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Case report

Primary squamous cell carcinoma of the colon: A rare case report

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ABSTRACT

Introduction and importance: Squamous cell carcinoma (SCC) of the colon is an extremely rare pathologic entity, accounting for less than 1 % of all colorectal cancer cases. They tend to be very aggressive with poor outcomes and treatment strategies are still controversial due to the paucity of data available to guide management.

Case presentation: A case of a cecal mass with metastatic liver lesions. Initially diagnosed as an adenocarcinoma, the patient underwent resection with metastasectomy. Despite achieving negative surgical margins and undergoing adjuvant chemotherapy, the patient relapsed and presented with a new mass in the descending colon. The patient underwent resection with subsequent follow-up revealing distant metastasis. The patient passed away soon after.

Clinical discussion: Primary colorectal SCC has similar presentation to adenocarcinoma of the colon. Unfortunately, it usually presents at a late stage. Diagnosis of colorectal SCC requires histologic confirmation of SCC plus exclusion of possible causes. Management is predominantly definitive radical resection followed by adjuvant chemotherapy and radiotherapy. Surgical margins should be at least 5 cm, preferably 10 cm. Lymph node yield greater than 20 was associated with improved survival. Studies assessing the prognosis of primary colorectal SCC following chemo-radiotherapy have not been done.

Conclusion: Surgery remains the most vital important step in the management of colonic SCC. The role of chemotherapy and/or radiation remains questionable. Depending on the aggressiveness of this disease the need for further frequent.

1. Introduction

Squamous cell carcinoma (SCC) of the colon is a rare type of colorectal cancer (CRC), with less than 0.5 % of all CRC [1] with the majority being adenocarcinoma (90 %) [2]. We are presenting a rare case of primary SCC in the ascending colon subsequently recurring in the descending colon. This case report has been reported in line with the SCARE Criteria [3].

2. Case presentation

A 53-year-old male patient, presented to our institution with blood per rectum of 2 months duration. Patient underwent investigations (Figs. 1 & 2) which revealed a cecal mass with multiple liver lesions in the liver. Colonoscopy revealed a cecal mass. Biopsy of this mass was positive for poorly differentiated adenocarcinoma. He underwent right hemicolectomy with liver resection of the metastatic lesions. Pathology revealed invasive poorly differentiated colonic adenocarcinoma with negative surgical margins in both colon and liver lesions (pT4 N1 (3/37) M1), for which he was given adjuvant chemotherapy (capecitabine plus oxaliplatin - CAPOX). A few months later, he presented with new-onset lower abdominal pain. Computed Tomography (CT) scan of the chest, abdomen and pelvis revealed an irregular circumferential segmental wall thickening of the descending colon up to 3.5 cm mass with pericolonic fat stranding (Fig. 3) with no new metastases in the chest, abdomen, or pelvis. Colonoscopy confirmed the presence of the mass in the descending colon, biopsies were taken, showed poorly differentiated adenocarcinoma. He underwent surgical resection for that lesion, pathology revealed invasive poorly differentiated squamous cell carcinoma, pathologically staged as pT4a (4 cm) N0 (0/26) M0 with a

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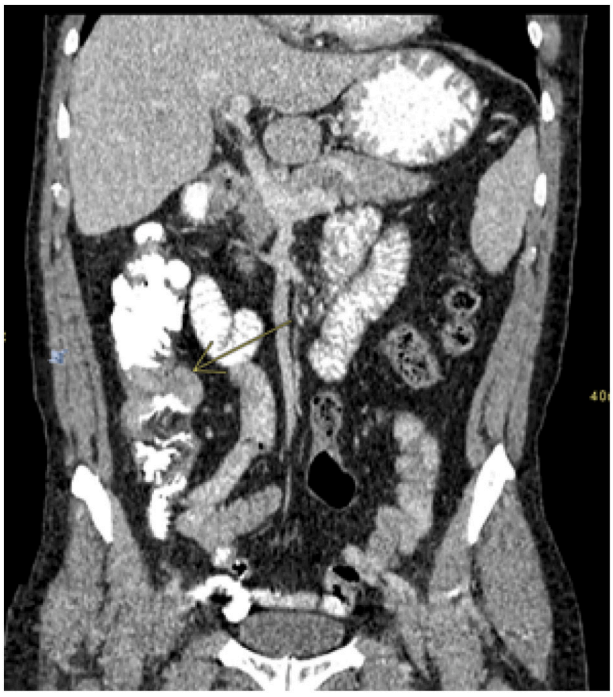


Fig. 1. Coronal CT scan post contrast revealing an ascending colon mass (yellow arrow).

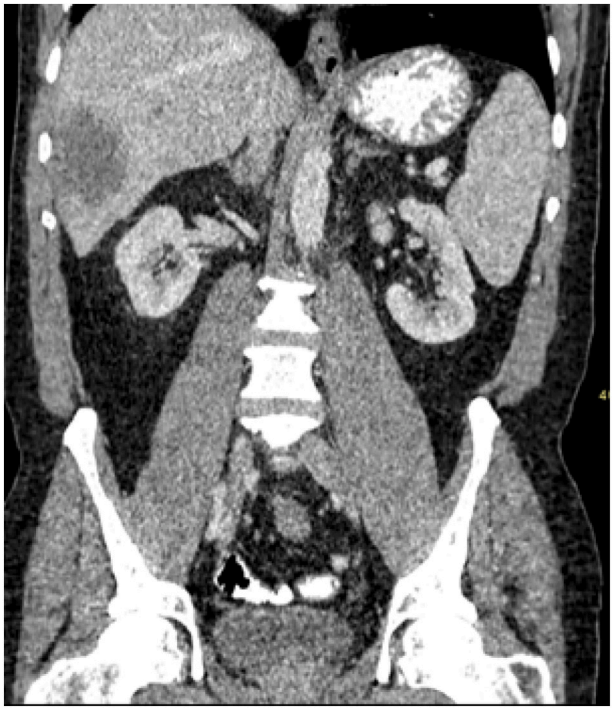


Fig. 2. Coronal CT scan of the abdomen and pelvis showing a mass in the liver, suggestive of metastases.

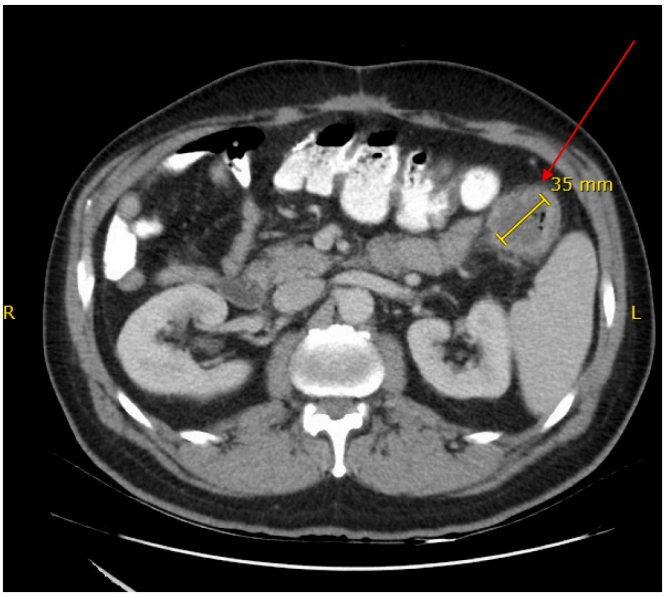


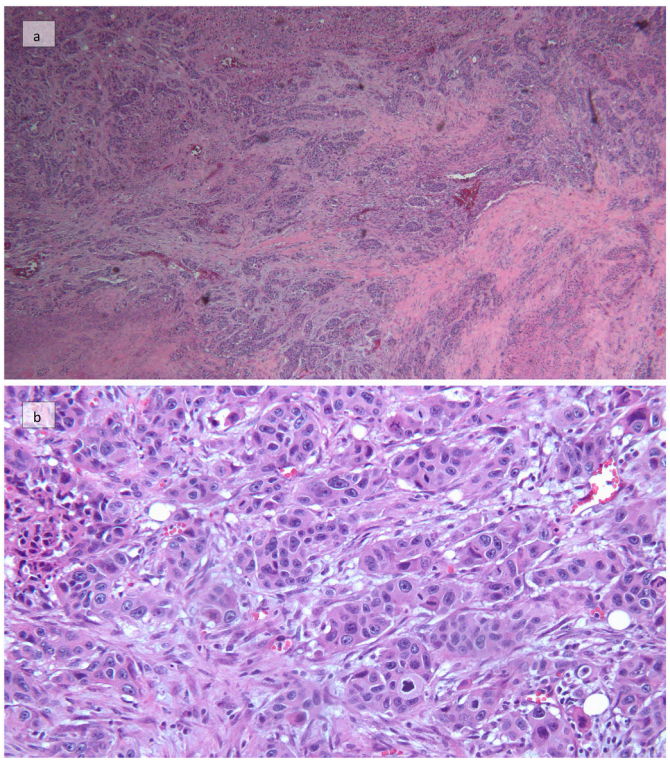
Fig. 3. Axial CT scan of the abdomen and pelvis showing the descending colonic mass with near complete obstruction of the inner lumen (red arrow).

positive P-40 and CK5/6. Re-evaluation of the initial ascending colon tumor was also reviewed, and was found to be adeno-squamous carcinoma with a predominant squamous component and a focal glandular component including the same exact pathology for the hepatectomy specimen., patient received adjuvant chemotherapy with carboplatin plus gemcitabine. Follow up PET-CT imaging showed metastatic disease to the right femur, sternum, lungs, kidneys, and brain. He underwent palliative radiotherapy for his right hip for debilitating symptoms. Also, the patient had radiotherapy for his brain lesion. He was started on immunotherapy with pembrolizumab until the patient passed away 10 months later due to disease progression.

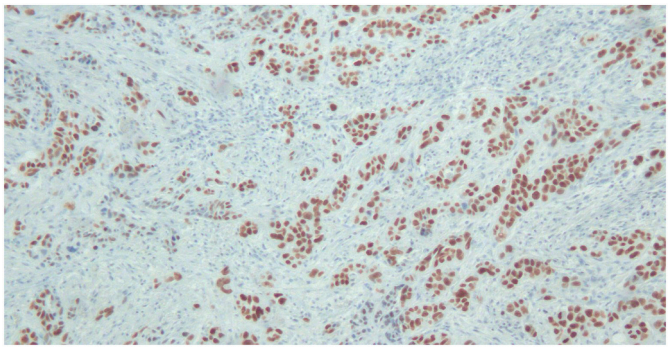
3. Microscopic description of the left (descending) colon mass

The histologic findings in the current tumor and the previously excised colonic tumor were unusual. Histologic section shows a diffuse proliferation of malignant cells forming nests. The tumor cells infiltrated the colonic wall. The tumor is keratinizing with high mitotic rate consistent with a high-grade squamous cell carcinoma; more so, an invasive poorly differentiated squamous cell carcinoma. There is no adenocarcinoma component identified. The immunohistological markers support the histologic findings as the P 40 and CK 5/6 were positive. There was focal positivity for CDX2. The current tumor is morphologically similar to the patient's previous colonic tumor, however, in the previous tumor there was a focal glandular component with a predominance of squamous cell carcinoma (Adeno-squamous carcinoma).

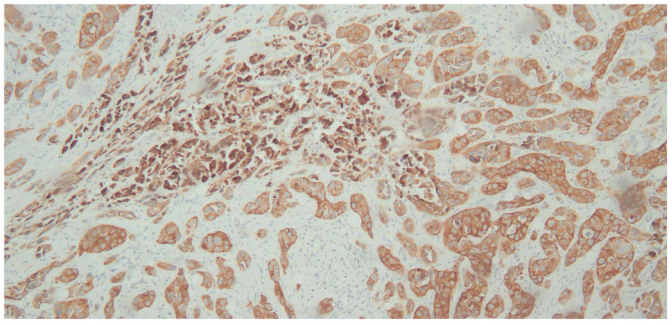
Specimen: Left Hemicolectomy, left (descending) colon.
Tumor: Squamous cell carcinoma, poorly differentiated (G3), greatest dimensions being 5.5 cm, Tumor invading the visceral peritoneum, with positive perineural invasion, absence of lymphovascular invasion. Negative surgical margins
Lymph nodes: 26 negative lymph nodes
Pathologic stage classification (TNM, AJCC 8th Edition): T4a, N0.



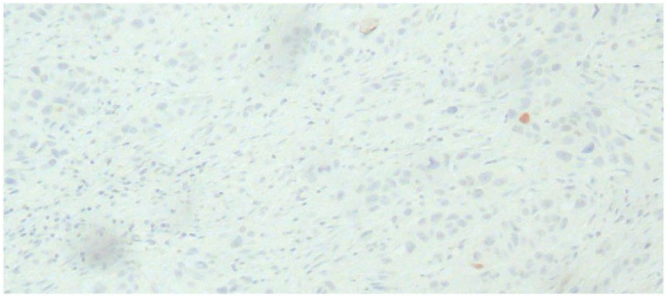
Slide 1. Descending colon mass with hematoxylin and eosin (H&E) staining. (a) Low power view showing invasive cords and nests of poorly differentiated squamous cell carcinoma associated with a desmoplastic stroma and presence of tumor necrosis (5×). (b) High power view showing tumor cells with atypical vesicular nuclei, conspicuous nucleoli, and prominent mitotic activity (20×).



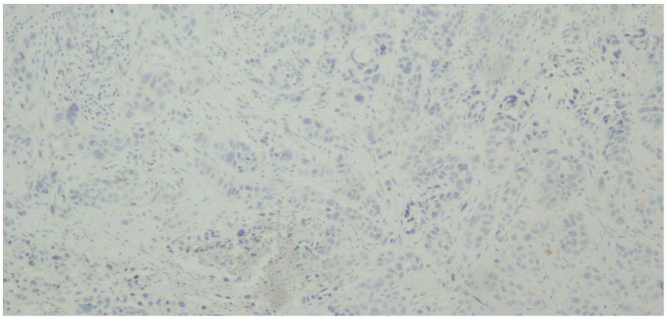
Slide 2. P 40 immunohistochemical stain showing nuclear positivity of the tumor cells (10×) – Inset: P40 positive control.



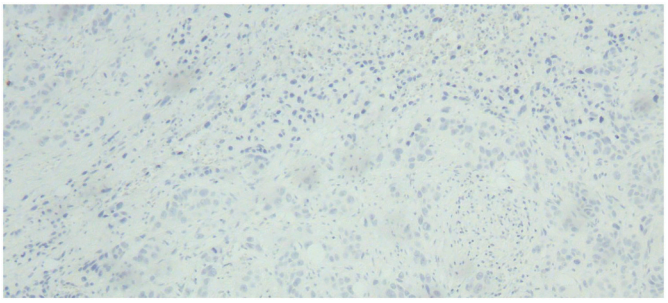
Slide 3. CK 5/6 immunohistochemical stain showing cytoplasmic positivity of the tumor cells (10×) – Inset: CK 5/6 positive control.



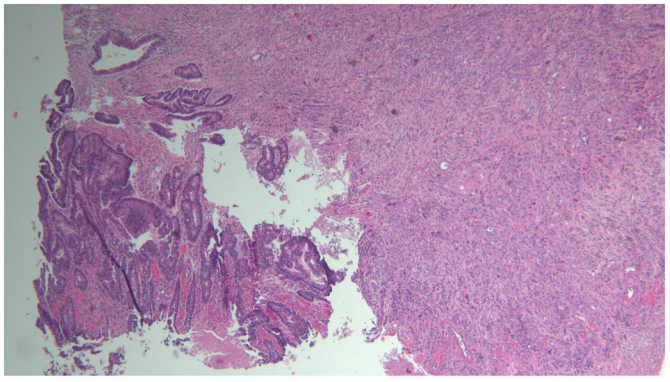
Slide 4. CDX2 immunohistochemical stain showing nuclear focal weak positivity of the tumor cells (10×) – Inset: CDX2 positive control.



Slide 5. CK20 immunohistochemical stain showing cytoplasmic negativity of the tumor cells (10×) – Inset: CK20 positive control.



Slide 6. CK7 immunohistochemical stain showing membranous/cytoplasmic negativity of the tumor cells (10×) – Inset: CK7 positive control.



Slide 7. Previous ascending colon mass with hematoxylin and eosin (H&E) staining, showing malignant glands with a complex architecture alongside a squamous component (5×).

4. Clinical discussion

Primary colorectal SCC has similarly presentation to adenocarcinoma of the colon. Unfortunately, it usually present at a late stage [4]. The risk factors for primary colorectal SCC have not been well established. It tends to be associated with ulcerative colitis, parasitic infections such as schistosomiasis and amebiasis, and pelvic radiation [5–7]. Unlike anal SCC, which has been reported to be strongly associated with HPV infection, studies have failed to show an association between primary colorectal SCC and HPV, with only one report in the literature finding HPV-16 DNA with a primary SCC of the rectum [8].

The pathogenesis of primary colorectal SCC is also controversial with various mechanisms being suggested. It may arise following mucosal injury from pluripotent stem cells that are capable of multidirectional differentiation [9]. Nahas et al. supported this hypothesis by identifying a pattern of keratin expression—which can act as a "fingerprint" for carcinomas—among rectal SCC that was like that of adenocarcinoma of the rectum [5]. This keratin expression pattern was found to be different from that of SCC of the anus [5].

Given the rarity of primary colorectal SCC, histological confirmation of SCC is not sufficient to establish the diagnosis; clinicians must first exclude possible secondary causes of SCC which are more common [10,11]. Williams et al. established three criteria that must be met in order to rule out secondary causes and to help confirm the diagnosis of primary colorectal SCC [10,11]. The first criterion requires that metastasis to the colorectum from other organ sites must be excluded [11]. The second criterion requires the exclusion of any possible squamous-lined fistula affecting the involved area [11]. The third criterion requires the exclusion of a possible SSC of the anus with proximal extension to the rectum and colon [11]. Therefore, the diagnosis of primary colorectal SCC requires histologic confirmation of SCC in addition to the exclusion of possible secondary causes.

The treatment of primary colorectal SCC historically involved surgical resection with or without adjuvant chemotherapy [5]. There is no consensus regarding the optimal management of SCC, rather a collection of cases reported in the literature; this is due to the scarcity of SCC of the colon. Therefore, management is the same as adenocarcinoma of the colon, which is predominantly definitive radical resection followed by adjuvant chemotherapy and radiotherapy [2,12]. [4]. Some studies demonstrated the ability of CRT to achieve complete tumor regression and avoidance of surgical resection [13,14]. This was achieved by using protocols that combined 5-fluorouracil with cisplatin or mitomycin-C [4,13]. Following such promising results, CRT has become the treatment of choice for primary colorectal SCC, with surgical resection being reserved for those with unresponsive or recurring tumors [4,13].

Like adenocarcinoma of the colon, the prognosis of primary colorectal SCC is determined by the TNM stage [4]. AJCC's TNM classification precludes all colorectal cancers and does not differentiate between adenocarcinoma and SCC. However, literature shows that SCC has a far worse prognosis. Similar to all colon cancers, Surgical margins should be at least 5 cm, preferably 10 cm. Moreover, adequate lymph node sampling should be done as it was shown that high lymph node yield was correlated to better survival [15,16]. Lymph node yield greater than 20 was associated with improve survival [16,17].

Michelassi et al. reported 5-year survival rates for patients with SCC following surgical resection of 50 % for Dukes' Class B lesions, 33 % for Dukes' Class C lesions, and 0 % for Dukes' Class D lesions [14]. Frizelle et al. reported 5-year survival rates of 65 % for Stage I to III disease [18]. Stage IV disease carried a 5-year survival rate of only 5 % and a mean survival time of 8.5 months [18]. Frizelle et al. also noted a significantly lower 5-year survival rate for node-positive disease (23 %) compared to node-negative disease (85 %). In addition to node-positivity and Stage IV disease, other features that predict a poor prognosis include right-sided lesions, annular or ulcerated carcinomas, and Grade 3 or 4 cancer [18]. However, studies assessing the prognosis of primary colorectal SCC following CRT have not been done.

5. Conclusion

Surgery remains the most vital important step in the management of colonic SCC. The role of chemotherapy and/or radiation remains questionable. Depending on the aggressiveness of this disease the need for further frequent recurrence surveillance is required compared to conventional colorectal cancers.

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CRediT authorship contribution statement

HN: Design, data editing, revision and final approval of final manuscript. KA: Concept, data interpretation, design and final approval of manuscript. BH: Literature review and writing the manuscript. AB: Data editing and revision. LG: Revision of pathological data and interpretation. WF: Final approval of manuscript.

Declaration of competing interest

No potential conflict of interest was reported by the authors.

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