Case Report

Collision tumor of the cecum and ileocecal valve composed of mucinous adenocarcinoma and neuroendocrine tumor: a case report

Leslie R. Elmore[1]^, Alexandra Drymon[1], Angel Toca[2], Andrei I. Gritsiuta[1]^, William Gilleland[1]

[1]Department of Surgery, University of Pittsburgh Medical Center, Pittsburgh, PA, USA; [2]Department of Pathology, University of Pittsburgh Medical Center, Pittsburgh, PA, USA

Contributions: (I) Conception and design: LR Elmore, A Drymon, A Toca; (II) Administrative support: A Toca, W Gilleland; (III) Provision of study materials or patients: W Gilleland; (IV) Collection and assembly of data: LR Elmore, A Drymon, A Toca; (V) Data analysis and interpretation: LR Elmore, A Drymon, A Toca; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

Correspondence to: Leslie R. Elmore, DO. Department of Surgery, University of Pittsburgh Medical Center, Pittsburgh, PA, USA; 2200 Memorial Drive, Farrell, PA 16121, USA. Email: elmorel@upmc.edu.

Background: Collision tumors of the gastrointestinal (GI) tract are thought to be uncommon, with those of the colon being rare with very few cases reported in current literature. There are three proposed theories regarding the etiology of collision tumors currently, including the "double primaries", the "biclonal malignant transformation", and the "tumor-to-tumor carcinogenesis" theories. Prognosis of collision tumors remains unclear. To our knowledge, this is the fifth case of a collision carcinoma involving the cecum and ileocecal valve and the first report of a collision carcinoma including both mucinous adenocarcinoma and neuroendocrine tumor of the cecum and the ileocecal valve. The aim of this paper is to explore the history of collision tumors and associated nomenclature, defined diagnostic criteria, and proposed theories for etiology in addition to patient presentation, approach to diagnosis, treatment options, and prognosis.

Case Description: We present the case of an 83-year-old female who presented to the emergency room with a 4-month history of cramping abdominal pain associated with nausea, emesis, and decreased appetite with associated weight loss. Diagnostic imaging demonstrated a bowel obstruction secondary to a mass in the cecum and she underwent an exploratory laparotomy with right hemicolectomy. She was found to have a collision carcinoma of the cecum and ileocecal valve containing both mucinous adenocarcinoma and neuroendocrine tumor. Diagnosis was confirmed post-operatively with pathologic examination and immunohistochemical testing.

Conclusions: Diagnosing collision tumors upon patient presentation is exceedingly difficult as the symptoms are often identical to other neoplasms of the GI tract and vary based on location of the tumor. It is thought that the true prevalence of collision tumors is underestimated due to history of changing nomenclature, unclear diagnostic criteria, unreported cases, and unrecognized cases. Furthermore, new advances in immunohistochemical evaluation have allowed for better characterization of these neoplasms. With clarification regarding nomenclature, diagnostic criteria and expanding awareness, it is our hope that this leads to an increase in reported cases, allowing for an expanded discussion and resulting growth of literature and further studies. Further knowledge regarding the pathogenesis, treatment, and prognosis is needed.

Keywords: Collision tumor; adenocarcinoma; neuroendocrine tumor; cecum; case report

Received: 22 April 2024; Accepted: 26 July 2024; Published online: 13 September 2024.

doi: 10.21037/acr-24-87

View this article at: https://dx.doi.org/10.21037/acr-24-87

^ ORCID: Leslie R. Elmore, 0009-0008-1422-7747; Andrei I. Gritsiuta, 0000-0003-2265-9992.

Introduction

Adenocarcinoma is the most common malignancy of the colon. Glandular-endocrine tumors of the gastrointestinal (GI) tract were first discovered in 1924 and are uncommon, especially those of the colon. They are classified into three groups: collision (side by side), composite (combined), and amphicrine (features of both neuroendocrine and glandular within one cell). Collision tumors of the GI tract are uncommon, with those of the colon being rare. These will be the focus of this case report. Collison carcinomas of the GI tract have been described to include variations of adenocarcinoma, neuroendocrine tumors, transitional cell carcinoma, lymphoma, ovarian granulosa cell tumors, and melanoma. To our knowledge, there have been four cases of known collision tumors of the cecum and ileocecal valve reported, all of which involve adenocarcinoma and lymphoma (*Table 1*). We report the fifth case of a collision carcinoma involving the cecum and ileocecal valve. To our knowledge, this is the first report of a collision carcinoma including both mucinous adenocarcinoma and neuroendocrine tumor in the cecum and the ileocecal valve. This case is written in accordance with the CARE reporting checklist (available at https://acr.amegroups.com/article/view/10.21037/acr-24-87/rc).

Highlight box

Key findings

- To our knowledge, we present the second case of a collision carcinoma including both mucinous adenocarcinoma and neuroendocrine tumor of the colon and the first involving the cecum and ileocecal valve.

What is known and what is new?

- Collision tumors of the of the gastrointestinal (GI) tract are uncommon, with those of the colon being rare. To our knowledge, there have been four reported cases of collision carcinoma involving the cecum and ileocecal valve. Diagnosis relies on pathological examination and immunohistochemical evaluation.
- It is thought that the true prevalence of collision tumors is underestimated due to history of changing nomenclature, unclear diagnostic criteria, unreported cases, and unrecognized cases.

What is the implication, and what should change now?

- Diagnosing collision tumors upon patient presentation is exceedingly difficult as the symptoms are often identical to other neoplasms of the GI tract and vary based on tumor location. With clarification regarding nomenclature, diagnostic criteria and expanding awareness, it is our hope that this leads to an increase in reported cases, allowing for an expanded discussion and resulting growth of literature and further studies. Further knowledge regarding the pathogenesis, treatment, and prognosis is needed.

Case presentation

An 83-year-old female with a past medical history significant for hypertension, hypothyroidism, and osteoporosis presented to the emergency room with a 4-month history of cramping abdominal pain associated with nausea and emesis. She endorsed a decreased appetite, which resulted in a 40-pound weight loss over 6 months. Her last colonoscopy was over 10 years prior. She denied a family history of colon cancer. On physical exam, her abdomen was noted to be soft, non-distended, and tender to palpation of the right low quadrant. Vitals on arrival revealed her to be afebrile and hemodynamically stable. Laboratory analysis noted white blood count 4.1×10^5/L, hemoglobin 14.5 g/dL, hematocrit 43.1%, lactate 1.0 mmol/L, sodium 134 mmol/L, potassium 3.4 mEq/L, blood urea nitrogen 10 mg/dL, creatinine 0.66 mg/dL, and albumin 2.9 g/dL. Tumor markers noted carcinoembryonic antigen (CEA) 2.5 ng/mL, carbohydrate antigen 19-9 (CA19-9) 16 U/mL, and carbohydrate antigen 125 (CA-125) 72 U/mL. Computed tomography (CT) abdomen and pelvis with intravenous (IV) contrast demonstrated a hyperattenuating mass in the cecum and ascending colon suspicious for neoplasm with resulting obstruction. A nasogastric tube was placed for decompression, and she was admitted for further workup and management. Colonoscopy was attempted, which demonstrated colonic diverticulosis, however, was aborted due to poor bowel preparation. Repeat CT abdomen and pelvis with IV and oral contrast demonstrated worsening small bowel obstruction with redemonstration of mass occupying the cecum with extension to the ileocecal valve (*Figure 1*).

She was taken to the operating room and underwent bilateral ureteral stent placement, exploratory laparotomy, lysis of adhesions, right hemicolectomy with side-to-side anastomosis, and abdominal closure. Intraoperatively, a mass located to the cecum was visualized without demonstration of gross metastasis on exploration of the abdomen.

Grossly, pathologic examination noted an 8.5 cm × 6.7 cm × 2.8 cm tan-red, friable, circumferential, obstructing mass centered within the cecum with gross extension through the ileocecal valve into the ileum with an abutting 3.1 cm × 3.0 cm × 2.0 cm tan-brown, firm polypoid mass in the ileum with invasion through the bowel wall abutting the serosa with calcifications. The appendix was not

Table 1 Reported cases of collision tumors involving cecum and/or ileocecal valve in current literature

Author	Year	Age (years)	Sex	Presentation	Histology	Treatment	Follow-up
Bao (1)	2020	77	M	RLQ pain	MD-AC; DLBCL	Surgery, chemotherapy	No recurrence (4 months)
Kus (2)	2016	73	M	Muscle weakness, fatigue, weight loss, abdominal pain	AC; follicular lymphoma	Surgery, chemotherapy	Recurrence, expired
Sasaki (3)	2010	62	M	RLQ pain, anemia	MD-AC; follicular lymphoma	Surgery, chemotherapy	Unknown
Shigeno (4)	2011	76	F	RLQ pain, anemia	MD-AC; DLBCL	Surgery	Recurrence, expired

Those described as mixed without delineation into collision were assumed to be composite tumors and excluded. M, male; F, female; RLQ, right lower quadrant; MD, moderately differentiated; AC, adenocarcinoma; DLBCL, diffuse large B cell lymphoma.

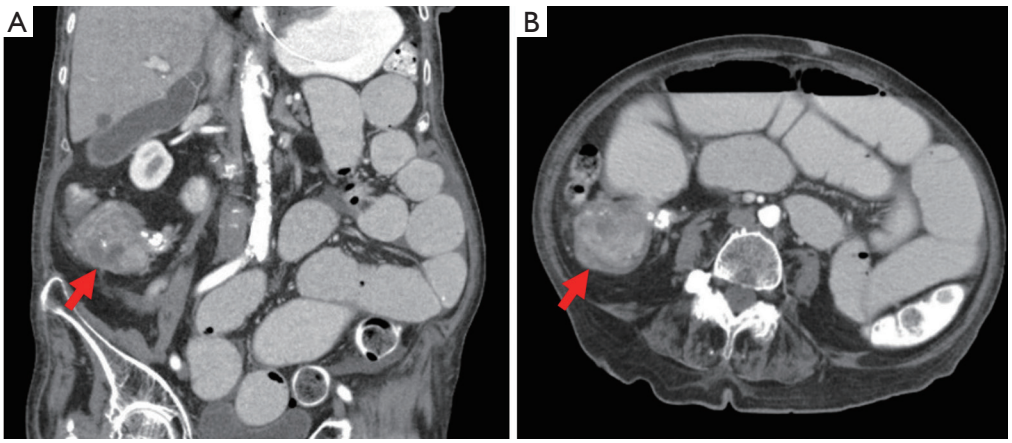

Figure 1 Computed tomography of the abdomen and pelvis demonstrating collision tumor of cecum and ileocecal valve: (A) coronal and (B) axial cross-sectional imaging, demonstrated with red arrows.

involved. Microscopically, the larger mass was noted to be moderately differentiated mucinous adenocarcinoma, with invasion into the subserosa without lympho-vascular or perineural invasion (*Figure 2*). Staging was noted to be pT3N0. The smaller mass was noted to be a well differentiated neuroendocrine tumor with invasion of the subserosa and perineural invasion without lympho-vascular invasion. Staging was noted to be pT4N1, low grade. Two/16 lymph nodes were positive for metastatic neuroendocrine tumor while 16/16 lymph nodes were negative for metastatic colonic mucinous carcinoma (*Figure 3*). Immunohistochemically, the tumor was noted to be strongly positive for cytokeratin 20 (CK20) and CEA in the adenocarcinoma and strongly positive for chromogranin A and synaptophysin in the neuroendocrine tumor. Ki67 was noted to be <1% (*Figure 2B,2C*). A mucicarmine special stain was performed, which highlighted mucin in the adenocarcinoma.

Further clinical evaluation and radiological imaging completed as an outpatient did not demonstrate metastatic disease. Upon outpatient oncology discussion with the patient regarding treatment plans, the decision was made to proceed with surveillance alone without planned adjuvant chemotherapy. Octreotide treatment was not pursued as the tumor was removed *en bloc*. At 6-month follow-up, her CEA was noted to be 1.6 ng/mL and chromogranin A was noted to be 70 ng/mL. At 1-year follow-up appointment, CEA was noted to be 2.5 ng/mL and chromogranin A was noted to be 289 ng/mL. These increasing numerical values demonstrate progression of disease. CT chest abdomen and pelvis demonstrated no evidence of disease recurrence. One-year follow-up colonoscopy noted persistent diverticulosis, however, was aborted due to technical difficulty of procedure secondary to redundancy, restricted colon mobility, and severe diverticular disease. Passing a pediatric colonoscope was also attempted without

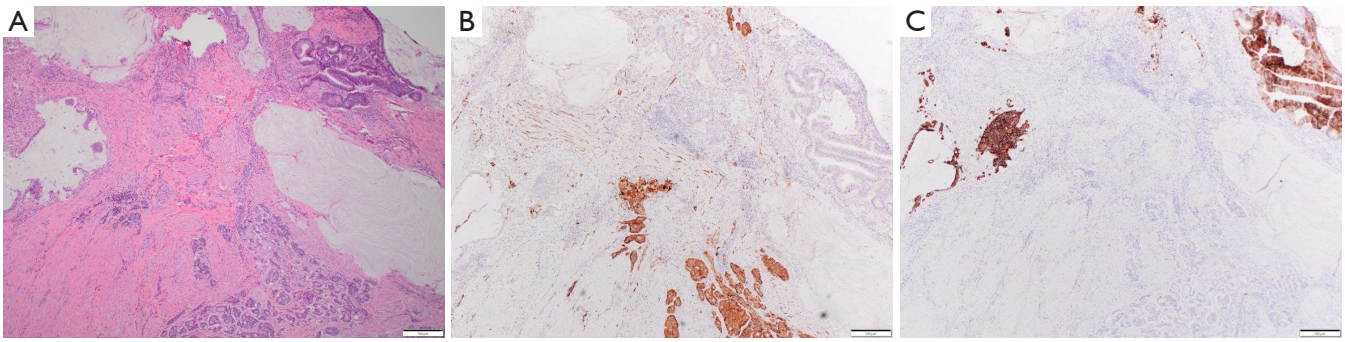

Figure 2 Collision tumor, with colon adenocarcinoma shown on the superior aspect and well differentiated neuroendocrine tumor shown on the inferior aspect: (A) H&E stain ×40; (B) CK20 immunohistochemical stain highlighting adenocarcinoma ×40; (C) synaptophysin immunohistochemical stain highlighting neuroendocrine tumor ×40. H&E, hematoxylin and eosin; CK20, cytokeratin 20.

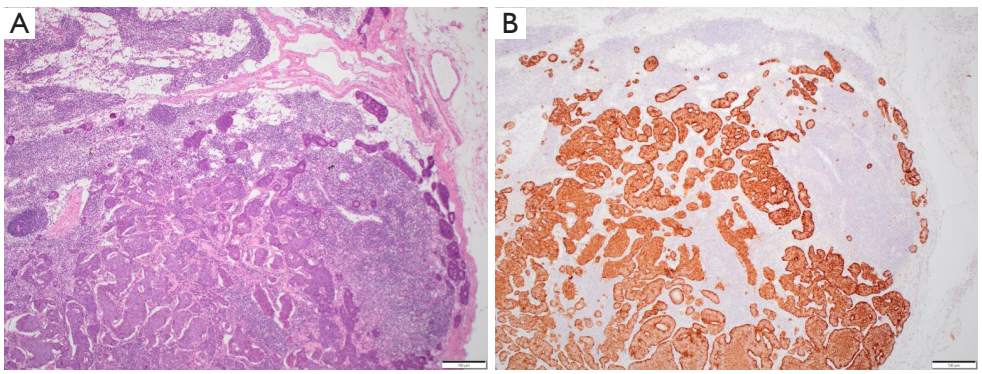

Figure 3 Metastatic neuroendocrine tumor to regional lymph node: (A) H&E stain ×40; (B) synaptophysin immunohistochemical stain decorates the metastatic neuroendocrine tumor ×40. H&E, hematoxylin and eosin.

success. Repeat colonoscopy 1 month later was also aborted secondary to technical difficulty of procedure. At the 2-year follow-up appointment, the patient unfortunately refused to have a positron emission tomography (PET) scan or CT scan imaging completed. CEA was noted to be 3.0 ng/mL and chromogranin A was noted to be 148 ng/mL. Colonoscopy was not pursued secondary to failed past attempts. Throughout the post-operative period, CEA and chromogranin A levels were obtained every 3 months. The patient has been noted to be doing well overall in the post-operative and post-diagnosis period with 6-month to 1 year follow-up appointments.

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent for publication of this case report and accompanying images was not obtained from the patient or the relatives after all possible attempts were made.

Discussion

In 1924, the first glandular-endocrine tumor of the GI tract was documented by Lewin (5). In 1987, Lewin classified these tumors with two distinct neoplasms into three categories: collision, composite, and amphicrine. Collision tumors are described as having both glandular and endocrine characteristics in a “side by side pattern”. Composite tumors are described as having both glandular and endocrine characteristics in an intermixed pattern. Amphicrine tumors are described as having both glandular and endocrine characteristics within the same cell (6). In 1980, Spagnolo and Heenan proposed guidelines that included three criteria for diagnosing collision tumors: (I) two distinct topographically separate sites of origin from the two components must be present, e.g., squamous cell

carcinoma arising from esophageal squamous epithelium and adenocarcinoma arising from gastric mucosa; (II) there must be at least some separation of the two components so that, despite intimate mixing at the points of juxtaposition, dual original can still be recognized; and (III) at the areas of collision, in addition to intimate mixing of the two components, some transitional patterns may be seen, such as a mucoepidermoid appearance in the case of collision between squamous carcinoma and adenocarcinoma (7).

In 2000, the World Health Organization (WHO) designated these tumors as mixed exocrine-endocrine carcinomas (MEECs) (8). In 2010, the WHO designated these tumors with two separate neoplasms as mixed adeno-neuroendocrine carcinomas (MANECs) (6,9,10). Diagnostic requirements require that each tumor must create 30% of the overall tumor, both tumors must be malignant, and both neoplasms need to be graded separately. Amphicrine tumors were also removed from the classification at this time (6). In 2017, MANEC was redefined as mixed neuroendocrine-non-endocrine neoplasms (MiNENs) to allow tumors aside from adenocarcinoma, such as sarcoma or squamous cell carcinoma to be included under one broad category. It is noted this name was changed from carcinoma to neoplasm to include low grade tumors (9). Collision tumors remain a separate entity under the umbrella of mixed neuroendocrine neoplasms, separate from MiNENs, as MiNENs are thought to be derived from one common pluripotent stem cell (8). Furthermore, MiNENs are described as mixed cells, much as composite tumors are described.

Collision tumors are thought to be two distinct tumors juxtaposed with two different primary origins. In contrast, composite tumors are thought to be from "multidirectional differentiation of a single cell" (6). The exact mechanism of collision tumors remains unclear. Current theories include two independent tumors in close approximation that propagated from two different cell lines (known as "double primaries" theory or "biclonal malignant transformation"), a common progenitor stem cell that differentiates into two different neoplasms (known as "monoclonal origin" theory), and the phenomenon of "tumor-to-tumor carcinogenesis" in which the initial neoplasm stimulates the development of the second neoplasm (5,6,11,12).

Collision tumors have been documented throughout the GI tract, with those in the colon being rare. They are estimated to compose about 3% to 9.6% of all colorectal tumors (6). Furthermore, Bhattacharya *et al.* estimates that the incidence of collision carcinoma tumors of the GI tract consisting of both adenocarcinoma and a neuroendocrine tumor is approximately 0.3% to 4.3% (7). According to Minaya-Bravo *et al.*, of those found in the colon, 56% are thought to present in the right colon, 3% in the transverse colon, and 41% in the left colon (6). Of those found in the colon, it is thought that 58% are collision tumors and 42% are composite tumors, according to Li *et al.* (13). The prevalence of collision tumors of the cecum and ileocecal valve is unknown as very few cases have been reported. *Table 1* demonstrates four known collision tumors of the cecum and ileocecal valve with all four cases involving both lymphoma and adenocarcinoma. To our knowledge, this is the second report of a collision carcinoma including both mucinous adenocarcinoma and neuroendocrine tumor in the colon and the first involving the cecum and ileocecal valve. Reported components of collision carcinoma throughout the GI tract have included variations of adenocarcinoma, neuroendocrine tumors, transitional cell carcinoma, lymphoma, ovarian granulosa cell tumors, and melanoma (1,2,7,11,14-19).

According to Li *et al.*, collision tumors usually affect elderly patients with an average age of 61.9 years. Collision tumors are thought to affect males to females in a 1.1:1.0 ratio (13). Of the four known cases of collision tumors of the cecum and ileocecal valve plus the case we report, the average age was noted to be 74.2 years at time of diagnosis and the male to female ratio was found to be 1.5:1.0. Unfortunately, diagnosis is difficult as patients present with non-specific symptoms and radiologically, findings are non-specific (11,12). Collision tumors of the colon usually present with abdominal pain, bowel obstruction, weight loss, and a positive occult blood fecal test with a palpable mass and diarrhea being less common (13). We found that in patients with collision tumors of the cecum and ileocecal valve, 3 out of 4 patients presented with right lower quadrant (RLQ) abdominal pain with the fourth patient's abdominal pain not being characterized. Additional symptoms of those with a collision tumor of the cecum and ileocecal valve include anemia, muscle weakness, fatigue, weight loss, obstructive symptoms, and nausea. Li *et al.* found that in most cases, the neuroendocrine neoplasm was found to be nonfunctioning. During endoscopic evaluation of the mass, 9% demonstrated an ulcerating lesion, 30% demonstrated a mass, and 57% demonstrated a polyp or polypoid lesion (13). These tumors are difficult to identify pre-operatively and are identified and diagnosed based on pathologic examination and immunohistochemical evaluation (11,12). Histologically, collision tumors demonstrate a clear demarcation between the two neoplasms; this is in contrast to composite tumors in

which the neoplasm characteristics are intermixed (12).

It is thought that the true prevalence of colorectal collision tumors is underestimated due to previously unclear diagnostic criteria, rapidly changing nomenclature, unreported cases, and unrecognized cases. It is also thought that prior to the advances of immunohistochemical evaluation, a second neoplastic component may not have been recognized, impacting reported cases (8).

There are few pieces of literature that have looked at the metastasis patterns of collision carcinomas. Minaya-Bravo *et al.* described a case of a collision carcinoma composed of adenocarcinoma and neuroendocrine tumor of the transverse colon in which metastasis showed both glandular and neuroendocrine characteristics (6). Pecorella *et al.* also reported a case of an adenocarcinoma and neuroendocrine collision carcinoma with lymph node metastasis containing both components (5). Meşină *et al.* also described similar findings with metastasis containing both components (11). These examples refute the "double primaries" theory set forth previously. Interestingly, the collision tumor we are reporting had metastatic disease to a regional lymph node demonstrating only neuroendocrine tumor. Given the rare nature and rapidly changing nomenclature of these tumors, unfortunately, there are currently no definitive guidelines for management or treatment (12). Currently, treatment is focused on the more aggressive tumor (8,12). Surgical excision is noted to be at the forefront of treatment, with the role of adjuvant chemotherapy and/or octreotide being unclear (6). Management utilizing multidisciplinary cancer teams and close surveillance has, however, remained a mainstay of treatment (10). Prognosis is largely based on the two types of malignancies present; there are very few reports in current literature expanding on prognosis of collision tumors. According to Nannar *et al.*, colorectal neuroendocrine tumors typically have a poor prognosis secondary to their aggressive nature and late diagnosis. The median survival rate is 7.1 to 14.7 months at 5 years for neuroendocrine tumors and 36 months for colorectal adenocarcinoma (12). Li *et al.* reported that tumor related deaths associated with collision tumors composed of glandular and neuroendocrine components was 68% at 20 months (13).

Conclusions

Collision tumors of the of the GI tract are uncommon, with those of the colon being rare. To our knowledge, there have been four reported cases of collision tumors of the cecum and ileocecal valve, all of which involve both lymphoma and adenocarcinoma. The etiology of collision tumors remains poorly understood, although several theories exist including "double primaries", "monoclonal origin", and "tumor-to-tumor carcinogenesis". Diagnosis of these neoplasms on presentation tends to be exceedingly difficult, as the symptoms are often variable and present based on tumor location. In our case, diagnosis was made by cross sectional imaging with post-operative pathologic evaluation, in addition to immunohistochemical analysis. Treatment options usually include resection, however, vary based on characteristics of the two neoplasms present. A multidisciplinary team approach in addition to close surveillance remains the mainstay of treatment. Prognosis is based on the two neoplasms present. To our knowledge, we present the second case of a collision carcinoma including both mucinous adenocarcinoma and neuroendocrine tumor in the colon and the first involving the cecum and ileocecal valve.

In conclusion, new advances in immunohistochemical evaluation have allowed for better characterization of these neoplasms. With clarification regarding nomenclature, diagnostic criteria and expanding awareness, it is our hope that this leads to an increase in reported cases, allowing for an expanded discussion and resulting growth of literature and further studies. Further knowledge regarding the pathogenesis, treatment, and prognosis is needed.

Acknowledgments

Funding: None.

Footnote

Reporting Checklist: The authors have completed the CARE reporting checklist. Available at https://acr.amegroups.com/article/view/10.21037/acr-24-87/rc

Peer Review File: Available at https://acr.amegroups.com/article/view/10.21037/acr-24-87/prf

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://acr.amegroups.com/article/view/10.21037/acr-24-87/coif). A.I.G. serves as an unpaid editorial board member of *AME Case Reports* from February 2023 to January 2025. The other authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately

investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent for publication of this case report and accompanying images was not obtained from the patient or the relatives after all possible attempts were made.

References

1. Bao L, Feng X, Wang T, et al. Collision tumor of carcinoma and lymphoma in the cecum: case report and review of literature. Int J Clin Exp Pathol 2020;13:2907-15.
2. Kus T, Aktas G, Kalender ME, et al. Collision tumor consisting of primary follicular lymphoma and adenocarcinoma in the cecum: A case report and literature review. Oncol Lett 2016;11:2801-5.
3. Sasaki S, Hatanaka K, Sahara N, et al. Collision tumor of primary malignant lymphoma and adenocarcinoma in the colon: report of a case. Surg Today 2010;40:975-81.
4. Shigeno T, Fujimori K, Tsuruta F, et al. Ileocecal collision tumor composed of adenocarcinoma and primary malignant lymphoma. Clin J Gastroenterol 2011;4:79-84.
5. Pecorella I, Memeo L, Ciardi A, et al. An unusual case of colonic mixed adenoendocrine carcinoma: collision versus composite tumor. A case report and review of the literature. Ann Diagn Pathol 2007;11:285-90.
6. Minaya-Bravo AM, Garcia Mahillo JC, Mendoza Moreno F, et al. Large cell neuroendocrine - Adenocarcinona mixed tumour of colon: Collision tumour with peculiar behaviour. What do we know about these tumours? Ann Med Surg (Lond) 2015;4:399-403.
7. Bhattacharya A, Saha R, Biswas J, et al. Collision tumors in the gastrointestinal tract: a rare case series. Int Med Case Rep J 2012;5:73-7.
8. Toor D, Loree JM, Gao ZH, et al. Mixed neuroendocrine-non-neuroendocrine neoplasms of the digestive system: A mini-review. World J Gastroenterol 2022;28:2076-87.
9. Kanthan R, Tharmaradinam S, Asif T, et al. Mixed epithelial endocrine neoplasms of the colon and rectum - An evolution over time: A systematic review. World J Gastroenterol 2020;26:5181-206.
10. Brathwaite S, Rock J, Yearsley MM, et al. Mixed Adeno-neuroendocrine Carcinoma: An Aggressive Clinical Entity. Ann Surg Oncol 2016;23:2281-6.
11. Meşină C, Vasile I, Ciobanu D, et al. Collision tumor of recto-sigmoidian junction - case presentation. Rom J Morphol Embryol 2014;55:643-7.
12. Nannar J, Anciro A, Nanda AK. Collision tumor at the rectum consisting of a neuroendocrine carcinoma and adenocarcinoma: A case report. Ann Med Surg (Lond) 2022;78:103748.
13. Li Y, Yau A, Schaeffer D, et al. Colorectal glandular-neuroendocrine mixed tumor: pathologic spectrum and clinical implications. Am J Surg Pathol 2011;35:413-25.
14. Furlan D, Cerutti R, Genasetti A, et al. Microallelotyping defines the monoclonal or the polyclonal origin of mixed and collision endocrine-exocrine tumors of the gut. Lab Invest 2003;83:963-71.
15. Lin HH, Jiang JK, Lin JK. Collision tumor of low-grade B-cell lymphoma and adenocarcinoma with tuberculosis in the colon: a case report and literature review. World J Surg Oncol 2014;12:147.
16. Lin YS, Hamilton AER, Henderson C, et al. Tiny but Mighty: Collision Tumor of a Superficial Adenocarcinoma Arising From a Tubulovillous Adenoma with Associated Low-Grade Follicular Lymphoma. Int J Surg Pathol 2021;29:759-63.
17. Cornacchia C, Rutigliani M, Belli F, et al. Collision Tumor Composed of an Inflammatory Myofibroblastic Tumor and Adenocarcinoma of the Colon: a Rare Entity. J Gastrointestin Liver Dis 2020;29:461-3.
18. Dias AR, Pinto RA, Mory E, et al. Synchronous collision malignant melanoma and adenocarcinoma of the rectum. Tech Coloproctol 2010;14:181-4.
19. Moussa S, Nechi S, Chaabane A, et al. Collision tumor of the appendix incidentally discovered in a patient diagnosed with an adenocarcinoma of the colon: a case report. Pan Afr Med J 2022;43:161.

doi: 10.21037/acr-24-87

Cite this article as: Elmore LR, Drymon A, Toca A, Gritsiuta AI, Gilleland W. Collision tumor of the cecum and ileocecal valve composed of mucinous adenocarcinoma and neuroendocrine tumor: a case report. AME Case Rep 2024;8:109.