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A resected case of medullary carcinoma of the ascending colon followed by infarction of the greater omentum mimicking anastomotic leakage



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ABSTRACT

INTRODUCTION: Medullary carcinoma is a rare type of colorectal adenocarcinoma, and omental infarction is a rare cause of acute abdomen.

PRESENTATION OF CASE: A 72-year-old woman underwent single-incision laparoscopic right hemicolectomy for ascending colon cancer. Pathological examination showed a medullary carcinoma (MC) of T4aN0M0 Stage IIB. Her postoperative course was uneventful, and she was discharged on postoperative day (POD) 6. From POD 7, she suffered from fever, and she returned to the hospital on POD 9. Plain computed tomography showed free air beside the anastomotic site around the elevated density of fat tissue and gallbladder wall thickening with a gallstone. Suspecting anastomotic leakage with acute cholecystitis, probe laparotomy was performed. Intraoperative observation confirmed omental infarction with acute cholecystitis, and no leakage was found at the anastomotic site. Therefore, the necrotic part of the greater omentum was resected, and cholecystectomy was performed. She has remained well, with no evidence of recurrent cancer during the 12 months of follow-up without chemotherapy after the surgery for MC of the ascending colon.

DISCUSSION: MC should be distinguished from other more aggressive, non-glandular tumors of the colon because MC appears to have a better survival outcome than undifferentiated colon adenocarcinoma. Omental infarction should be considered in the differential diagnosis of acute abdomen after surgery.

CONCLUSION: A rare case of medullary carcinoma of the ascending colon followed by infarction of the greater omentum mimicking anastomotic leakage is presented.

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1. Introduction

Medullary carcinoma (MC) is a rare type of colorectal adenocarcinoma that is almost always microsatellite-unstable, and it has a strong association with hereditary non-polyposis colorectal cancer (HNPCC or Lynch syndrome) [1,2]. Omental infarction is caused by torsion of the greater omentum, and it is a rare cause of acute abdomen [3]. A rare case of a patient undergoing single-incision laparoscopic colectomy of MC of the ascending colon followed by infarction of the greater omentum mimicking anastomotic leakage is described. The work has been reported in line with the SCARE criteria [4]

2. Case presentation

A previously healthy 72-year-old woman visited our hospital because of blood in the stool. Colonoscopy showed a type 2 colon tumor located at the hepatic flexure. Examination of biopsy specimens showed solid-type poorly differentiated adenocarcinoma. The serum levels of carcinoembryonic antigen (CEA) and carbohydrate antigen 19-9 (CA19-9) were 0.8 ng/mL and <2.0 U/mL, respectively. Computed tomography (CT) showed colonic wall thickening at the hepatic flexure, but no distant or lymph node metastases. These findings led to a diagnosis of advanced ascending colon cancer of T3N0M0 stage IIA according to the International Union Against Cancer TNM classification (UICC), 7th edition. Therefore, single-incision laparoscopic right hemicolectomy was performed.

The resected specimen revealed a type 2 cancer, measuring 60 × 37 mm (Fig. 1). Hematoxylin and eosin staining of the tumor

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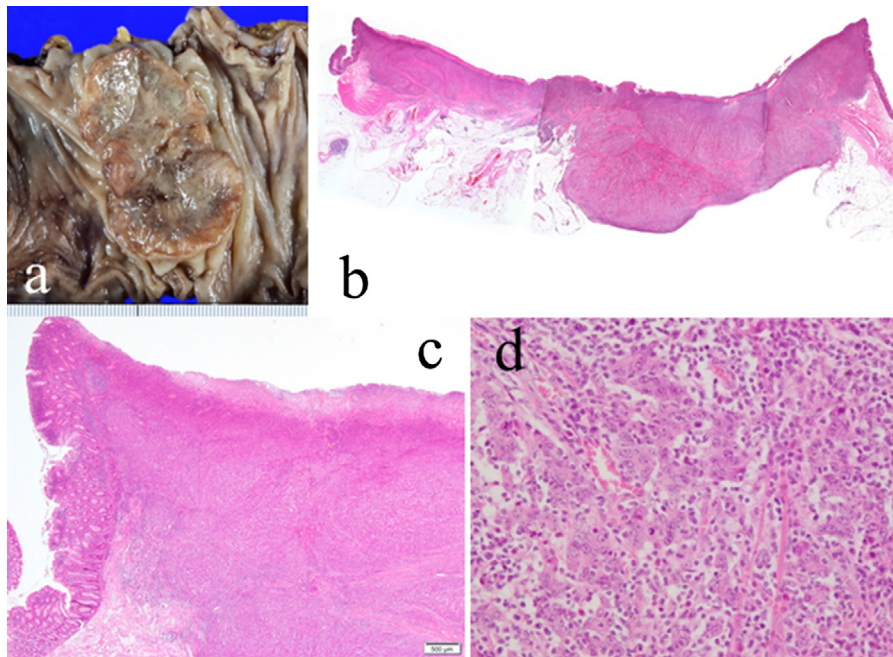


Fig. 1. Colectomy, hepatic flexure mass, hematoxylin and eosin stain.

a) The resected specimen reveals a type 2 cancer, measuring 60 × 37 mm. b–d) Hematoxylin and eosin stain demonstrates diffuse proliferation of markedly pleomorphic tumor cells occurring in a background of intense peri- and intra-tumoral lymphocytic infiltration. The tumor nuclei are vesicular, with frequent prominent nucleoli, and they are surrounded by abundant eosinophilic cytoplasm.

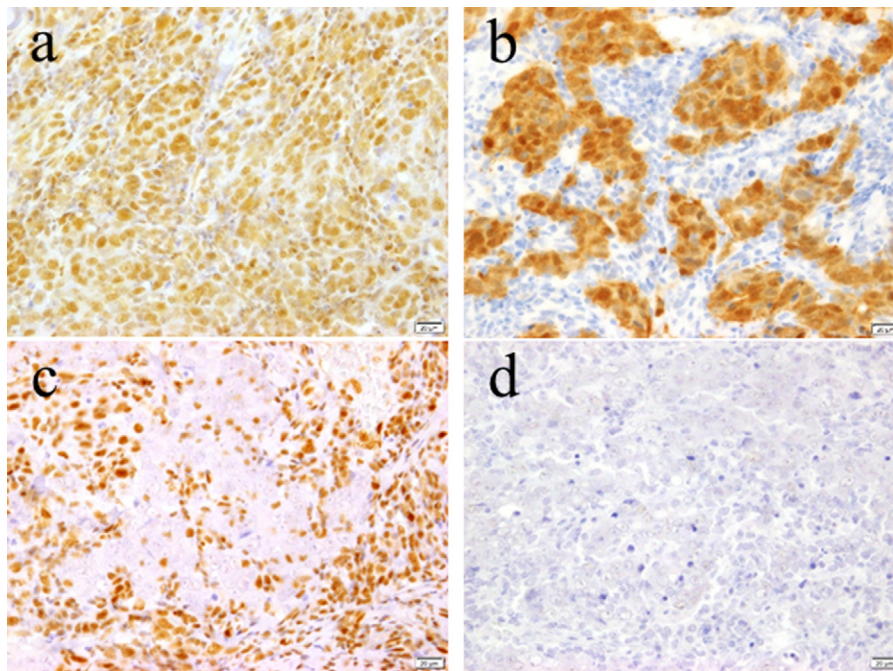


Fig. 2. Immunohistochemical staining.

a) Positive staining for MSH2 in both the medullary carcinoma and lymphocytes.
 b) Positive staining for calretinin in the medullary carcinoma.
 c) Loss of MLH1 protein expression by the medullary carcinoma, while the background lymphocytes retain their normal expression.
 d) Loss of CDX2 protein expression by the medullary carcinoma

demonstrated diffuse proliferation of markedly pleomorphic tumor cells occurring in a background of intense peri- and intra-tumoral lymphocytic infiltration. The tumor nuclei were vesicular with frequent prominent nucleoli, and they were surrounded by abundant eosinophilic cytoplasm (Fig. 1). The tumor cells infiltrated through the serosa, with no lymph node or distant metastases. Immunohis-

tochemical staining was performed, and the cells were positive for MSH2, calretinin, and vimentin but negative for MLH1, CDX2, CK7, CK20, CD3, and CD20 (Fig. 2). Therefore, the ascending colon cancer was diagnosed as MC of T4aN0M0 Stage IIB.

The patient's postoperative course was uneventful, and she was discharged on postoperative day (POD) 6. From POD 7, she suf-

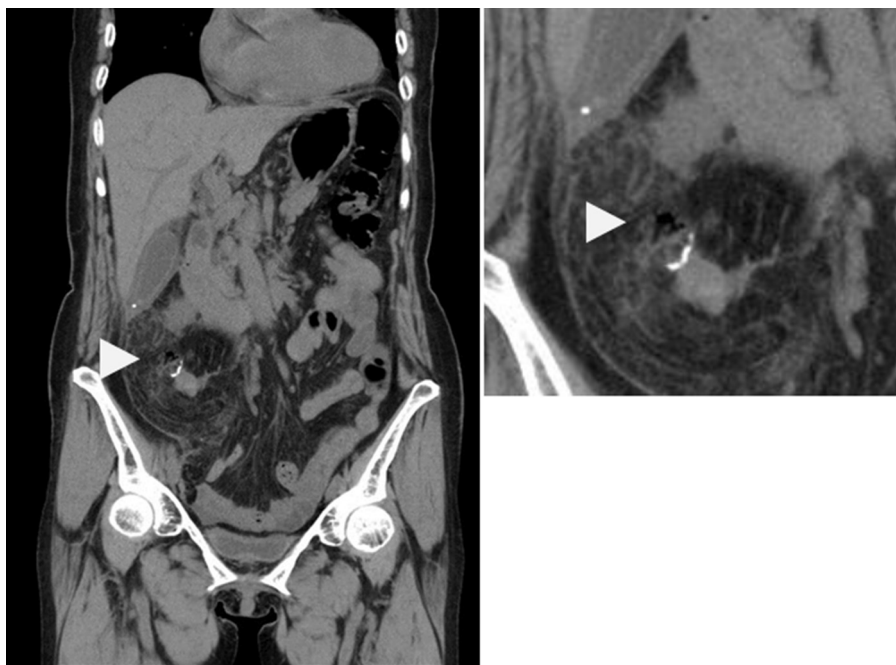


Fig. 3. Computed tomography. Computed tomography shows free air (arrowhead) beside the anastomotic site with the elevated density of fat tissue.

ferred from fever and returned to the hospital on POD 9. She had slight tenderness in the right lower quadrant, but no guarding. Laboratory tests showed white blood cell count (WBC) of 10,500 IU/L (normal 4000–9000/ μ L) and C-reactive protein (CRP) of 22.3 IU/L (normal 0–0.4 mg/dL), with total bilirubin (T-Bil) of 0.4 mg/dL (normal 0.2–1.2 mg/dL), aspartate aminotransferase (AST) of 17 U/L (normal 12–30 IU/L), and alanine aminotransferase (ALT) of 17 U/L (normal 6–30 U/L). Plain computed tomography (CT) showed free air beside the anastomotic site around the elevated density of fat tissue (Fig. 3), a small amount of ascites in the pouch of Douglas, and gallbladder wall thickening with a gallstone. Puncture of the pouch of Douglas showed bloody ascites, with no intestinal fluids.

Suspecting anastomotic leakage with acute cholecystitis, probe laparotomy was performed. Intraoperative observation confirmed hemorrhagic infarction with necrosis of a section of the greater omentum attached to the anastomotic site with a foul smell. However, the torsion point with occlusion of the blood vessels feeding the infarcted greater omentum was not identified (Fig. 4). No leakage was found in the anastomotic site. Edematous thickening of



Fig. 4. Intraoperative observation. Intraoperative observation confirms hemorrhagic infarction with necrosis of the section of the greater omentum attached to the anastomotic site.

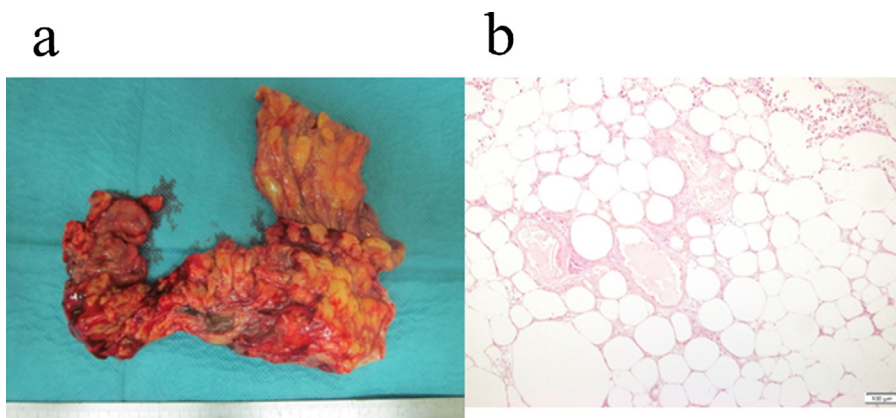


Fig. 5. Resected specimen, hematoxylin and eosin stain.
 a) The torsion point with occlusion of the blood vessels feeding the infarcted greater omentum cannot be identified.
 b) Hematoxylin and eosin stain of the omentum demonstrates fat tissue necrosis.

the gallbladder wall was also observed. Therefore, the necrotic part of the greater omentum was resected, and cholecystectomy was performed. Histopathological examination of the omentum demonstrated fat tissue necrosis and was compatible with omental infarction (Fig. 5). Histopathological examination of the gallbladder was compatible with acute cholecystitis. Preoperative culture of ascitic fluid from the pouch of Douglas was negative. The patient's subsequent course was unremarkable, and she was discharged on POD 10. She has remained well, and no evidence of cancer recurrence has been seen during the 12 months of follow-up without chemotherapy after the surgery for MC of the ascending colon.

3. Discussion

The clinical course of this patient suggests two important clinical issues. First, MC should be distinguished from other more aggressive, non-glandular tumors of the colon, because MC appears to have a better survival outcome than undifferentiated colon adenocarcinoma. Second, omental infarction should be considered in the differential diagnosis of acute abdomen after surgery.

MC is a rare type of colorectal adenocarcinoma that is almost always microsatellite-unstable, and it has a strong association with hereditary non-polyposis colorectal cancer (HNPCC or Lynch syndrome). Thirunavukarasu et al. [1] reported that MCs were rare tumors, constituting approximately 5–8 of every 10,000 colon cancers diagnosed. The mean age at diagnosis was 69 years, with the incidence increasing with age. MCs were twice as common in females, and they were extremely rare in African-Americans. MCs were most common in the proximal colon (74%), where they presented at a later age than in the sigmoid colon.

MC should be distinguished from other more aggressive, non-glandular tumors of the colon because MC appears to have a better survival outcome than undifferentiated colon adenocarcinoma [1,5]. MCs commonly present with Stage II disease, with 10% presenting with metastases. Early outcome analyses have shown that MCs have 1- and 2-year relative survival rates of 92.7% and 73.8%, respectively [1]. MC of the colon can be differentiated from poorly differentiated and undifferentiated colon adenocarcinoma by microsatellite instability, with loss of staining for MLH1 and intestinal transcription factor CDX2. There is also strongly positive calretinin staining compared to other poorly differentiated colonic adenocarcinomas [6]. These epidemiologic and histopathological features are compatible with the findings of this case, and the present patient can be expected to have a good prognosis.

Omental infarction should be considered in the differential diagnosis of acute abdomen after surgery. Omental infarction is primarily caused by torsion of the greater omentum. Torsion of the greater omentum is an uncommon cause of acute abdomen, accounting for 1.1% of all cases of acute abdominal pain [7]. Omental torsion can be classified as primary or secondary, and secondary omental torsion is more common [3]. Secondary torsion is associated with other diseases such as inguinal hernia, tumors in the omentum, cysts, intraabdominal inflammation, and post-surgical wounds or scarring.

Omental torsion is difficult to diagnose preoperatively, because omental torsion could mimic a variety of other acute abdominal conditions. Because the clinical findings are not specific, the differential diagnosis should include acute appendicitis, cholecystitis, cecal diverticulitis, perforated duodenal ulcer, abdominal wall hematoma, and intestinal obstruction [8–10]. Ultrasonography might show a complex mass and a mixture of solid material and hypoechoic zones located between the stomach and transverse colon [8]. Enhanced CT might demonstrate the whirl sign of a fatty mass with characteristic concentric linear strands in the greater omentum [11] and could identify the twisted vessels that contributed to the omental infarction.

This is the first case of omental infarction mimicking anastomotic leakage after surgery reported in the English literature. Preoperative diagnosis of omental infarction was quite difficult in this case. Anastomotic leakage was suspected initially because of the free air beside the anastomotic site and the intense inflammatory response of the ascending colon MC on POD 9. Though the preoperative culture of ascitic fluid from the pouch of Douglas was negative, a gas-forming anaerobic bacteria infection with a foul smell might have been present around the necrotic greater omentum attached to the anastomotic site, confusing the preoperative diagnosis.

4. Conclusions

MC should be distinguished from other more aggressive, non-glandular tumors of the colon, because MC appears to have a better survival outcome than undifferentiated colon adenocarcinoma. Omental infarction should be considered in the differential diagnosis of acute abdomen after surgery.

Conflict of interest

The authors have no conflict of interest to declare.

Funding

None.

Ethical approval

Ethical approval for a case report is not required by our institution.

Consent

Written informed consent was obtained from the patients for the information to be included in our manuscript. His information has been de-identified to the best of our ability to protect his privacy.

Author contribution

Each author participated in writing the manuscript and all agreed to accept equal responsibility for the accuracy of the content of the paper.

Guarantor

Masaki Wakasugi.

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