SAGE Open Medical Case Reports
Volume 11: 1–10

DOI: 10.1177/2050313X231175295
journals.sagepub.com/home/sco
Sage

Intestinal manifestation of Buerger's disease in a middle-age female with subsequent transverse colon perforation: A case report and review of literature

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Abstract

Thromboangiitis obliterans, or Buerger's disease, is a relatively rare nonatherosclerotic, segmental inflammatory and obliterative vascular disease that affects the small- and medium-sized arteries, veins, and nerves. In the acute phase, the lesion presents as an inflammatory, nonsuppurative panarteritis or panphlebitis with vascular thrombosis without necrosis. In the late stage of the disease, the thrombus becomes organized leading to varying degrees of recanalization and subsequent gangrene and amputation. There have been rare reports of thromboangiitis obliterans with involvement of the gastrointestinal trace and even more unusual is the occurrence of this manifestation of disease in women. Here, we report a case of a 45-year-old female patient with a history of thromboangiitis obliterans who presented with ischemic colitis.

Keywords

Thromoboangiitis obliterans, Buerger's disease, gastrointestinal manifestation, colonic perforation, female presentation of thromboangiitis obliterans

Date received: 1 November 2022; accepted: 20 March 2023

Introduction

Thromboangiitis obliterans (TAO), or Buerger's disease, is a non-arteriosclerotic, segmental, progressive, inflammatory vaso-occlusive disorder that primarily affects arteries and veins of small to medium caliber and surrounding neural elements. The disease usually occurs in young male smokers.[1,2] TAO is more prevalent in the Middle East and Far East than in North America and western Europe.[2] The incidence of TAO in women is low[3] and involvement of the gastrointestinal (GI) tract is exceedingly rare. The rise of incidence of TAO in women is attributable to the increased incidence of women cigarette smokers in the past few decades.[3,4] Use of or exposure to tobacco is central to the initiation and progression of the disease.[5,6] The strong correlation with cigarette use is thought to involve a direct idiosyncratic toxicity caused by a component of tobacco or an immune response to the same agents that have modified host vascular wall proteins.[7] Lesions in the brain, heart, and abdominal viscera are rare. TAO differs from other forms of vasculitis in its pathophysiology: the resultant thrombus is highly cellular and inflammatory with relative sparing of the blood-vessel wall with normal acute-phase reactants and autoantibodies.[8] The lesion starts as an inflammatory, nonsuppurative panarteritis or panphlebitis with vascular thrombosis without necrosis. The vascular thrombus becomes organized at the late stages in the disease, leading to varying degrees of minor recanalization of the thrombi with subsequent possible gangrene and amputation. The histopathological

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findings vary according to the duration of disease and findings are most likely to be diagnostic in the acute phase of the disease, as in the end-stage phase of the disease, only an organized thrombus with associated fibrosis is seen.[9–11] Throughout all stages of the disease, the normal structure of the vessel wall, including the internal elastic lamina, generally remains intact, which distinguishes TAO from other types of systemic vasculitis, in which the internal elastic media and lamina are disrupted.[12] Signs and symptoms of the disease are directly proportionate to the degree of vessel and neural involvement and include pain, edema, recurring episodes of thrombophlebitis, claudication, skin changes, tissue malnutrition, and gangrene of the extremity.[13] The Allen test in patients with leg ulceration is used to assess the circulation in the hands and fingers, and an abnormal test is highly suggestive of TAO.[12,14] However, an abnormal result can be indicative of other types of small-vessel occlusive disease of the hands. A complete serological profile with acute-phase reactants and a full antibody and hypercoagulability panel helps to differentiate TAO from these entities. Traditional diagnosis is based on the fulfillment of Shionoya's five criteria: smoking history, onset before the age of 50 years, infrapopliteal arterial occlusive disease, upper limb involvement or phlebitis migrans, and absence of atherosclerotic risk factors other than smoking.[15] The diagnostic criteria of Olin[12] include age below 45 years, current or recent history of tobacco use, presence of distal-extremity ischemia indicated by claudication, pain at rest, ischemic ulcers or gangrenes and documented by non-invasive vascular testing, exclusion of autoimmune diseases, hypercoagulable states and diabetes mellitus, exclusion of proximal source of emboli by echocardiography or arteriography, and consistent arterographic findings in the clinically involved and non-involved limbs. However, these five diagnostic criteria are not universally accepted, and angiographic finding is not pathognomonic. In addition, as there is no specific diagnostic test or serologic finding, the diagnosis of TAO is primarily clinical. Discontinuation of cigarette smoking is the only proven tertiary prevention,[4,16–18] as no forms of therapy are definitive. The rare cases of TAO with involvement of the GI tract have manifested in the small intestine,[19–21] and cases of TAO causing ischemic colitis with perforation are scarce.[22] The majority of confirmed cases of visceral intestinal TAO occur in men[13,19,21–52] (Table 1). We present a unique case of a 45-year-old female patient with a history of TAO who presented with ischemic colitis.

Case report

A 45-year-old Caucasian female with a past medical history significant for Buerger's disease complicated by toe and finger amputations, possible systemic lupus erythematosus/rheumatoid arthritis, chronic obstructive pulmonary disease, peptic ulcer disease, hypothyroidism, and drug and tobacco abuse (one pack per day since her teenage years) presented with altered mental status and was found to be hypoxic and hypotensive. Laboratory work was significant for hypoglycemia, hyponatremia, hypocalcemia, lactic acidosis, and acute kidney injury. The patient was initially diagnosed with Buerger's disease at an outside institution in 2017 with a right second finger wound which required amputation and first presented to our institution with complications of her disease in 2019 after partial amputation of her fingers. At this time, her right wrist brachial index and forearm Pulse Volume Recording (PVR) waveforms indicated mild arterial occlusive disease with non-pulsatile digits and left forearm PVR waveforms indicated mild arterial occlusive disease despite normal wrist brachial index with non-pulsatile digits. Per chart review, the patient endorsed bilateral calf claudication after walking a few hundred feet, relieved by rest. Chest X-ray was significant for pneumoperitoneum. The patient was taken immediately for exploratory laparotomy and was found to have a transverse colon perforation which was repaired primarily. She had other suspicious lesions, so was left open for re-evaluation. On take back, the patient was found to have multiple punctate lesions of full-thickness necrosis every few centimeters throughout the colon, most prominently in the ascending colon (Figure 1). A total abdominal colectomy with end ileostomy was performed given these findings. Upon gross examination, full-thickness transmural elliptical-shaped perforations were noted 12 cm from the proximal margin with nearby areas of ulceration (Figure 2). There were longitudinal areas of dimpling with fibrosis near the anastomotic site (the patient had a previous primary repair of the colonic perforation). Adjacent to the anastomotic site was an area of ulceration with overlying granulation tissue, and several similar punctate ulcers were noted distal to this area. Microscopic examination confirmed the presence of multiple foci of ulceration with granulation tissue as well as scattered medium and small-sized vessel vasculitis, consistent with the patient's known Buerger's disease (Figures 3 and 4). The remainder of the unaffected colon showed signs of early TAO with panarteritis and inflammatory infiltrate, whereas the areas of ulceration with overlying granulation tissue were indicative of end-stage disease. Grocott's methianine silver special stain for fungi was negative and elastic stain highlighted the involved vessels (Figures 5 and 6).

Discussion

TAO, or Buerger's disease, is a segmental, progressive, inflammatory vaso-occlusive disorder that primarily affects small- and medium-sized arteries, veins, and nerves of the extremities in young male smokers. This type of vasculitis differs from other systemic vasculitidies, involving small or medium-sized vessels that can manifest in the colon in its segmental nature and lack of immune

Table 1. Comprehensive comparison of case reports of Buerger's disease with gastrointestinal manifestations.

Paper	Patient demographics (age in years, sex)		Clinical presentation	Physical examination and imaging findings	Gross findings (intraoperative, pathology)	Microscopic description
Herrington and Grossman[13]	33	M	Left lower quadrant abdominal pain and tenderness	Barium enema X-ray showed sigmoid colon constriction 6 cm	Mid sigmoid colon was hyperemic, discolored, edematous, and thickened.	Mucosal ulceration and submucosal hemorrhage in segment of sigmoid colon, numerous thrombi within vessels of submucosa with surrounding inflammatory reaction, submucosal fibrosis. The serosal layer had marked vascularity and hyperemia.
Herrington and Grossman[13]	42	M	Abdominal pain, anorexia, weight loss	Abdomen distended, bilateral lower extremities were edematous with areas of pigmentation	500 cc of straw-colored fluid in the peritoneal cavity, numerous thrombi scattered in the terminal mesentery of small and large intestine	Segmental ulceration with intense inflammatory reaction in the submucosa with thrombosis of the submucosal vessels with fresh submucosal hemorrhage, hyaline and fibrinoid changes in small vessels; thrombi in small vessels of accompanying mesentery
Sachs et al.[24]	29–32	M	Multiple instances of bowel obstruction	Bowel obstructed	Necrosis of mid-jejunum, gangrene of six inches of terminal jejunum and upper ileum, bowel infarction	Thromboangiitis obliterans
Sobel and Ruebner[25]	45	M	Constant left upper quadrant abdominal pain, intermittent claudication non-walking and anorexia with a 14 kg weight loss in the past year	Cachexia, left upper quadrant abdominal tenderness, femoral bruits bilateral, weak left popliteal pulse and absent left posterior tibial pulse, barium enema showed circumferential narrowing of the distal transverse colon	Segment of transverse colon with a 9 cm area of narrowing with ulcerated mucosa	Large segments of mucosal ulceration with acute and chronic inflammation; arterial branches occluded by organized and recanalized thrombi within areas of ulcerations; arterial thrombotic occlusions seen in areas free of mucosal ulceration in a large artery of mesocolon and of mesoappendix; small venules were occluded by recent thrombi; no atheromatous changes and internal elastic lamina was well preserved with mild, focal perivascular fibrosis.
Fakour and Fazeli[26]	35	M	Gangrene of left foot	Severe stasis changes with ulcerations on lower extremities, digital cyanosis bilaterally, large hemorrhagic effusion with clots in the peritoneal cavity with necrotic material in the pancreas, spleen, and loops of small bowel	Mucosa of the gastrointestinal tract was diffusely congested without focal ulceration or infarction	Posterior tibial artery had occlusion by fibrous tissue and mild inflammatory infiltrate, internal elastic was partially destroyed
Rosen et al.[29]	48	M	Dull, colicky suprapubic pain worse on coughing and moving associated with anorexia	Pain shifted to right iliac fossa, generalized abdominal tenderness maximal at the right iliac fossa with guarding and rebound tenderness	Malodorous peritoneal fluid, 8 cm infarcted sigmoid colon on the verge of perforation	Infarction of all layers of the wall of the sigmoid colon, thrombosed vessels present throughout the necrotic segment; larger arteries and veins were thrombosed with infiltration of neutrophils; marked perivascular infiltrate of neutrophils, plasma cells, and lymphocytes; perivascular fibrosis and marked intimal thickening of some vessels.

(Continued)

Table 1. (Continued)

Paper	Patient demographics (age in years, sex)		Clinical presentation	Physical examination and imaging findings	Gross findings (intraoperative, pathology)	Microscopic description
Enshaei et al.[33]	31	M	Dysentery with 12 kg weight loss	Colonoscopy showed diffuse superficial ulcerations, linear or aphtoid, with deep ulceration of the sigmoid colon; cecum appeared shrunken like a radicle	2 cm diameter perforation of sigmoid colon	Nonspecific inflammatory reaction; partial or total venous and arterial thromboses, vascular lumens were occluded, one with histiocytic granuloma with rare giant multinucleated cell[8]
Bouomrani et al.[34]	39	M	Acute abdominal pain for 8 h	Muscle guarding, small bowel ischemia	Ischemic gangrene of distal ileum, multiple perforations of small bowel 3–4 cm from each other	Ischemic perforations on anti-mesenteric sides and showed terminal branches of mesenteric arteries with thrombosis, and they were swollen and infiltrated with neutrophils and thickening of the intima.
Bouomrani et al.[34]	42	M	Recurrent duodenal ulcer for 5 years	Persistent epigastric pain with endoscopy showing a rounded duodenal ulcer of 5 mm with congestive gastritis and superficial ulcerations of pre-pyloric antrum	5 mm duodenal ulcer	Deep ulcerations associated with microthrombosis and absence of Helicobacter pylori, deep and digging duodenal ulcer associated with multiple microthrombosis and polymorphic inflammatory infiltrate (predominantly lymphocytic)
Kamiya et al.[36]	48	F	Acute abdominal pain for 6 h	CT showed occlusion of the SMA and multiple kidney infarction with thrombus floating in the thoracic aorta, with neither aortic aneurysmal nor atheroma lesions	Acute obstruction of the SMA	Mucosal layer of the small intestine was extensively necrotic and villous structures were left out. Prominent congestion and edema were observed in the submucosa. There were not observed any finding of vasculitis.
Lee et al.[37]	65	M	Periumbilical and right lower quadrant pain for 2 months, worsening with development of constipation and abdominal distension for 3 days	Mild abdominal distension, diffuse pain upon palpation, hyperechoic bowel sounds, CT with marked intestinal distension with segmental bowel wall thickening and pericolic haziness in sigmoid colon	Marked luminal narrowing without any mass lesion	Prominent vascularization of the media and infiltration of inflammatory cells with intact normal architecture
Lee et al.[37]	39	M	Right lower quadrant pain and bloody stool for 5 days	Diffuse abdominal pain on palpation, decreased bowel sounds	Sigmoidoscopy showed circumferential ulceration and hard coated exudates with mucosal edema, segmental involved and clear distinction between normal and lesion in the proximal sigmoid colon	N/A, treated conservatively

(Continued)

Table 1. (Continued)

Paper	Patient demographics (age in years, sex)		Clinical presentation	Physical examination and imaging findings	Gross findings (intraoperative, pathology)	Microscopic description
Cho et al.[41]	37	M	2 days of diffuse abdominal pain and 2 months of claudication of his left hand	Rigid, tender abdomen, diminished bowel sounds, no palpable pulses of the left radial artery	Small intestine was extensively infracted, from the distal jejunum to the proximal ileum, and thrombi were seen in the small, distal mesenteric arteries and veins; small bowel specimen was involved by extensive full-thickness necrosis and occluded small- and medium-sized mesenteric arteries and veins	Mesenteric arteries were swollen and infiltrated with neutrophils in all three layers, but there was no sign of fibrinoid necrosis, lumen occluded by a highly cellular thrombus with the appearance of a micro abscess
Kobayashi et al.[42]	42	M	Acute abdominal pain 7 months s/p thrombectomy	Bilateral femoral artery pulses were hardly palpable	Ileum end, cecum, and proximal side of the ascending colon and sigmoid colon were necrotic	Small intestine demonstrated ischemic ulcers, and capillaries of the ischemic intestine exhibited thickening of the intima and fresh thrombus formation, with some inflammatory cells. The marginal arteries and veins had well-preserved architecture
Cho et al.[43]	38	M	Obstipation and diffuse abdominal pain for 5 days, nausea, vomiting	Rigid, tender abdomen and diminished bowel sounds	Small amount of serosanguinous fluid in peritoneal colon, small intestine, from proximal jejunum (about 15 cm distal to the Treitz ligament) to ileum (about 20 cm proximal to the ileocecal valve), showed multifocal necrotic and hemorrhagic patches on the wall; small bowel specimen showed contracted, indurated, and occluded small- and medium-sized mesenteric arteries and veins	Recanalization, organization of the thrombus, and a fibrous thickening of the all layers of arterial wall were present; mild lymphoproliferative cell infiltration and hemorrhage were also observed in the vessel wall; internal elastic membrane was relatively intact and the general architecture of the vessel wall was well preserved; the hallmarks of early stage disease such as granulomatous foci, microabscesses, mixed inflammatory cell infiltrate or giant cells were not seen.
Kurata et al.[44]	35	M	Sudden onset abdominal pain	Hypotensive, X-ray with abdominal free air	Generalized peritonitis caused by two perforated ulcers in the ileum, 9 and 13.5 cm proximal to the ileocecal wall	Typical ulcers with purulent exudates and granulation tissue formation. Small-sized ulcers and one of the middle-sized ulcers were shallow; however, other ulcers were deep. The two perforation sites were confirmed by histology. Initial routine histopathological examination revealed only one occluded artery beneath an ulcer. However, additional examinations with serial sections demonstrated that at least five ulcers were accompanied with occluded arteries beneath in the submucosal layer, the muscularis propria, and the subserosal layer. Recanalization vessels were found in the lumen of those arteries.

(Continued)

Table 1. (Continued)

Paper	Patient demographics (age in years, sex)		Clinical presentation	Physical examination and imaging findings	Gross findings (intraoperative, pathology)	Microscopic description
Iwai[47]	43	M	Intermittent claudication for 2 years, cold fingers, abdominal pain after eating, weight loss	Angiography revealed stenotic lesion of the right popliteal artery, slight dilatation in midpopliteal region, occlusion of posterior tibial and peroneal arteries, celiac trunk occlusion, 95% stenosis near origin of SMA	Stenosis of SMA	intimal hyperplasia of the mesenteric artery
Michail et al.[48]	42	M	Acute abdominal pain and vomiting	X-ray with small bowel obstruction with free air under the diaphragm, normal colonoscopy, periumbilical pain	Rupture of the terminal ileum	Small bowel segment showed 10 cm long area with congestion, thinning, and deep ulceration of the wall; chronic ulceration penetrating the bowel wall in setting of chronic ischemic bowel disease; medium-sized mesenteric arteries and veins showed mild inflammatory infiltration and many were occluded and recanalized; inflammatory cells throughout media and adventitia, but no necrotizing lesions in media; vessel wall was generally well preserved and no arteriosclerosis was detected

CT: computed tomography; SMA: superior mesenteric artery.

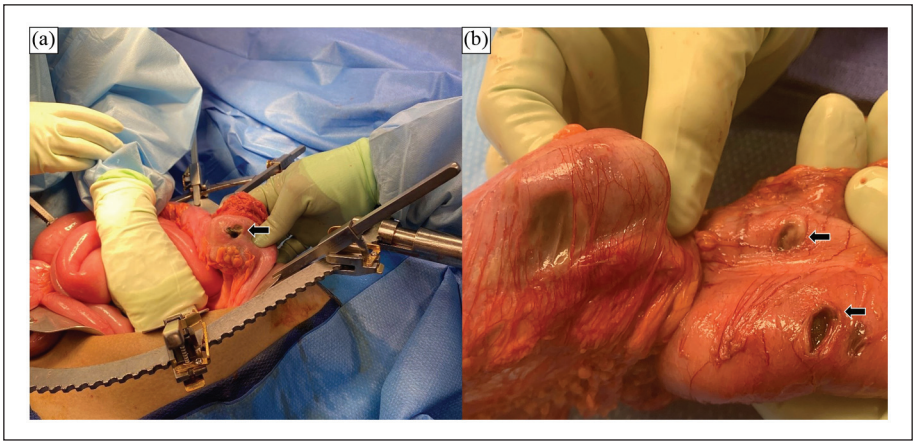

Figure 1. (a, b) The patient was found to have multiple punctate lesions of full-thickness necrosis every few centimeters throughout the colon, most prominently in the ascending colon (black arrows).

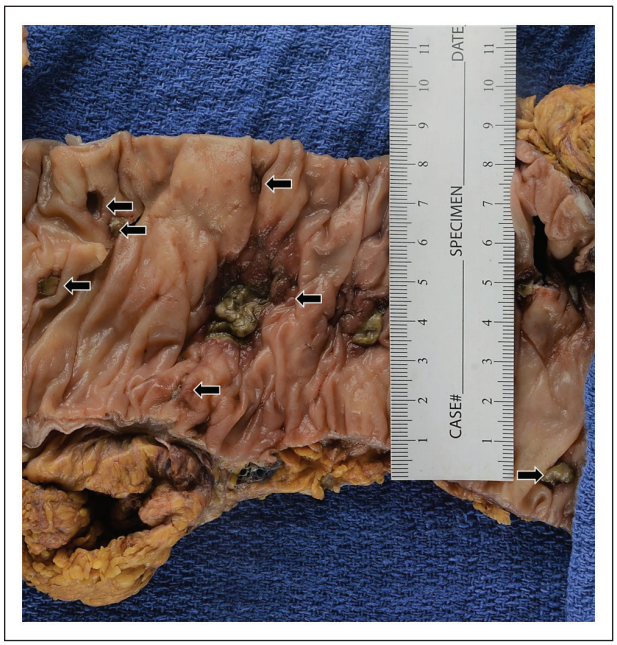

Figure 2. Macroscopic examination after formalin fixation reveals transmural elliptical-shaped perforations were noted 12 cm from the proximal margin with nearby areas of ulceration (black arrows).

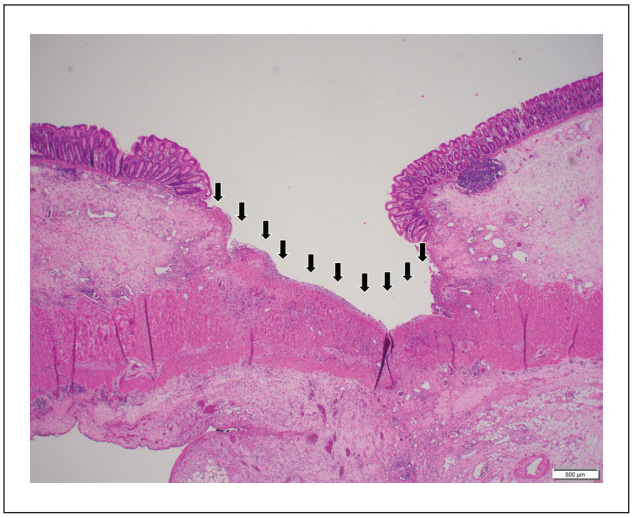

Figure 3. Focus of ulceration with granulation tissue (black arrows).

complexes or vascular wall involvement. Histologically, systemic vasculitis presents as an inflammatory infiltrate in the wall of dermal or subcutaneous vessels, which may be neutrophilic, lymphocytic, or granulomatous mediated by immune complexes. Often, there is microscopic red blood cell extravasation and variable fibrinoid necrosis of vessel walls as well secondary changes in overlying epidermis and sweat glands. The development of TAO in the acute phase is characterized by a lesion that presents as an inflammatory, nonsuppurative panarteritis or panphlebitis with vascular thrombosis without necrosis. The thrombus becomes organized in the late stage of the disease leading to varying degrees of recanalization and consequent gangrene and amputation. The incidence of TAO in women is low[3] and involvement of the GI tract is exceedingly rare. The rare cases of TAO with involvement of the GI tract have manifested in the small intestine,[19–21] and cases of TAO causing ischemic colitis with perforation are scarce,[22] and the majority of confirmed cases of visceral intestinal TAO occur in men.[13,19,21–32,34–52]

Our patient is unique with features of TAO. While only the upper extremities were reported to be affected and we do not have the full diagnostic workup to support the provided history of TAO, given the histology and fulfillment of Olin's criteria, we can attribute the patient's diagnosis to Buerger's disease after excluding other causes of her vasculitis.

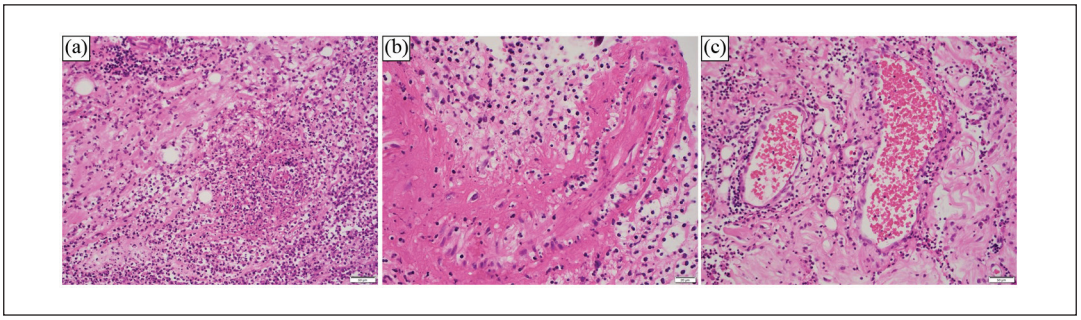

Figure 4. (a) Vasculitis (20× magnification), (b) arteritis (40× magnification), and (c) venulitis (40× magnification).

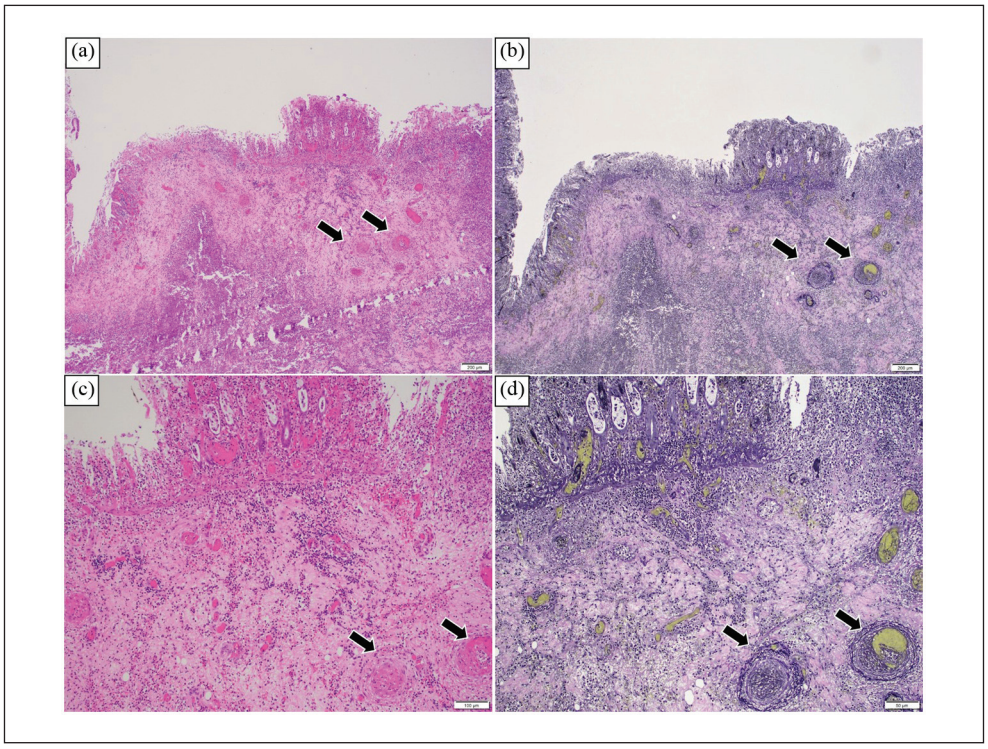

Figure 5. (a, c) Mucosal ulcer with vasculitis (black arrows, 4× and 10× magnification, respectively). (b, d) Elastic special stain highlights the involved vessels (black arrows, 4× and 10× magnification, respectively).

However, she may have suffered from another unknown vasculitis which mimicked TAO. The patient has an unconfirmed medical history of two inflammatory and autoimmune diseases (rheumatoid arthritis and lupus) and a history of intravenous (IV) drug abuse and tobacco use with transverse colon perforation with subsequent septic shock. However, the histological findings and serological studies support TAO as the cause of this patient's ischemic colitis rather than other types of systemic vasculitis given the intact elastic lamina, the lack of serological findings and immune complexes, the segmental nature, and lack of necrosis. Given the brief interval between surgeries, the patient had few serosal adhesions or areas of advanced ischemia that could have led to the multifocal ischemic colitis. The patient had an open wound that could have contributed to her initial clinical presentation. She is one of the first reported female patients with TAO with intestinal manifestation and subsequent colonic perforation. The patient passed away 2 months after the surgery from septic shock.

Conclusion

This case demonstrates the importance of clinical correlation with gross findings especially in rare presentation of a disease.

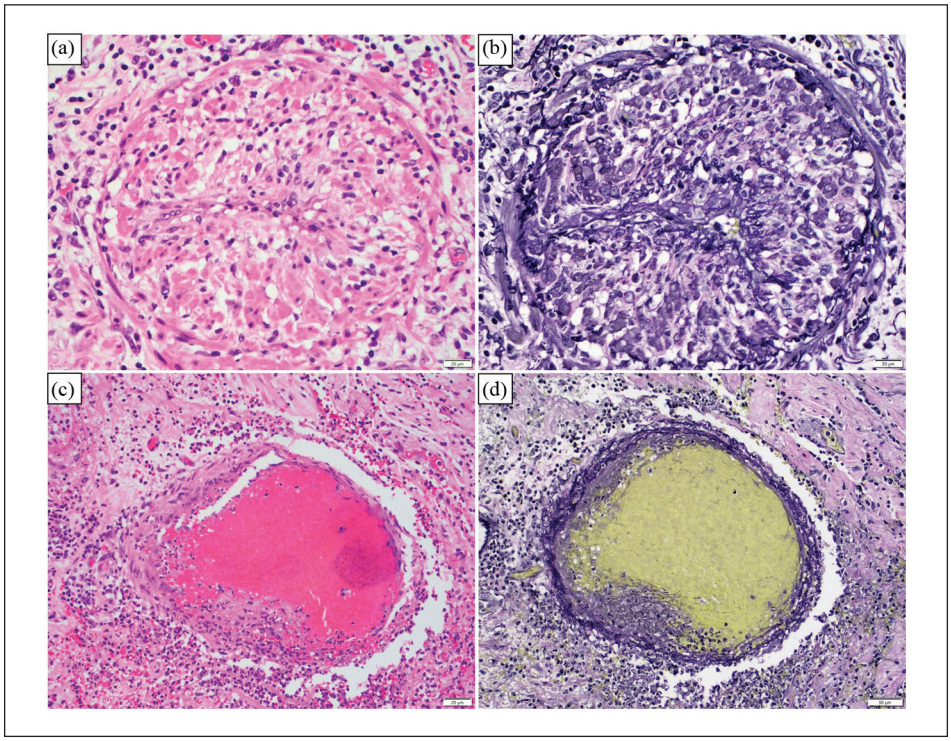

Figure 6. (a) Vasculitis (arteritis) almost occluded (40× magnification) with elastin staining shown in (b, c). Venulitis (20× magnification) with elastin staining shown in (d).

Acknowledgements

The authors thank the surgical team for their contributions.

Author contributions

M.B. performed the macroscopic examination of the resected specimen and drafted the manuscript. R.D.B. performed the surgery and edited the manuscript. S.A.D. confirmed the histologic diagnosis and led the team.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed consent

Written informed consent was obtained from a legally authorized representative(s) for anonymized patient information to be published in this article.

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