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Case Report

Lymphogranuloma venereum (LGV) proctocolitis mimicking rectal lymphoma

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ABSTRACT

Lymphogranuloma venereum is a sexually transmitted infection caused by serotypes L1-3 of *Chlamydia trachomatis* and may present as hemorrhagic proctocolitis. The diagnosis of an active infection is difficult to establish, as confirmatory testing can be unreliable or unavailable. Imaging findings can be nonspecific and mimic malignancy or other chronic infectious and inflammatory disorders. In this report, we present a case of lymphogranuloma venereum proctocolitis and its computed tomography features to highlight the relevant imaging findings and importance of timely diagnosis.

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Introduction

Isolated proctitis usually presents with rectal urgency, tenesmus, dyschezia, and/or mucopurulent discharge. When inflammation spreads to the colon beyond the distal 10-12 cm, symptoms of colitis manifest, including abdominal pain or cramping, bloating, and diarrhea [1–3]. If inflammation is severe, it can lead to significant rectal wall thickening, masses, or lymphadenopathy. The differential diagnosis for proctocolitis includes infectious, inflammatory, ischemic, or neoplastic processes [1]. Lymphogranuloma venereum (LGV) is an uncommon cause of proctocolitis, and delay in diagnosis can lead to morbidity. The purpose of this case report is to increase radiologists' awareness of LGV and its imaging characteristics to aid in timely diagnosis and appropriate management.

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Fig. 1 – (A) Axial image from contrast-enhanced CT of the abdomen and pelvis at the level of the femoral heads demonstrating circumferential wall thickening of the rectum (black arrows) and perirectal fat stranding and induration (curved arrow). (B) Axial image of contrast-enhanced CT at the level of the low rectum demonstrating rectal wall thickening (black arrows), submucosal edema (right angle arrow) and right-sided perirectal lymphadenopathy (black arrow heads).

Case report

A 23-year-old male presented with 1 day of profuse rectal bleeding. He reported 3 months of "blood streaked stool," 1 month of night sweats, and 1 week of dyschezia and tenesmus. He denied fevers, urinary symptoms, weight loss, joint pain, or rashes. He had presented to the emergency department 2 weeks earlier where a CT scan demonstrated irregular rectal wall thickening and multiple enlarged perirectal and left iliac lymph nodes (Fig. 1). He was told that these findings were "concerning for lymphoma," and urgent outpatient follow-up was arranged.

The patient's medical history was notable for human immunodeficiency virus (HIV) with poor medication adherence and low-grade anal dysplasia, for which he was previously lost to follow-up. He was known to be an asymptomatic chlamydia carrier without confirmed eradication and reported unprotected sexual intercourse with a male HIV-positive partner. He had no family history of colon cancer or inflammatory bowel disease (IBD).

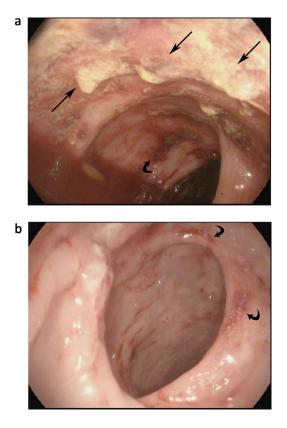


Fig. 2 – (A) Areas of discontinuous, ulcerated mucosa (arrows) with stigmata of bleeding (curved arrows) in the descending colon and (B) rectum.

On hospital admission, he was found to have a palpable, tender mass on rectal exam. Laboratory tests were notable for new anemia and serum positivity for HIV and syphilis. Tests for *Neisseria gonorrhoeae*, *Chlamydia trachomatis*, herpes simplex virus, fecal bacteria, and parasites were negative.

Given the concern for malignancy, a flexible sigmoidoscopy was performed which showed red blood as well as discontinuous areas of ulcerated mucosa from the rectum to the descending colon (Fig. 2). Microscopic examination revealed focal active colitis with superficial erosion. Immunohistochemistry for syphilis, cytomegalovirus, and adenovirus was negative. There was no chronicity to suggest a diagnosis of IBD. Given these findings and presumed bacterial etiology, empirical antibiotic treatment was initiated.

The patient experienced additional hematochezia, prompting a colonoscopy 5 days after the initial procedure. Rectal findings were substantially improved, notable only for mild proctitis with a single ulcer (Fig. 3). Biopsies of the rectum demonstrated acute colitis with ulceration. Endoscopic and histologic findings of the rest of the colon showed no evidence of active or chronic inflammation.

Given the rapid clinical, endoscopic, and histologic response to empirical therapy, the patient was diagnosed with stage II LGV and completed 21 days of antibiotic therapy. It was recommended that his partner be screened for sexually transmitted infections. In follow-up 2 months later, he was without

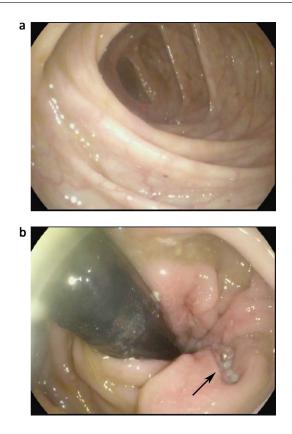


Fig. 3 – After treatment, (A) normal colonic mucosa was visualized throughout the colon, while only (B) mild proctitis with a single area of ulceration (arrow) remained.

hematochezia or pain, and tests for syphilis, gonorrhea, and chlamydia were negative.

Discussion

This patient presented with symptoms consistent with proctocolitis with imaging findings of rectal wall thickening which were initially concerning for malignancy. However, the clinical history combined with an endoscopic and histologic evaluation suggested an infectious etiology, which led to the diagnosis and successful treatment of LGV proctocolitis.

LGV is caused by the L1, L2, or L3 serotypes of *C* trachomatis. This disease may be under recognized, as LGV is not always reportable and symptoms may be absent or mild, which promotes the spread and progression among at-risk individuals. As such, numerous outbreaks of LGV proctitis and proctocolitis have been identified in North America and Europe since 2003, particularly in HIV-infected men who have sex with men [4–6]. Therefore, it is useful for radiologists to consider this condition in the differential diagnosis of proctitis and proctocolitis in the appropriate clinical context so that patients can be appropriately treated and to decrease risk of disease spread.

Inoculation via the rectum rarely results in the pathognomonic "groove sign" for LGV, characterized by swollen, unilateral lymph nodes that form along the inguinal ligament. Rather, upon exposure by anogenital contact, the patient may notice a transient, painless ulcer at the site of infection but otherwise may only become symptomatic if hemorrhagic proctocolitis develops [2,4].

Initial testing during an acute presentation of proctocolitis can be nonspecific or misleading, and so the diagnosis requires a high index of suspicion from the clinician and radiologist. CT characteristics of LGV proctocolitis are circumferential rectal wall thickening, perirectal fat stranding, submucosal edema, enlargement of seminal vesicles, and pelvic and/or retroperitoneal lymphadenopathy [6]. Furthermore, CT multiplanar reformations help evaluate for chronic complications of untreated disease such as ischioanal or supralevator abscesses, pelvic fibrosis, anogenital strictures, and fistulae [7-9]. In contrast to LGV proctocolitis, colorectal carcinoma can have more focal, irregular wall thickening rather than long-segment circumferential wall thickening. Carcinoma also can spread beyond the rectal serosa. Colorectal lymphoma can affect longer and multiple colonic segments. Although fistulae and strictures can occur late in LGV, they are more commonly seen with IBD. Crohn's disease may have other segments of gastrointestinal tract involvement, whereas LGV is limited to the rectum and sometimes the sigmoid. Concerning other forms of infectious colitis, location is helpful. Salmonella, Yersinia, tuberculosis, and amebiasis affect the right colon, whereas schistosomiasis, shigellosis, herpes, gonorrhea, and syphilis preferentially affect the left colon. Pseudomembranous colitis, cytomegalovirus, and Escherichia coli can be a pancolonic process. Ischemic colitis follows a vascular distribution and can lead to pneumatosis. Diverticulitis by definition includes the presence of diverticulosis.

Unfortunately, while nucleic acid amplification testing on anorectal samples is the preferred method to identify LGV, this testing has not been approved by the FDA and is not widely available [3–4]. Serologic testing is available but difficult to interpret, as the results cannot differentiate between current versus prior infection, may not be serovar specific, and have not been validated for rectal infections [2–5]. Colonoscopy can help rule out malignancy, but may demonstrate nonspecific friable and ulcerated mucosa with histologic findings that mimic IBD, including lymphocytic infiltrates, crypt distortion or abscesses, or granulomatous changes [2].

In summary, a variety of pathologies can cause proctocolitis and CT findings of colorectal wall thickening and adjacent fat stranding are nonspecific. LGV is an increasingly important cause of infectious proctocolitis that can mimic other forms of infectious colitis and neoplasms. However, with increased awareness of more specific imaging findings for LGV, such as submucosal edema and seminal vesicle enlargement, the radiologist can help refine the differential diagnosis and the clinical management algorithm. This is especially important as confirmatory testing can be unreliable or unavailable, and so early treatment of a presumptive diagnosis may be required.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.radcr.2018.08.015.

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