

Obstructive and Perforated Primary Colonic Squamous Cell Carcinoma of the Hepatic Flexure with Direct Extension into the Gallbladder: A Case Report

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Abstract

Primary squamous cell carcinoma of the colon is exceedingly rare. We present the case of an 83-year-old man who arrived at the Emergency Department of Laredo Medical Center complaining of abdominal pain. He was found to have signs of bowel obstruction and imaging studies revealed the presence of a 7 cm intraabdominal mass. Colonoscopy confirmed there was an obstructive colonic lesion in the hepatic flexure. Intraoperatively, it was found to have perforated the colon, forming an intraabdominal mass that directly extended into the gallbladder. Histopathologic examination confirmed a malignant neoplasm with morphology and immunophenotype diagnostic for poorly differentiated squamous cell carcinoma. An extensive workup failed to demonstrate squamous cell carcinoma in any other anatomic sites; therefore, it was concluded that it was not metastatic and, corresponded to a primary squamous cell carcinoma of the colon. Given the patient's age and comorbidities, he experienced a rapid decline in health and expired two days after surgery. We present this case to add to the existing literature and discuss the clinical characteristics, significance and pathogenesis, as well as emerging therapeutic strategies for this rare and aggressive form of colorectal cancer.

Keywords

Colorectal Neoplasms, Colonic Squamous Cell Carcinoma, Bowel Obstruction

1. Introduction

Colorectal cancer (CRC) continues to be a significant cause of morbidity and mortality in both men and women [1]. The vast majority of CRCs are adenocarcinomas with squamous cell carcinoma representing less than 1% of cases and having an incidence of 0.1 to 0.25 per 1000 diagnosed cases [2]. In fact, in analyzing several comprehensive reviews of primary colorectal squamous cell carcinoma (PCRSCC) [2]-[4], it has been determined that, as of 2026, only 100 cases have been published since it was first described in 1919 by Schmidtman *et al.* [5]. Clinical presentation of PCRSCC is similar to that of adenocarcinoma with the most frequently affected sites being the rectum, followed by the right colon, with a mean age at diagnosis of 56.9 years and with a nearly even gender distribution, given only a slight predilection for females [2]-[4]. PCRSCC has an aggressive clinical course with most cases already in late stages at initial diagnosis [6]. Therefore, early detection is essential, especially since node-negative cases detected still in Stages I and II showed a similar prognosis to adenocarcinoma [2]. The diagnosis also requires ruling out a colonic metastasis from another anatomic site, as per the criteria of Williams *et al.* [7]; this includes, but is not limited to, spread from the esophagus, anal canal, urinary bladder, uterus or squamous-lined fistulous tract [8]. The pathogenesis of PCRSCC is poorly understood with theories including squamous metaplasia of the colonic mucosa, squamous differentiation from a preexisting adenocarcinoma and development from pluripotential stem cells [9].

2. Case Presentation

The case is that of an 83-year-old man with a complicated medical history including type II diabetes mellitus, hypertension, coronary artery disease (CAD), chronic obstructive pulmonary disease (COPD), anemia since 2010, and adenocarcinoma of the prostate for which he underwent total prostatectomy more than 10 years before his present illness, which began in February of 2025. It was characterized by general malaise, nausea, vomiting, loss of appetite, and intermittent, cramping pain in both lower abdominal quadrants. His primary care physician performed a fecal immunochemical test for occult blood in stool, which was positive. However, in March of 2025, his symptoms worsened before further workup could be performed, so he went to the ER at Laredo Medical Center, at which time a CT scan of the abdomen and pelvis without contrast was performed, showing sigmoid diverticulosis (**Figure 1**) and an upper GI endoscopy showed Barrett's esophagus but was negative for malignancy. By late-April, he was referred for a colonoscopy, which revealed a large infiltrative, bleeding, circumferential mass of the hepatic flexure (**Figure 2**). The rest of the colon, rectum and anus were unremarkable. Biopsies were taken of the colonic mass and he was discharged home pending histopathologic results; these were diagnostic for a malignant neoplasm composed of large, variably-sized cells with eosinophilic cytoplasm and pleomorphic nuclei; some of the cells exhibited individual keratinization and immunohistochemical stains showed strong positivity for high molecular weight cytokeratins p63 and CKbetaE12,

patchy, weak staining for CK7 and negative staining for villin, CK20, CDX2, uroplakin II and neuroendocrine markers synaptophysin and chromogranin-A (**Figure 3**). The morphology and immunophenotype were, therefore, considered consistent

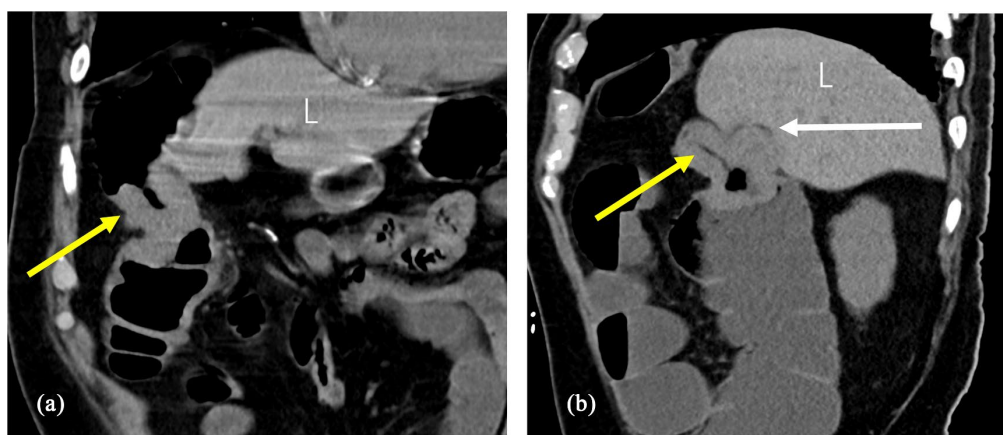


Figure 1. (a) Coronal CT noncontrast image showing a 4 cm. mass within the ascending colon about the hepatic flexure (yellow arrow). Liver (L). (b) Sagittal CT noncontrast image of the mass in the hepatic flexure (yellow arrow) invading the adjacent collapsed gallbladder (white arrow).

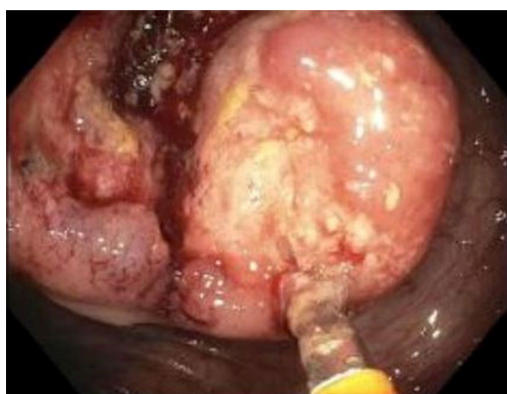


Figure 2. Colonoscopy showed an infiltrative, circumferential, obstructive large mass at the hepatic flexure.



Figure 3. An extended right hemicolectomy showing a multinodular, circumferential mass affecting a 7 cm segment of bowel at the hepatic flexure.

with poorly differentiated squamous cell carcinoma (SCC). Given the rarity of this finding, further workup that included a repeat upper GI endoscopy and CT of the thorax was performed; these studies did not reveal tumor in any other possible anatomical site. In addition, serum markers CEA, CA-125 and CA19-9 were negative. Therefore, it was deemed to be a primary colorectal squamous cell carcinoma (PCRSCC).

His condition worsened significantly, now presenting hypotension, dyspnea on exertion and dehydration, so he was readmitted on May 3, 2025. Laboratory tests revealed acute kidney injury with BUN of 35 mg/dL, creatinine of 2.3 mg/dL, hyperglycemia of 212 mg/dL and lactic acidosis. A repeat CT scan of the abdomen and pelvis now showed a large obstructive mass appearing to extend into the peritoneal cavity with involvement of the gallbladder. Given the degree of bowel obstruction, on May 5, 2025, he underwent a right hemicolectomy that was extended to include a portion of the transverse colon and cholecystectomy. Grossly, the mass was circumferential, affecting a 5.7 cm segment of bowel with invasion of the entire thickness of the bowel wall, perforation of peritoneum and direct invasion of the gallbladder (**Figure 4**). Microscopically, it exhibited features of squamous cell

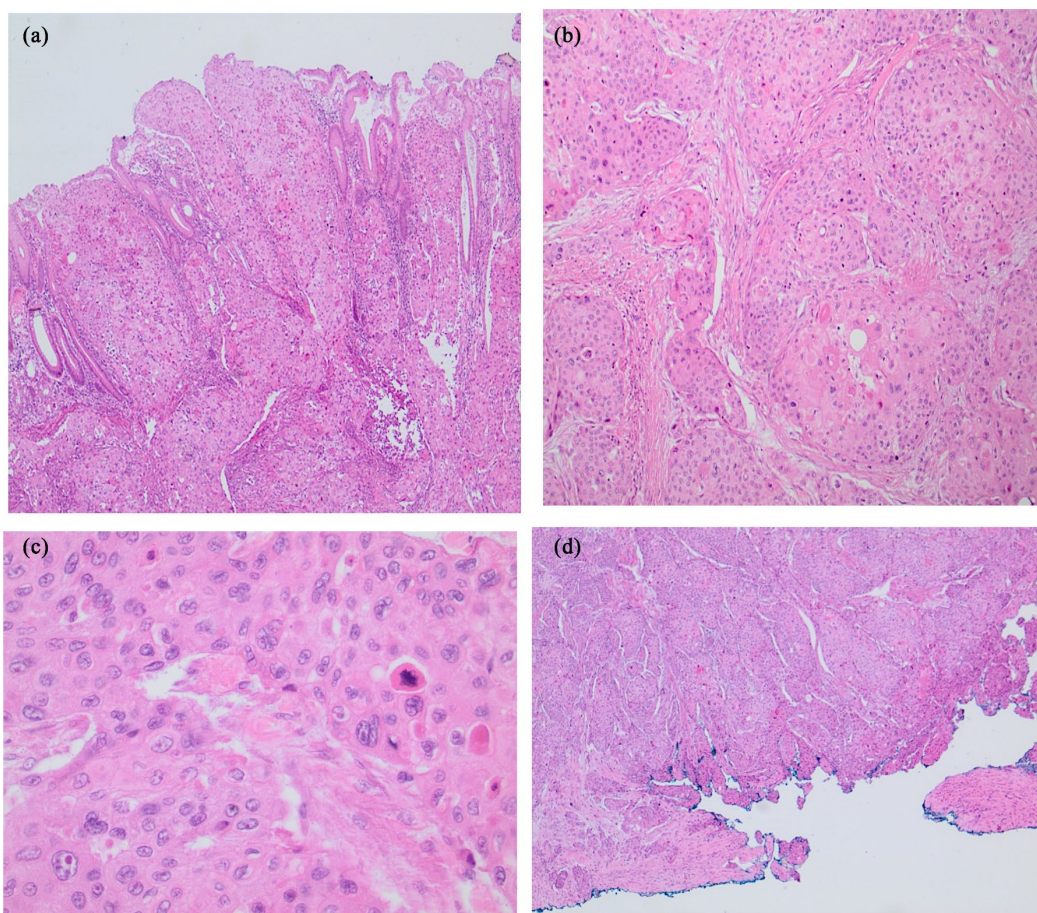


Figure 4. (a) Squamous cell carcinoma (SCC) in the colonic mucosa, possibly *in situ* (H&E, 100×); (b) Invasive SCC in muscularis propria (H&E, 200×); (c) Invasive SCC with individual keratinization and mitotic activity (400×); (d) Invasive SCC with positive radial margin (200×).

carcinoma as described on the biopsy; extensive sampling did not reveal morphology of adenocarcinoma anywhere in the tumor and no adenomatous precursor lesion was identified (**Figure 5**). Mucosal margins were negative, but given the large adherent mass it formed with the gallbladder the radial margin was considered positive. A total of 13 of the lymph nodes were examined and were negative for metastasis. Pathologic staging was determined to be pT4b, pN0, pM0.

He was referred to the Oncology Department, however, no further treatment was possible because his condition continued to deteriorate due to cardiac complications and the second postoperative day, he presented cardiac arrest from which resuscitation was not possible and the patient expired.

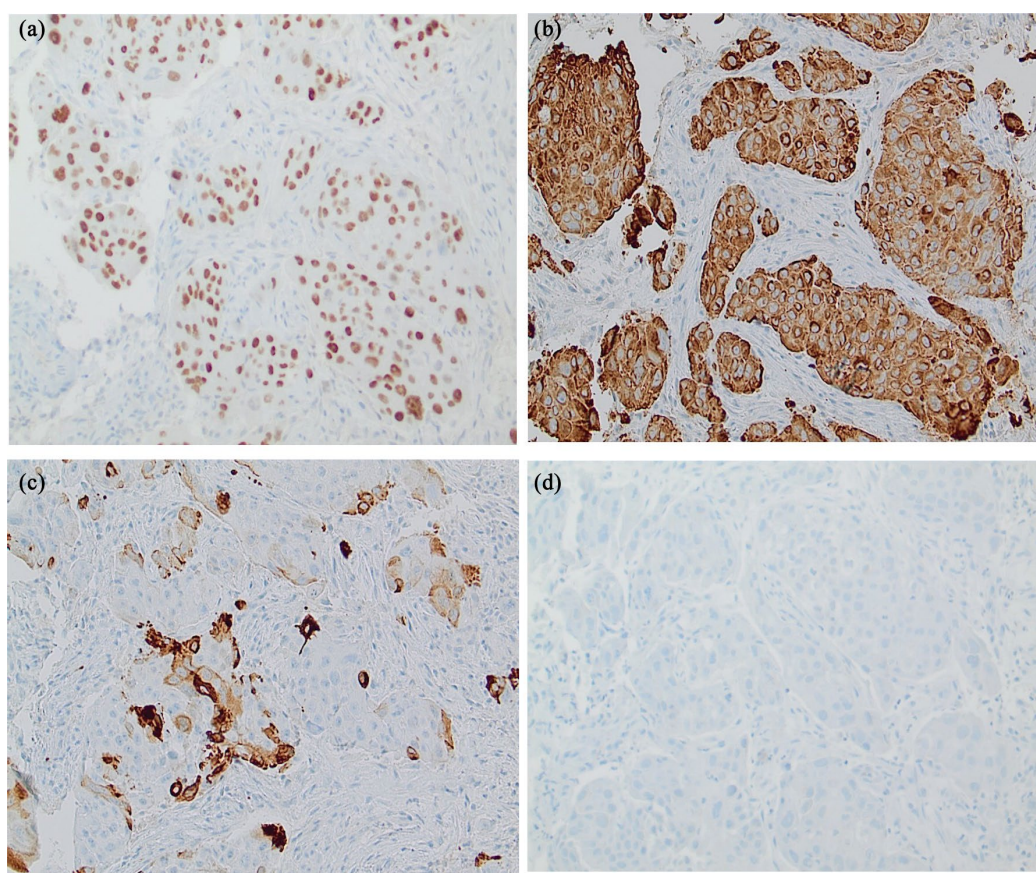


Figure 5. (a) Positive nuclear staining of tumor cells for p63 by immunohistochemistry (IHC) (200×); (b) Positive cytoplasmic staining of tumor cells for CK34/βE12 by IHC (200×); (c) Focal positive IHC staining of tumor cells for CK7 (200×); (d) Tumor cells negative for CDX2 by IHC; they were also negative for p16, GATA-c, villin and TTF-1.

3. Discussion

This case illustrates the aggressive nature of PCRSSC since it went virtually undetected by imaging until within a period of one month, it had grown significantly, creating a clinical picture of bowel obstruction, perforation and an intraabdominal mass that involved the gallbladder. This behavior is in keeping with what has been published either as single case reports or in series of cases. To date, a total of 100

cases have been published, according to several large systematic reviews of the English-language medical literature with the majority (61.6%) arising in the rectum, followed by the right colon (17.2%), left colon (10.1%) and sigmoid/rectosigmoid junction (8.1%) [2]-[4].

Several mechanisms have been considered regarding the physiopathology of squamous differentiation in colorectal malignancies, which usually is focal, but can be extensive to the point where the histologic appearance becomes one of a neoplasm that is entirely squamous in nature. One proposed mechanism is the presence of pluripotential stem cells of endodermal origin that undergo squamous differentiation with ultimate malignant transformation; indeed, two cases reported by Palvio *et al.* in 1985 were called “stem cell carcinoma” because the tumors were composed mainly of undifferentiated cells, focally merging with areas showing glandular and squamous differentiation, as well as neuroendocrine differentiation by histochemical staining and electron microscopy [9]. Another theory mentions proliferation of basal cells following significant mucosal injury from irritation, chronic inflammatory bowel disease (IBD), radiation exposure, infection and even exposure to asbestos [10]. One publication implicates human papillomavirus (HPV) infection [11]. Along this same line, Matsuda *et al.* found that HPV in combination with HIV-induced inactivation of retinoblastoma (RB) tumor suppressor gene, led to the development of squamous differentiation [12]. However, probably what most lends support to squamous metaplasia as a mechanism are reports of its focal occurrence in colonic adenomas, with some already showing high-grade dysplasia in both the glandular and squamous component [13]-[16].

In fact, in some of the cases presented by Comer *et al.* [17], the tumors were referred to as adenocanthoma, an older descriptive term (not found in the WHO 2026 classification of digestive tumors) which refers to adenomas with either low-grade or high-grade squamous differentiation. Comer *et al.* also include in their series a case with two separate SCCs and a third tumor corresponding to a synchronous adenocarcinoma [17]. Furthermore, primary mixed adenosquamous carcinoma of the colon is also a well-described and equally rare variant of colorectal carcinoma [18] [19]. Also, of great interest is the report by Linardoutsos *et al.* of a case of PCRSCC with lymph node metastases showing morphology entirely of adenocarcinoma [3].

We believe that the present case is of importance because of the unusual presentation in the hepatic flexure, of which there are only seven previous cases reported for this anatomic site [6] [10] [17] [20]-[23]. The case reported by Guo and Huang, similar to our case, exhibited rapid growth and perforation, but is different in that it extended into the retroperitoneum to involve the right renal fascia [23].

In our case, as mentioned, there was rapid growth with peritoneal perforation and formation of a large, right upper quadrant, intraabdominal mass with extension through the wall of the gallbladder, ultimately reaching the luminal surface.

This led us to consider whether this neoplasm was actually a primary squamous cell carcinoma of the gallbladder that extended into the colon. With this in mind, and despite a normal appearing gallbladder on the first abdominal CT, a meticulous examination of the entire gallbladder was performed and it failed to reveal an *in-situ* squamous component, whereas we were able to identify an area within the colonic mucosa that could be considered an *in-situ* SCC lesion. This is in keeping with the extensive review by Ayabe *et al.* of 1084 cases of SCC of the gallbladder, where there is no mention of this aggressive form of gallbladder malignancy extending into the colon in any of their cases [24].

Finally, there should be mention of the management of PCRSCC, which up until now has been based on surgical resection. In an interesting case report from Korea, a colonic squamous cell carcinoma presented as a 1 cm sessile polyp; endoscopic resection achieved lack of progression and survival of the patient in this case [25]. This seems to indicate that survival from this aggressive form of colorectal cancer may be dependent on very early detection. Even with adjuvant chemotherapy, outcomes have varied. The case presented by Nassar *et al.* involved a colectomy with negative margins and resection of liver metastases, followed by chemotherapy, which failed to prevent rapid progression of the disease [26]. Whereas a report by Oza *et al.* describes a 72-year-old woman with squamous cell carcinoma of the sigmoid colon with locally advanced pT4 disease who remained disease-free more than 12 years following left hemicolectomy and adjuvant modified FOLFOX-6 chemotherapy [4]. Similarly, two cases by Juturi *et al.* were also responsive to chemotherapy [27]. Wang *et al.* recently reported a case of PCRSCC in a postpartum patient; the tumor exhibited mutations in *BRAF*^{V600E} and *TP53*, as well as microsatellite stability. She was treated with radical resection and adjuvant chemotherapy including oxaliplatin, irinotecan, calcium, folinate and fluorouracil. She had a good initial response but eight months later, a metachronous lesion was detected [28]. This is interesting and highlights the data regarding *BRAF* mutation. Barras *et al.* have established that approximately 30% *BRAF*-mutated colorectal cancer (mCRC) occur with deficient MMR/microsatellite instability (high MSI), primarily due to CpG island methylation and MLH-1 methylation. Furthermore, 70% of *BRAF*-mutated tumors display a CMS-1 transcriptome signature characterized by inflammation and immune infiltration, implying that *BRAF*-mutated tumors may benefit from immune checkpoint inhibitors [29]. This is nicely illustrated by the case published by Liu *et al.* of a large, obstructive mass of the ascending colon with liver metastases that was treated successfully with immunotherapy. The tumor was mismatch repair proficient and microsatellite stable (pMMR/MSS) but exhibited high levels of PD-L1 expression, as well as a missense mutation in codon 600 of the *BRAF* oncogene. PD-L1 blockade in combination with chemotherapy resulted in an effective therapy with a disease-free survival at three years of follow-up [30]. These cases seem to indicate that determination of mismatch repair (MMR) proteins and PD-L1 expression, either by molecular testing or immunohistochemistry, is of essence in PCRSCC, as in all

forms of colonic malignancy, and further molecular testing of a diversity of genetic alterations in search of molecular targets also seems indicated. Astaras *et al.* conducted an in-depth genomic analysis in ten examples of rectal SCC and detected a similar mutational gene map and copy number profile in all of them. However, the frequently gained or lost genes observed in rectal adenocarcinoma were not detected in any of their samples [31].

The role for adjuvant radiotherapy is less defined; while some locally advanced and/or node-positive cases of PCRSCC have been treated with chemoradiation with limited success [32] [33]. Routine use for radiation on completely resected, node-negative cases, as stated by Oza *et al.*, is “not supported by current case-based evidence and is generally reserved for margin-threatened disease or pelvic fixation” [4].

4. Summary

We present the case of a rare, primary colorectal squamous cell carcinoma (PCRSCC) occurring in an 83-year-old man. He had rapidly evolved in four months from onset of symptoms to an advanced disease that had perforated the colon at the level of the hepatic flexure, forming a large intraabdominal mass that involved the gallbladder. Given his many comorbidities and extent of the tumor, the patient expired on the second postoperative day. This case illustrates the aggressive nature of this exceedingly rare neoplasm. Treatment is stage-dependent and usually involves surgical resection but cumulative data based on case reports (of which there have only been 100 since it was first described in 1919) and descriptive synthesis in the form of literature searches have brought to light additional treatment options for combining chemotherapy and/or immunotherapy with promising results.

Disclosure Statement

The authors declare they have approval from Laredo Medical Center for publication of images and clinic details.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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