

CASE REPORT

Basaloid Squamous Carcinoma arising at Splenic Flexure of Colon

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ABSTRACT

Basaloid squamous carcinoma occurs most commonly in the upper aerodigestive tract. In the colon, the most common location is in the anal canal and is rarely reported proximal to the anal verge. Six cases are reported in the published literature, of which five are located distal to the splenic flexure. Outside the anal canal, it has been postulated that they arise from the basal cells, squamous metaplastic cells, or the cloacogenic embryologic cell rests. We report a case of a 57-year-old male patient who presented with short duration of abdominal pain and weight loss. Computed tomography (CT) abdomen revealed an irregular circumferential thickening in the terminal transverse colon, splenic flexure, and proximal descending colon. Colonoscopy showed a stricture in the splenic flexure, which was suspected for malignancy. Endoscopic biopsy was reported as poorly differentiated carcinoma. Left extended hemicolectomy was done, and a diagnosis of basaloid squamous carcinoma was made on histopathology. The tumor cells were positive for P63, CK 5/6; focally for CDX2; and negative for CK20, chromogranin, and CD56, confirming the histopathological diagnosis of basaloid squamous carcinoma. The patient is currently on adjuvant chemotherapy.

Keywords: Basaloid carcinoma, Colon, Splenic flexure.

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INTRODUCTION

Conventional squamous cell carcinoma (SCC) is rare in the colon and accounts for nearly 0.1 to 0.25 cases per 1,000 colorectal carcinomas.^{1,2} Basaloid squamous cell carcinoma (BSCC), otherwise called as transitional cloacogenic carcinoma, occurs most commonly in the upper aerodigestive tract, cervix, and thymus. In the colon, the most common location is the anal canal and is rare outside it. In the anal canal, it usually arises from the anal transitional epithelium or the cloacogenic remnants.

Some ultrastructural studies revealed that they may also arise from the transitional lining of the anal ducts or from the totipotential basal cells of the squamous epithelium.³⁻⁵ Outside the anal canal, it has been postulated that the tumor may arise from cloacogenic embryologic rests, squamous metaplastic cells, or the totipotential basal cells.⁶⁻⁸ Till date, only six cases of BSCs of the colon have been reported in the published literature, with only one occurring at the splenic flexure and the rest distal to it.⁶⁻¹¹ Here, we report one such rare case of BSC occurring at the colonic splenic flexure.

CASE REPORT

A 57-year-old male patient presented with complaints of severe continuous abdominal pain in the left lower quadrant for 3 months and weight loss of about 6 kg in 2 months. Other than a history of tubercular epididymitis, there was no other significant medical history. On per abdominal examination, a soft mass was felt in the left lumbar region. The laboratory investigations (including complete blood count, blood urea nitrogen, creatinine, blood sugar, serum electrolytes, and liver function tests) revealed only mild anemia. The carcinoembryonic antigen was mildly elevated at 8.91 µg/L (normal is 0–2.5 µg/L). Contrast-enhanced computed tomography (CT) abdomen showed an irregular circumferential thickening involving terminal transverse colon, splenic flexure, and proximal distal colon, with features suggestive of carcinoma colon. There were no signs of metastasis. On colonoscopy, an ulcerated stricture was identified at a distance of 40 cm from the anal verge, which was suspected for malignancy. Scope could not be negotiated, and rest of the mucosa was normal (Fig. 1).

On colonoscopic biopsy, a diagnosis of poorly differentiated carcinoma was made. Left extended hemicolectomy was done. Intraoperatively, a large ulceroproliferative splenic flexure mass measuring 15 × 15 cm was identified with multiple mesocolon lymph nodes. Liver surface was normal. The postoperative period was uneventful. On gross examination, a circumferential ulceroproliferative lesion measuring 16 × 8 × 8 cm was seen infiltrating the entire thickness of colon (Fig. 2). On microscopic examination, the neoplasm was composed of moderately pleomorphic atypical cells arranged in lobules and nests with peripheral palisading of basaloid cells and

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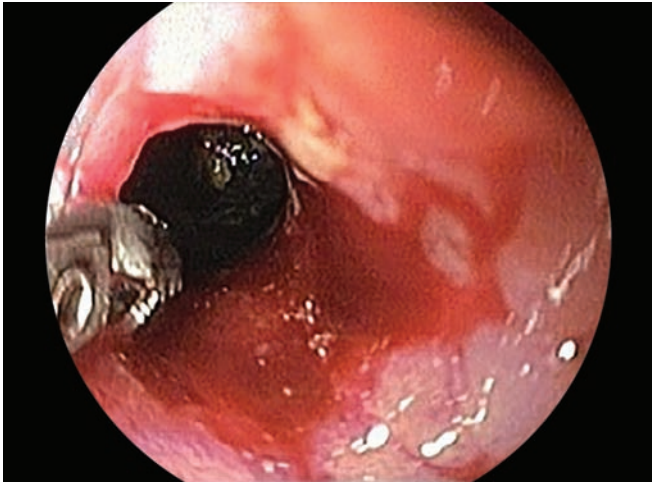


Fig. 1: Colonoscopy image showing an ulcerated stricture



Fig. 2: Ulceroproliferative growth infiltrating into the perimuscular connective tissue

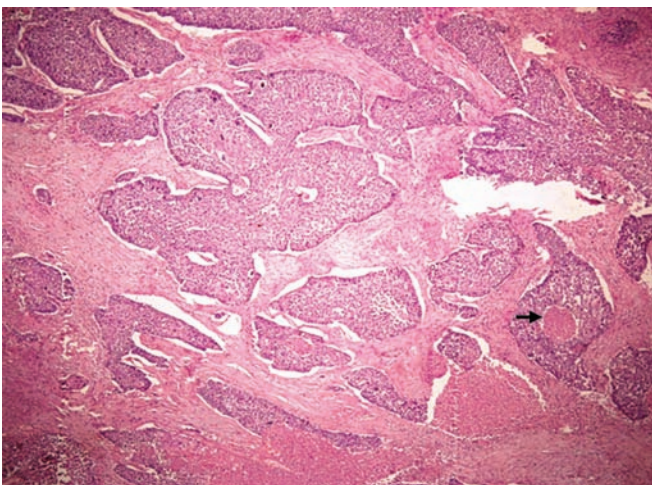


Fig. 3: The neoplasm comprises lobules and nests of atypical cells with central necrosis (arrow mark) (hematoxylin–eosin stain. Original magnification 10×)

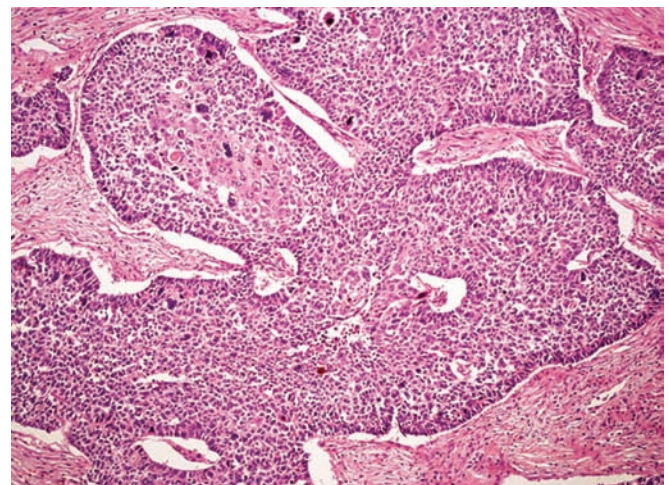


Fig. 4: High-power view showing peripheral palisading of basaloid cells with central squamous differentiation (hematoxylin–eosin stain. Original magnification 40×)

central areas of necrosis in few of the lobules. The individual basaloid cells exhibited oval hyperchromatic nuclei, inconspicuous nucleoli, and scant eosinophilic cytoplasm. The center of the lobules showed squamous differentiation with keratin pearl formation (Figs 3 and 4). Mitotic figures were frequent. The adjacent stroma showed moderate desmoplasia and moderate lymphocytic host response. The neoplasm was seen infiltrating beyond the muscularis into the perimuscular connective tissue. Perineural invasion and lymphovascular emboli were absent. The overlying epithelium did not show any evidence of squamous metaplasia or dysplasia. Out of the 11 pericolic lymph nodes available for assessment, one node showed metastasis without perinodal extension. Immunohistochemistry was performed using polymer technique for the following antibodies: CK20 (mouse monoclonal IT ks 20.8, BioGenex), CK5/6 (rabbit monoclonal Ep67, PathnSitu), p63 (mouse monoclonal

4A4, BioGenex), CD56 (mouse monoclonal 123C3, DAKO), Chromogranin (mouse monoclonal LK2H10/PR, DAKO), and CDX2 (mouse monoclonal CDX2, DAKO). The neoplastic cells were diffusely positive for CK5/6, P63; focally for CDX2; and negative for CK20, chromogranin, and CD56 (Figs 5 and 6). The histopathology was supported by the immunoprofile, and a diagnosis of BSC was made. The patient was started on 1st cycle of adjuvant chemotherapy, which included bevacizumab 2.5 mg/kg body weight, oxaliplatin 85 mg/m² over 2 hours, and capecitabine 1,000/m²/d for the first 2 weeks of each 3-week cycle. The patient is on regular treatment and follow-up. No complications have been reported till date.

DISCUSSION

Conventional SCCs are rarely reported in the colon. Before making a diagnosis of primary SCC in the colon, the following criteria need to be considered: (1) Care

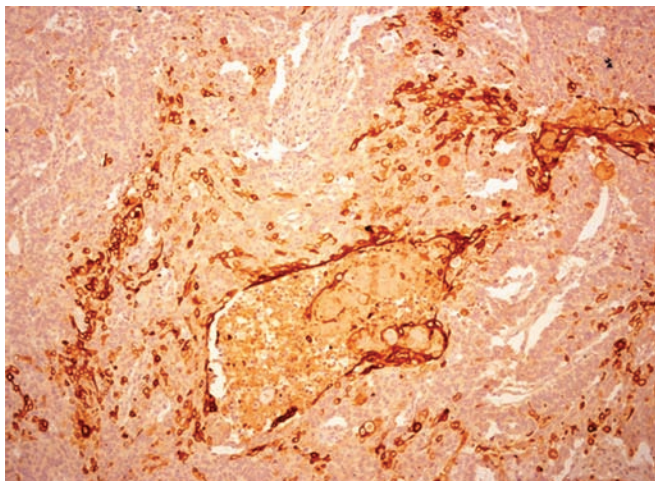


Fig. 5: CK5/6 showing membranous positivity in the neoplastic cells (hematoxylin–eosin stain. Original magnification 40×)

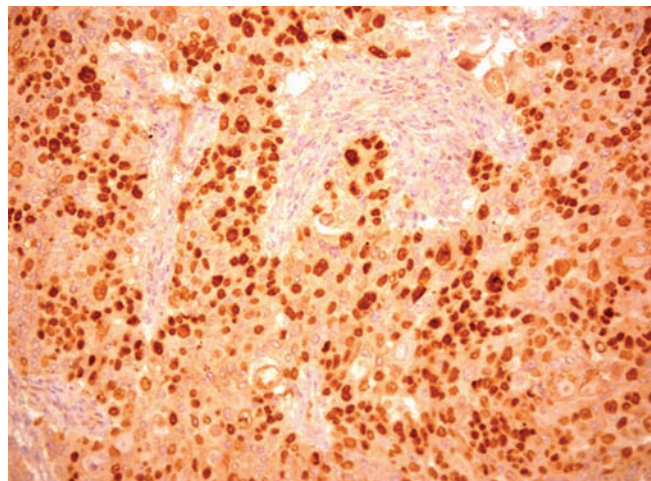


Fig. 6: P63 showing diffuse nuclear positivity in the neoplastic cells (hematoxylin–eosin stain. Original magnification 40×)

should be taken to exclude contiguous spread from other sites; (2) direct extension of primary tumor from anal canal (in case of rectal SCCs); (3) affected bowel should not be involved in fistulas lined by squamous cells; and (4) histological confirmation of SCC is mandatory.¹ Our case had fulfilled all the above-mentioned criteria.

The BSC, also known as transitional cloacogenic carcinoma, occurs in both sexes, but is more common in males between 60 and 80 years of age. It usually arises from the anal transitional epithelium, otherwise called as cloacogenic membrane, bounded superiorly by the rectal mucosa and inferiorly by the pectinate line. It may also arise from the anal ducts with transitional lining epithelium or from the basal cells.³⁻⁵ Cloacogenic carcinomas have a range of microscopic patterns of which two are well recognized: Well-differentiated transitional form as it resembles urothelial carcinoma, and the basaloid form, as it simulates cutaneous basal cell carcinoma (BCC).¹²

The BSC is rare in other sites of the colon. Strate et al⁶ had reported the first case of BSC in the sigmoid colon above the pelvic brim at the peritoneal reflection. In the literature, six cases are reported, of which five are located distal to the splenic flexure.⁶⁻¹¹ In this case, the tumor is located in the distal part of the transverse colon, splenic flexure, and proximal part of descending colon. Intriguing is the origin of the BSCs in the colon. Three mechanisms are postulated: (1) Preexisting squamous metaplasia of the colonic glands that later became dysplastic from which the tumor evolves; (2) it may also arise from cloacogenic embryological transitional cell rests; or (3) from the totipotent basal cells.⁶⁻⁸ We did not find squamous metaplasia or cloacogenic remnants in our case.

When the tumor arises close to the anal canal, it is difficult to distinguish BSCs from BCCs. The retraction spaces around the infiltrating nests, lack of increased atypical mitosis, and absence of precursor lesions aid in

differentiating BCCs from BSCs. In the present case, the location of the tumor was far from the anal verge, and the histological differentials considered were adenosquamous carcinoma or a neuroendocrine carcinoma due to presence of rosette-like structures around the necrotic foci in some sections. The characteristic histological features of BSC that differentiate it from small cell carcinoma are presence of central comedo-like necrosis, central squamous differentiation, and peripheral palisading of basaloid cells. Immunohistochemically, these cells are immunoreactive for CK5/6, P63, and 34βE12, whereas the small cell carcinomas are positive for CD56, chromogranin, and synaptophysin.

Histological diagnosis of BSCs on diagnostic colonoscopic biopsies is often difficult due to smaller tissue sampled and presence of necrosis. These tumors may also produce ectopic hormones, such as parathyroid hormone, adrenocorticotropic hormone (ACTH), or ACTH-like substance.^{6,9} Although the clinical presentation, CT findings, and gross appearance of these neoplasm and further adjuvant chemotherapy are similar to conventional adenocarcinomas, the prognosis has been reported to be worse as compared with the latter.¹³ These tumors pose a diagnostic challenge due to their rare location in the splenic flexure, the difficulty in diagnosis on initial confirmatory colonoscopic biopsies, and histological similarities with small cell neuroendocrine and BCCs in less-differentiated variants.

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