

Intrahepatic Bile Duct Adenoma Mimetizing Metastatic Liver Disease in a Case of Colorectal Adenocarcinoma: A Case Report

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Abstract: Colorectal cancer (CRC) is a common malignant tumour of the large intestine or rectum, the incidence of which is rising worldwide, particularly in countries undergoing transition. Liver metastases are common in patients with CRC, occurring in up to 50% of cases, with the liver being the most frequent site of spread. Intrahepatic bile duct adenoma, a rare benign tumour, can mimic liver metastases in patients with CRC, and is often confused with adenocarcinomas due to histological and radiological similarities. Correct differentiation is essential to avoid unnecessary aggressive treatments. Based on the above, this project aims to study the case of a patient with a history of colorectal adenocarcinoma and an intrahepatic bile duct adenoma mimicking metastatic liver disease.

Keywords: Colorectal neoplasm; Adenoma; Adenocarcinoma; Case Report.

Citation: Eller AAB, Barg AB, Simas R, Lanzarin LD, Ogata DC. Intrahepatic Bile Duct Adenoma Mimetizing Metastatic Liver Disease in a Case of Colorectal Adenocarcinoma: A Case Report. Brazilian Journal of Case Reports. 2026 Jan-Dec; 06(1):bjcr210.

<https://doi.org/10.52600/2163-583X.bjcr.2026.6.1.bjcr210>

Received: 20 April 2026

Accepted: 30 June 2026

Published: 10 July 2026



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1. Introduction

Colorectal cancer (CRC) is the third most common malignant neoplasm worldwide, with approximately 1,200,000 new cases annually [1]. The increasing incidence of this disease is attributed to population aging, poor diet, smoking, physical inactivity, and obesity [2]. Around half of CRC patients either have or will develop liver metastases during their lifetime, with 20% to 50% of these cases presenting metastatic disease confined to the liver [1]. Intrahepatic bile duct adenomas (BDA) are rare benign liver tumors originating from the epithelium of intrahepatic bile ducts [3]. These lesions are often misdiagnosed as well-differentiated metastatic adenocarcinomas by surgeons and pathologists [4]. This study aims to provide a detailed description of a case of intrahepatic bile duct adenoma mimicking metastatic liver disease in a patient with a history of colorectal adenocarcinoma, contributing valuable insights to the scientific community.

2. Case Report

A 33-year-old female patient was referred to the clinical oncology department following a diagnosis of rectosigmoid adenocarcinoma. She had initially undergone evaluation at another medical facility due to a two-month history of abdominal distension, ribbon-like stool, and episodes of hematochezia. A biopsy of a rectal lesion identified high-grade epithelial dysplasia, classified as Vienna category 4.3. The patient subsequently underwent proximal rectosigmoidectomy, and histopathological analysis confirmed a moderately differentiated adenocarcinoma (G2) in the rectosigmoid region, measuring 4.5 cm in extension, with clear surgical margins and pathological staging of pT3 pN1b pM0. Table 1 presents a detailed chronology summarizing the sequence of events.

Table 1. Chronological timeline of clinical events and imaging findings.

Date	Clinical event
Jun/20	Rectal lesion biopsy demonstrating high-grade epithelial dysplasia (Vienna category 4.3)
Jul/20	Proximal rectosigmoidectomy performed; pathology confirmed moderately differentiated adenocarcinoma (pT3 pN1b cM0)
Aug 2020	XELOX chemotherapy initiated
Sep 2020	Switched to FOLFOX due to dose-limiting toxicity; 10 cycles completed
Nov/21	PET scan without evidence of disease; abdominal MRI revealed a new 4 mm diffusion-positive hepatic nodule not detected on PET
Apr 2022	Chest CT demonstrated enlargement of a pulmonary nodule, suggestive of pulmonary recurrence
Jul/22	Pulmonary metastasectomy performed; pathology confirmed metastatic adenocarcinoma
Aug–Oct 2022	Adjuvant XELOX administered (4 cycles)
Mar/23	Abdominal MRI demonstrated persistent hypervascular hepatic nodule measuring 0.9×0.8 cm
Oct 2023	PET scan without evidence of disease
Nov/23	Abdominal MRI demonstrated enlargement of hepatic lesion to 1.1×1.0 cm;
Feb 2024	Liver biopsy diagnosed atypical bile duct adenoma
Mar/24	Follow-up MRI demonstrated interval enlargement with hemorrhagic content
Jan/25	Follow-up MRI demonstrated progressive decrease in lesion size

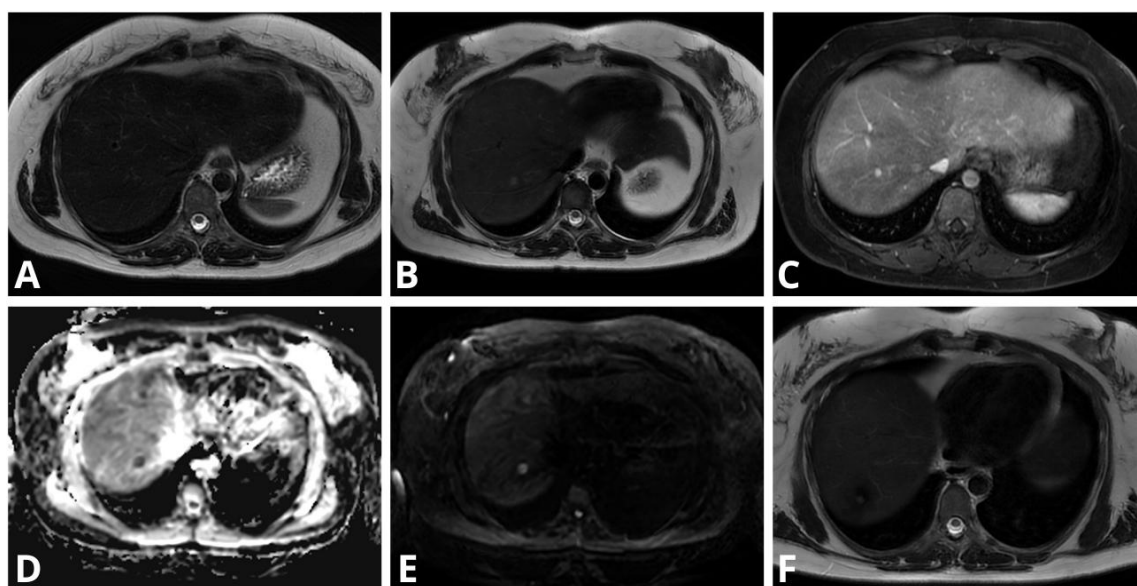
Her medical history is notable for familial dyslipidemia and systemic arterial hypertension. The patient has been using oral hormonal contraception since the age of 17 and has a family history of malignant neoplasms. She denies smoking, alcohol consumption, and a sedentary lifestyle.

The initial management consisted of prescribing the capecitabine plus oxaliplatin (XELOX) regimen. After monthly follow-up, XELOX was discontinued, and the 5-fluorouracil/leucovorin plus oxaliplatin (FOLFOX) regimen were initiated due to dose-limiting toxicity. A total of 10 cycles of FOLFOX were administered without complications. The patient demonstrated favorable clinical progression, with negative CEA, CA19-9, and CA125 markers. At the fourth-month follow-up, abdominal magnetic resonance imaging (MRI) revealed a newly detected, diffusion-positive, solitary hepatic nodule measuring 0.4 cm, of nonspecific nature, which had not been identified on PET scan. Chest computed tomography (CT) showed a new pulmonary nodule on the anterior basilar segment of the right lower lobe.

After five months, an increase in the size of a pulmonary nodule in the right lower lobe was observed. Given the diagnosis of pulmonary recurrence, the patient underwent nodule excision (pulmonary metastasectomy and mediastinal lymphadenectomy of station N7). Histopathological analysis confirmed adenocarcinoma (0.6 cm nodule), with no neoplastic involvement of the pleura. Cytopathological examination of the pleural fluid showed no criteria for malignancy. The management strategy included four cycles of XELOX as adjuvant therapy.

Subsequent imaging 12 months later revealed an increase in size of the hepatic nodule on abdominal magnetic resonance imaging (MRI), demonstrating high T2WI signal, with moderate restricted diffusion and hypervascular enhancing, located on the periphery of the hepatic segment VII, measuring 0.9×0.8 cm (Figure 1).

Figure 1. MRI characterization of hepatic nodule appearance, diffusion restriction, and post-biopsy evolution. A. Previous T2WI MRI with no hepatic nodule on segment VII. B - new hepatic nodule with high intensity signal on T2WI. C. T1WI post contrast showing hypervascular enhancement of the lesion. D and E. Hepatic nodule presenting restricted diffusion on DWI and ADC. F. T2WI demonstrating progressive regression after biopsy, with peripheral cicatricial findings.



Chest computed tomography (CT) and PET scan showed no abnormalities. During follow-up of the hepatic nodule, after eight months, its dimensions increased to 1.1×1.0 cm. Consequently, a liver nodule biopsy was performed, revealing an atypical bile duct adenoma (Figure 2). The biopsied nodule subsequently decreased in size progressively on follow up (Figure 1), and thoracic CT showed no new abnormalities. The progressive reduction in lesion size after biopsy was interpreted as a post-procedural phenomenon, likely related to tissue sampling, focal hemorrhage, stromal collapse and subsequent scar formation, rather than true biological regression.

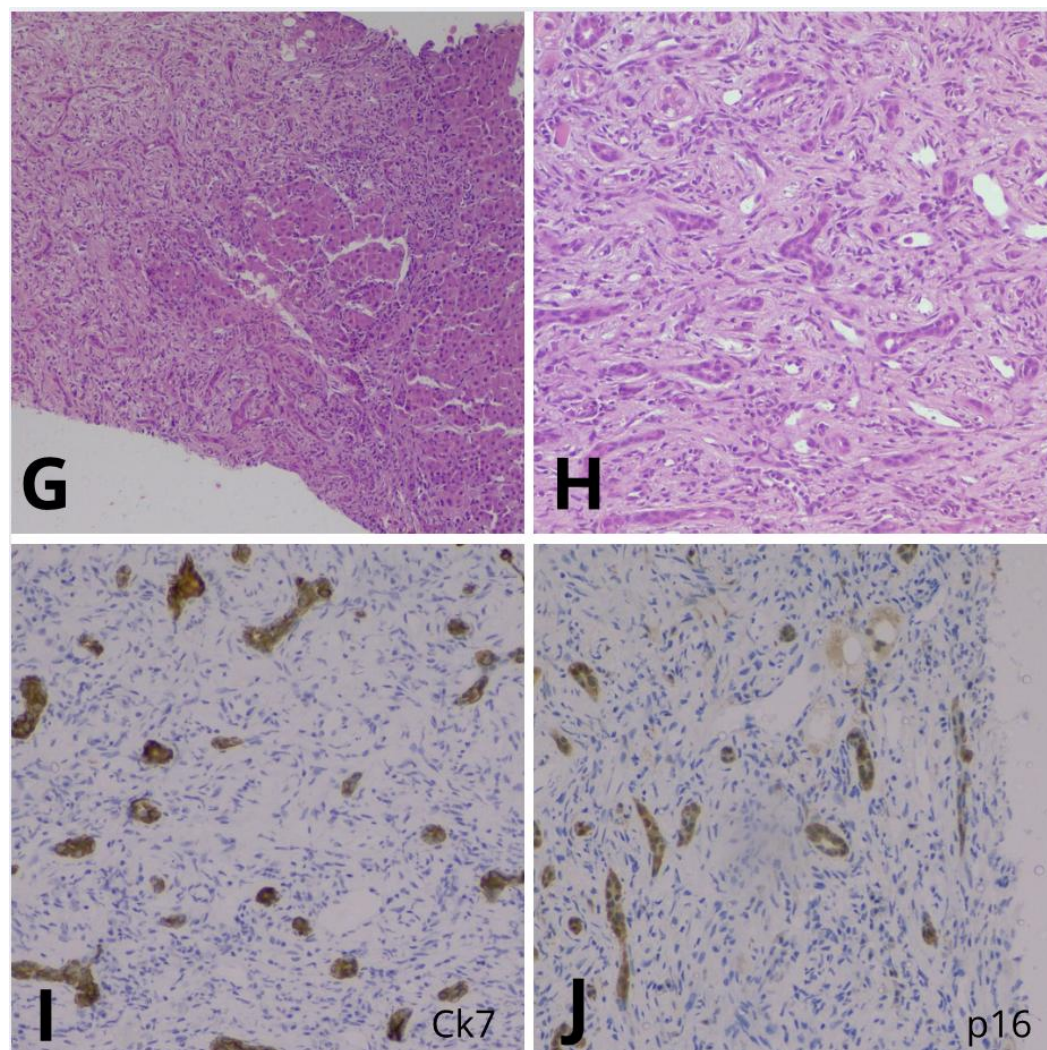
The thorough oncologic history and pathologic findings were available for the abdominal radiologist reading the MRI, which prompted complementary MRI sequences, looking for radiologic pearls suggestive of biliary duct adenoma. These complementary sequences revealed mild peripheral biliary duct dilation (Figure 3), a finding highly associated with biliary duct adenoma, along with the restricted diffusion, avid enhancement and high T2WI signal.

On further MRI follow-up, along with the slow progressive decrease in size after biopsy, the lesion also demonstrated changes in its enhancement pattern. Importantly, it did not exhibit the classic discontinuous nodular peripheral enhancement with complete centripetal fill-in typically observed in cavernous hemangiomas. Instead, a progressive peripheral globular enhancement pattern became more evident during late follow-up examinations, after the lesion had already demonstrated restricted diffusion, interval growth, and a clinical context strongly suggestive of metastatic disease.

If not interpreted in conjunction with the patient's oncologic history and histopathological findings, the combination of high T2 signal intensity and progressive globular enhancement could potentially have led to misclassification as a hemangioma (Figure 3). It

is a powerful example how a comprehensive and thorough clinical history should be always provided for the pathology team, as well as for the radiologists, when asking for complementary evaluations.

Figure 2. Liver biopsy photomicrographs. G (H&E 100x) and H (H&E 200x)- presence of glandular or ductal structures lined by cuboidal epithelium without atypia. I- positive immunostaining for cytokeratin 7. J – positive immunostaining for p16.



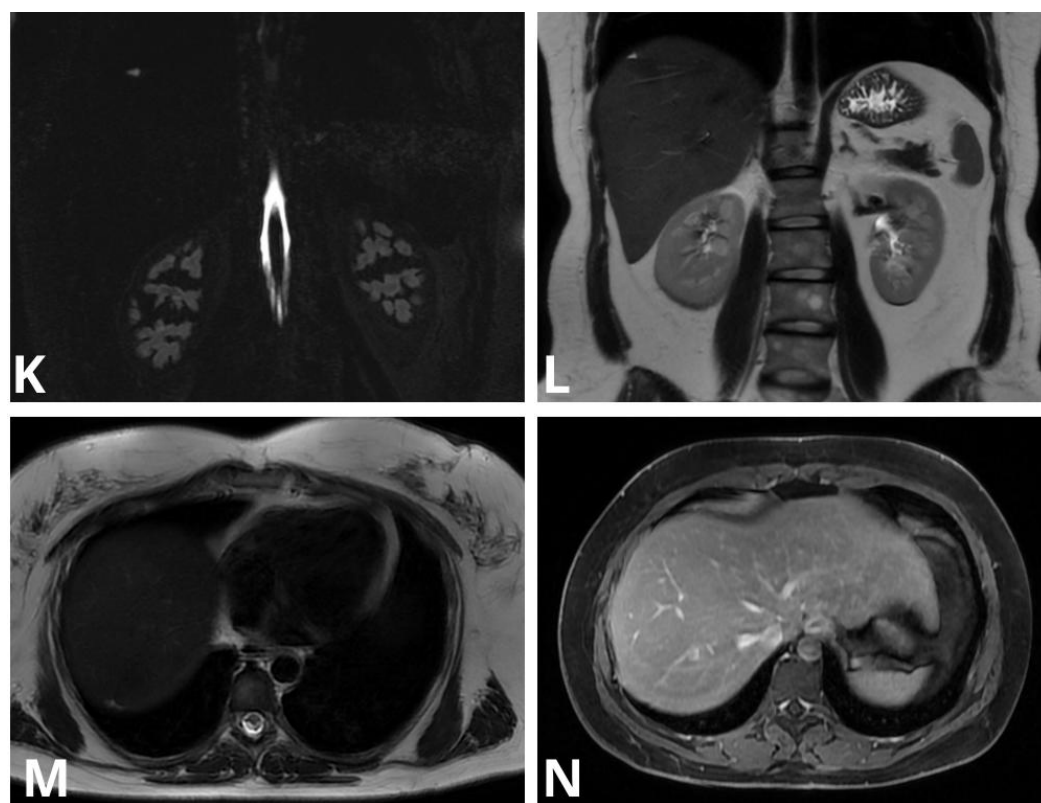
3. Discussion and Conclusion

In Western populations, the average lifetime risk of developing colorectal cancer (CRC) ranges from 3% to 5% [2]. In most cases, the disease remains asymptomatic until it reaches an advanced stage. However, patients may present with rectal bleeding, changes in bowel habits, anemia, or abdominal pain [5]. Colonoscopy is the gold standard for diagnosis [2], and histological analysis remains the cornerstone for pathological staging and subsequent management [5]. Treatment options include local endoscopic and surgical excision, preoperative radiotherapy for downstaging, systemic therapy, extensive surgery for locoregional and metastatic disease, local ablative therapies for metastases, as well as palliative chemotherapy, targeted therapy, and immunotherapy [5]. In addition to endoscopic surveillance following cancer resection, monitoring plasma carcinoembryonic antigen (CEA) levels and performing computed tomography (CT) scans can help detect recurrences [2].

The liver is the most common site of metastases, affected in up to 75% of patients who develop metastatic disease [1]. Hepatic metastases are synchronous in 15% to 25% of

cases [6]. Surgical treatment of colorectal liver metastases (CRLM) remains the only potentially curative option, with five-year survival rates ranging from 25% to 58%. However, only one-quarter of patients present with resectable disease at diagnosis [1].

Figure 3. MRCP and MRI findings demonstrating peripheral biliary duct dilation and hepatic lesion enhancement. K - MRCP showing mild peripheric biliary duct dilation. L and M - coronal T2WI demonstrating mild peripheric biliary dilation. N - T1WI with contrast demonstrating late persistent enhancement.



Although biliary ductal dilatation may be observed in both metastatic colorectal liver lesions and benign biliary tumors, its diagnostic significance depends on the associated imaging findings and growth pattern. In colorectal liver metastases, biliary involvement is typically associated with infiltrative tumor growth along the bile ducts, often accompanied by progressive lesion enlargement and features suggestive of malignancy. In contrast, bile duct adenoma may present with focal peripheral biliary ductal dilatation due to its biliary epithelial origin, without the aggressive radiological characteristics usually seen in metastatic disease. Therefore, isolated biliary dilatation should not be considered a specific indicator of metastasis but rather interpreted in conjunction with the overall radiological and clinical context [6]. The comparative MRI findings are summarized in Table 2.

As shown in Table 2, the imaging findings observed in the present case shared features with both bile duct adenoma and colorectal liver metastasis, illustrating the diagnostic challenge and emphasizing the importance of histopathological confirmation. According to Allaire et al. [3], intrahepatic bile duct adenoma (BDA) consists of benign, non-cystic ducts with varying degrees of inflammation and fibrosis. It can be distinguished from adenocarcinoma by the absence of nuclear hyperchromasia, mitotic activity, and vascular invasion [3]. BDAs are generally discovered incidentally, and surgical resection remains the primary treatment approach. The differential diagnosis includes cholangiocarcinoma, metastatic adenocarcinoma, biliary adenofibroma, Von Meyenberg complex, and primary sclerosing cholangitis [4].

Table 2. MRI features of the present case compared with BDA and colorectal liver metastasis.

MRI Feature	Present Case	BDA	Colorectal Liver Metastasis
T2 hyperintensity	Yes	Common	Variable
Restricted diffusion	Yes	Reported	Common
Hypervascular enhancement	Yes	Common	Variable
Peripheral biliary dilatation	Yes	Characteristic	Uncommon
Progressive growth	Yes	Possible	Common
Post-biopsy regression	Yes	Possible	Unusual

Clinically and macroscopically, BDA usually presents as a single lesion, most often subcapsular, flattened, well-circumscribed but not encapsulated, ranging from 1 to 20 mm in size. Histologically, it is composed of uniformly shaped and spaced tubules, branching and densely packed, without evident atypia or mitotic figures. The nuclei of the cells are lighter than those observed in normal bile ducts. From an immunohistochemical standpoint, lesions composed of biliary cells are positive for cytokeratins 7, 8, 18, and 19. Although p16 positivity supported a biliary epithelial lesion, this marker alone does not exclude metastatic colorectal adenocarcinoma. Additional markers such as CDX2, SATB2 and SMAD4 would have further strengthened the differential diagnosis [7].

Despite their benign nature, BDAs can closely mimic malignant lesions on imaging studies. They are often located near the liver capsule and are usually small and solitary. Their imaging features can closely resemble those of malignant lesions, such as cholangiocarcinoma or metastatic disease, making definitive diagnosis challenging based solely on imaging. Histopathological examination remains the gold standard for diagnosis [8]. On Magnetic Resonance Imaging (MRI), BDAs are typically T2WI hyperintense, exhibiting DWI restricted diffusion and moderate contrast enhancement. A superficial frond-like appearance and bile duct dilatation at or upstream of the lesion may be characteristic features [9]. On Computed Tomography (CT): On non-contrast CT, BDAs generally appear as hypo- or isoattenuating lesions relative to the surrounding liver parenchyma. Following contrast administration, they often demonstrate hypervascularity in the arterial phase with prolonged enhancement in the delayed phase, reflecting their rich blood supply [10].

This case highlights an important diagnostic pitfall in patients with colorectal adenocarcinoma presenting with hepatic nodules. Although metastatic disease is the leading clinical suspicion in this setting, benign lesions such as bile duct adenoma may closely mimic metastasis on imaging studies. Furthermore, this case highlights the diagnostic challenge of distinguishing between bile duct adenoma and metastatic colorectal disease in patients with known systemic spread. Integrating detailed MR imaging (including MRCP features) with immunohistochemical profiling (p16, CK7) is essential to avoid misdiagnosis and unnecessary treatment.

The current study presents an important limitation: the lack of an immunohistochemistry panel analysis. Accurate histopathological evaluation, including expression of some specific marker, such as CDX2 and SMAD4, remains essential to establish the correct diagnosis and to avoid potential overtreatment. Awareness of this entity is particularly relevant for both clinicians and pathologists, as it directly impacts staging, prognosis, and therapeutic decision-making. Correlation with clinical, radiological, and pathological findings is strongly recommended to ensure appropriate patient management.

Funding: None.

Research Ethics Committee Approval: In accordance with the Brazilian laws, the current study has been approved by the Ethics Committee on December 17th, 2024, under the protocol number 7.303.910.

Acknowledgments: None.

Conflicts of Interest: All other authors declare no conflicts of interest.

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