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Multiple “Apple-Core” Lesions in Chronic Ischemic Colitis: The Great Masquerader

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To the Editor:

Chronic ischemic colitis (CIC) is a rare disease, attributed to splanchnic atherosclerosis. Although stenotic lesions of the mesenteric circulation are common, the development of collateral circulation rarely allows progression to clinically significant colitis. The classical history (long-standing postprandial abdominal pain, weight loss, upper abdominal soufflé) is present in a minority of patients and diagnosis is mostly made based on supportive angiographic evaluation of the splanchnic circulation. We present a case of CIC due to extensive atherosclerotic disease of the superior mesenteric artery (SMA), which presented as a subacute diarrheal illness with “apple-core” lesions on imaging; an unusual, presentation masquerading as inflammatory and/or infectious colitides.

A 74-year-old Hispanic female from Mexico presented with a 6-week history of diarrhea associated with intermittent hematochezia and multiple ascending colonic “apple-core”-like strictures on a computerized tomography scan (Figure 1a–c). Past medical history was significant for a ~60 pack-year history of tobacco smoking and untreated systemic hypertension. She denied any coronary artery disease, chronic kidney disease, cerebrovascular or peripheral vascular diseases. Colonoscopy revealed three strictures (Figure 1d–i, stars), the largest (~10 cm) was just distal to the hepatic flexure (Figure 1d–f); and featured pseudopolyps. There were shallow, geographic ulcers in the ascending colonic segments between these strictures (Figure 1h). The terminal ileum (Figure 1j), and the transverse, descending and sigmoid colon appeared normal. The clinical impression was infectious or inflammatory colitides. Colonic biopsies showed sharp demarcation between normal colonic mucosa and adjacent atrophic glands (Figure 1k and 1), architectural distortion, reparative changes and ulceration with rare crypt abscesses (Figure 1m), consistent with a diagnosis of ischemic colitis. Amoebae, mycobacteria, and granulomatous inflammation were not identified.

Multiplanar reconstruction of the SMA and the celiac artery from the initial computerized tomography scan revealed dense calcification and critical stenosis at the origin from the

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CONFLICT OF INTEREST

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aorta. A computerized tomographyangiography and a mesenteric angiogram with selective catheterization of the celiac artery confirmed the complete occlusion of 1.7 cm of the proximal SMA and distal reconstitution via pancreaticoduodenal arcade collaterals. Because these extensive calcified atheromas were predicted to compromise the vascular supply in the identical segmental distribution corresponding to those affected in our patient, these lesions were attributed as the cause of ischemic colitis. Revascularization of the bowel was achieved by surgical placement of two (aorta-celiac and aorta-SMA) Dacron bypass grafts across the stenotic segments, and the patient had an uneventful recovery. A follow-up colonoscopy at 1 year showed healing of all ulcers and partial resolution of all strictures.

Three possible clinical presentations of vascular occlusion have been described to date (1–5): (i) rapid occlusion of a major blood vessel can cause catastrophic presentation with massive bowel necrosis; (ii) slow occlusion with formation of adequate collateral circulation, where infarction may not occur and complete healing is possible, and (iii) blood flow is adequate to prevent death of the intestine but recovery is incomplete with formation of fibrotic strictures. Our patient presents a fourth variation within this spectrum, manifested with multiple, reversible colonic strictures in a subacute clinical presentation, masquerading as infectious colitides (6). This case also underscores the importance of considering CIC among other differentials for ascending colonic strictures (for example, inflammatory bowel disease (7), nonsteroidal anti-inflammatory drug-induced (8) and infectious colitides due to amebiasis (9) and mycobacteria (10)). CIC is frequently associated with generalized atherosclerotic disease affecting cerebral, coronary, renal, and peripheral vascular beds (11,12). A complete arteriographic evaluation is necessary in ischemia of the colon; particularly assessment of atherosclerotic changes at or near the ostia of the major visceral arteries and the vascular arrangement at the Griffiths' point (13,14). Left untreated, CIC would progress to acute mesenteric ischemia due to progressive atherosclerosis and thus, surgical revascularization is often warranted and is the preferred therapeutic modality.

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Figure 1:

Chronic ischemic colitis presenting as multiple “apple-core” lesions on imaging.

(a) Computerized tomographic images of the largest pseudo-“apple-core” stricture (arrows) involving the transverse colon just distal to the hepatic flexure. Multiplanar reconstruction of the ascending colon demonstrates the second (b, arrow) and third (c, arrow) strictures.

(d) Schematic illustration depicting the size and distribution of all the strictures in the colon, numbered 1 – 3. (e and f) Colonoscopy revealed a long-segment stricture at the hepatic flexure, featuring inflammatory pseudopolyps (f, arrowhead), and two additional short-

segment strictures (g and i, stars) involving the proximal ascending colon. The terminal Ileum (j) appeared normal. Shallow, geographic ulcers with surrounding erythema (f– i, arrowhead in h) were seen in the ascending colon interspersed between the strictures as well as within the lumen of the structures. Light microscopy of hematoxylin- and eosin-stained colonic biopsies examined under $\times 10$ (k) and $\times 40$ (l) magnification showing sharp demarcation between normal colonic mucosa and adjacent atrophic glands. Visualization of

the ulcer bed at $\times 40$ magnification revealed the presence of architectural distortion, reparative changes with rare crypt abscesses (m, arrowhead).

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