genetic testing in these contexts [8].

Furthermore, the location of the initial tumor may influence the recurrence pattern: left-sided tumors, as shown in our instance, have a tendency for hepatic recurrence, whereas rectal or perforated cancers may reappear in the lungs [3]. Nonetheless, metachronous disease manifesting decades later, affecting several anatomical sites (descending colon, sigmoid, jejunum, peritoneum, renal capsule), as observed in this instance, is exceptionally unusual and implies either the presence of very slow-growing clones or the impact of secondary mutations over time. Bonadona et al. reported that MSH6 mutation carriers often develop their first cancer later than MLH1 or MSH2 carriers, frequently after age 50, consistent with our patient's presentation [9].

Cohen and Platell's research elucidates the intricacies involved in controlling metachronous colorectal cancer metastases, especially those that emerge years following initial treatment. Research indicates that micrometastatic illness, frequently imperceptible with conventional imaging, is identified in as many as 23% of patients undergoing treatment with curative intent [3]. Although the majority of metachronous metastases manifest within 3 years, there is a growing acknowledgment of intermediate (2–5 years) and late (>5 years) occurrences, particularly in the lungs or peritoneum [3].

From a diagnostic perspective, traditional biomarkers such as carcinoembryonic antigen (CEA) have limited sensitivity in detecting late metachronous

recurrence, especially in asymptomatic patients. The sensitivity for identifying hepatic metastasis is 73%, while for pulmonary metastasis it is 56% [3]. Conversely, circulating tumor DNA (ctDNA) has surfaced as a potential instrument for identifying recurrence many months prior to the appearance of radiologic evidence; nevertheless, its efficacy in surveillance beyond 10 years remains unexamined [3].

Management of metachronous CRC largely mirrors that of primary cancers, depending on resectability and spread. Surgical intervention involved total colectomy with ileorectal anastomosis and jejunal segment excision, highlighting the necessity of assertive surgical care when possible. Literature indicates that survival following curative-intent excision of metachronous lesions—especially if solitary—is analogous to outcomes of original resections, with 5-year survival rates varying from 30% to 60% based on the metastatic site [3].

Surveillance guidelines post-resection aim to detect recurrence and new lesions early. The US Multi-Society Task Force recommends colonoscopy at 3–6 months postoperatively to clear synchronous disease, followed by colonoscopies at 1 year, 3 years, and then every 5 years if results are normal [1]. Unfortunately, adherence to these long-term protocols declines significantly over time, especially in the elderly or comorbid populations.

This case underlines the importance of individualized, risk-adapted surveillance strategies. While current guidelines generally cease surveillance after 10 years for low-risk patients, such prolonged intervals may be insufficient in select cases with high initial tumor burden, family history, or poor documentation of complete initial evaluation.

Conclusions

Metachronous colorectal cancer remains a clinically significant risk even decades after

the initial curative resection. Although the majority of metachronous lesions are diagnosed within the first five years postoperatively, this case demonstrates that very late occurrences—beyond 20 years—are possible and clinically relevant. It highlights the necessity for long-term, perhaps lifelong, surveillance strategies in selected high-risk individuals. Colonoscopy remains the cornerstone for detection, while surgical management continues to offer the best curative potential in isolated or locally invasive cases. As surveillance technologies evolve, future guidelines must consider incorporating risk-adapted protocols and emerging biomarkers such as circulating tumor DNA to improve early detection and outcomes in patients with delayed metachronous CRC.

References

- [1] Rao D, Jayaraman S. Metachronous colorectal malignancies. *Indian J Surg*. 2011 Sep;73(5):368–9.
- [2] Nava HR, Pagana TJ. Postoperative surveillance of colorectal carcinoma. *Cancer*. 1982 Mar 1;49(5):1043–7.
- [3] Cohen R, Platell CF. Metachronous colorectal cancer metastasis: Who, what, when and what to do about it. *J Surg Oncol*. 2024 Jan;129(1):71–77.
- [4] Zhang Y, Karahalios A, Aung YK, Win AK, Boussioutas A, Jenkins MA. Risk factors for metachronous colorectal cancer and advanced neoplasia following primary colorectal cancer: a systematic review and meta-analysis. *BMC Gastroenterol*. 2023;23:421.
- [5] Pramateftakis MG, Hatzigianni P, Kanellos D, et al. Metachronous colorectal cancer. *Tech Coloproctol*. 2010 Apr;14(Suppl 1):63–64.
- [6] Lazarus J, Fazekas SA. Late development of metachronous colorectal cancer. *Ann Surg*. 1985;201(5):619–623.
- [7] Parry S, Win AK, Parry B, et al. Metachronous colorectal cancer risk for mismatch repair gene mutation carriers: the advantage of more extensive colon surgery. *Gut*. 2013 Nov;62(11):1624–30.
- [8] Liu J, et al. Epidemiology and familial risk of synchronous and metachronous colorectal cancers. *Clin Gastroenterol Hepatol*. 2014 Jul;12(7):1126–1131.
- [9] Bonadona V, et al. Cancer risks associated with germline mutations in MLH1, MSH2, and MSH6 genes in Lynch syndrome. *Gut*. 2017 Mar;66(3):464–473.
- [10] Cavallaro G, et al. Synchronous colorectal carcinoma: Clinical, pathological and molecular features. *World J Gastroenterol*. 2014 May;20(17):4282–9.