CASE REPORT

Rectal bleeding caused by a syphilitic inflammatory mass

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SUMMARY

A 47-year-old man presented with fatigue, decrease appetite, abdominal pain and rectal bleeding. His colonoscopy revealed a single, firm, raised, centrally ulcerated mass at the anorectal junction. During this same admission, he was diagnosed with HIV and syphilis, found to have multiple hepatic lesions and positive cerebrospinal fluid venereal disease research laboratory test (VDRL). Biopsies from both the hepatic lesions and rectal ulcer showed spirochaetes by immunostaining. The initial presentation was felt to be secondary to a rectal inflammatory mass caused by syphilis.

BACKGROUND

The prevalence of sexually transmitted diseases (STDs) continues to rise, with men who have sex with men (MSM) constituting a subgroup particularly at risk for these infections.¹ STDs involving the rectum have been well described, with *Chlamydia trachomatis* and *Neisseria gonorrhoeae* representing the most commonly implicated pathogens.² Although rare, syphilis should be considered as a cause of rectal lesions, especially in MSM and immunodeficient patients. The natural course of syphilis may differ in this specific population with more frequent overlap between the different stages, a less linear progression of the disease and occasionally with presence of atypical findings.³

CASE PRESENTATION

A 47-year-old man presented to the emergency department in October 2015 with a history of fatigue, decrease appetite, abdominal pain and rectal bleeding for 1 month. He described his fatigue and anorexia to have started insidiously, progressively limiting his functional capacity. Associated with this he also developed right-upper-quadrant abdominal pain that was intermittent, crampy in quality, moderately severe and not associated with food intake. Approximately 2 weeks after these symptoms started, the patient developed rectal bleeding. He described these episodes as red blood mixed with stool, not associated with constipation, retching or tenesmus; episodes were painless occurring approximately 5–6 times a week. He denied other associated symptoms including nausea, vomiting, or diarrhoea. He admitted to 8-pound weight loss in the month prior to presentation to the hospital.

The patient had a history of unprotected anoreceptive intercourse with multiple sexual partners,

no known HIV contact. He was last tested for HIV approximately 9–10 years ago at a walk-in clinic and was told that the test was negative. He denied any medical follow-up since other than an acute event in 2011 when he was admitted for gangrenous appendix requiring surgical intervention. He denied any history of STDs or genital lesions. He was a former smoker, consumed alcohol socially and denied illicit drug use. His mother had passed from colon cancer 1 month prior to these events.

On admission, his temperature was 39.4°C, blood pressure 134/69 mm Hg, pulse 90 beats/min and oxygen saturation 97% on room air. His abdominal examination was pertinent for normal bowel sounds, firm right and left upper quadrants, with palpable regular mass extending from left upper quadrant to left flank, epigastric area and right-upper-quadrant. Digital rectal examination was essentially normal, with no masses or irregularities felt, no condylomata lata, small amount of blood on finger. The patient had no rash and no lymphadenopathy.

INVESTIGATIONS

Patient's initial blood work showed haemoglobin of 8.5 g/L (normal range 13.5–17.6 g/L), white blood cell count of 3.4×10^9 (normal range $4.5\text{--}11.0 \times 10^9$), platelet count of 37×10^9 (normal range $150\text{--}450 \times 10^9$), a hepatic function panel was normal with normal alkaline phosphatase.

An abdominal CT showed several hypodense hepatic lesions, marked splenomegaly and left lateral wall thickening of the distal rectum (figure 1).

Colonoscopy was scheduled and revealed a single, firm, raised, centrally ulcerated mass at the anorectal junction measuring approximately 3 cm. Ulcer was noted to have irregular borders, friable, with active bleeding. The scope was advanced around the entire length of the colon into the cecum without any other mucosal abnormalities identified. Multiple biopsies were taken from centre and periphery of the ulcerated mass (figure 2).

alpha-fetoprotein (AFP) and carcinoembryonic antigen (CEA) were low, 2.0 ng/mL (normal range <10.0 ng/mL) and <0.50 ng/mL (normal range <3.0 ng/mL), respectively. During his stay he tested positive for HIV with an absolute CD4 count of 67 cells/mL (normal range 500–1500 cells/mL) and viral load of 75 000 copies/mL. Further testing revealed positive fluorescent treponemal antibody absorption (FTA-ABS) and positive VDRL with a titre of 1:512. QuantiFERON was negative. Rectal *C. trachomatis* and *N. gonorrhoeae* tests were

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Figure 1 An abdominal CT scan showing hepatic lesions (arrow 1), splenomegaly and rectal wall thickening (arrow 2).

negative. Patient tested positive for hepatitis A and had negative serologies for hepatitis B and C. Patient also underwent ultrasound guided liver biopsy to evaluate liver lesions identified on CT scan. Finally, a lumbar puncture was performed with cerebrospinal fluid (CSF) glucose levels of 53 mg/dL (normal range 45–80 mg/dL) (and plasma/CSF glucose ratio of 3.7), protein of 51 mg/dL (normal range 15–45 mg/dL), 3 red blood cells/mm³

Figure 2 A colonoscopy with direct visualisation of the rectum demonstrating ulcerated mass.

Figure 3 Biopsy of liver mass (H&E).

(normal range 0–5 cells/mm³), 0 white blood cells/mm³ (normal range 0–5 cells/mm³), and positive CSF VDRL.

Biopsies from the hepatic lesion (figure 3) and rectal mass (figure 4 and figure 5) showed architectural distortion with granulomatous inflammation and observable spirochaetes by immunostaining. No evidence of neoplastic disease.

DIFFERENTIAL DIAGNOSIS

In our case, the differential diagnosis of a rectal mass in HIV positive patient included colorectal cancer, lymphoma, Kaposi's sarcoma, cat scratch disease, *C. trachomatis* infection, solitary rectal ulcer syndrome.

TREATMENT

While in hospital patient was started on IV benzylpenicillin (4million units intravenously) every 4 hours for 10 days for treatment of neurosyphilis and on highly active antiretroviral therapy for treatment of HIV.

OUTCOME AND FOLLOW-UP

Patient was followed at our HIV clinic. He received penicillin G benzathine (2.4million units intramuscularly) once weekly for

Figure 4 Biopsy of rectal mass (H&E).

Figure 5 A rectal mass biopsy demonstrating spirochaetes by immunostaining (demonstrated by arrows).

3 weeks. His traceable partners were contacted for testing and treatment.

On follow-up visit's patient mentioned improvement in constitutional symptoms and no other episodes of rectal bleeding. No palpable splenomegaly on follow-up examinations. Repeat endoscopic evaluation performed at 6 weeks after completion of antibiotic therapy revealed completely healed rectal mucosa with negative immunostaining on biopsy specimens. At 6 months follow-up, the repeat plasma VDRL titre was 1:32.

DISCUSSION

Syphilis has classically been described as a condition that progresses through various stages of infection. Primary syphilis is defined by an initial lesion (chancre) and regional lymphadenopathy. The chancre generally heals in 4–6 weeks without treatment. This is followed by secondary syphilis during which time *Treponema pallidum* spreads to virtually all organs and is associated with systemic signs. Typical clinical manifestations in this stage are a maculopapular rash, distributed widely over the trunk and proximal extremities with frequent involvement of palms and soles. The rash is associated with generalised lymphadenopathy and constitutional symptoms such as fever, fatigue and weight loss. Like primary syphilis, the manifestations of secondary syphilis resolve spontaneously within 1–6 months. Tertiary syphilis (or latent syphilis) is the late stage of the infection. This phase is characterised by a plethora of manifestations affecting several systems including aortitis, meningeal syphilis, uveitis and wide spread gummas, a firm, proteinaceous mass, occasionally with necrotic centre.⁴

This linear progression may be altered, including inpatients coinfecting with HIV. In this population the progression to late stages of syphilis can occur more rapidly and the different findings of each stage may overlap. The rates of asymptomatic primary syphilis are higher and proportionally more HIV-patients present with late stage findings.⁵ Traditionally patients have been classified as having tertiary syphilis if diagnosed with neurosyphilis. There are now many case reports of HIV-infected patients that develop neurosyphilis in early stages of the disease^{6,7} and in a cohort of 231 HIV-infected patients with newly diagnosed syphilis, 41 were found to have neurosyphilis.⁸ Based on this evidence certain authors have recommended performing lumbar puncture in all patients with HIV and newly diagnosed syphilis,

even in the absence of neurological symptoms (this approach is not currently recommended by the Centers for Disease Control and Prevention).⁹

Gummas are also one of the hallmarks of tertiary syphilis. Most commonly found in the liver, they can also be found in the brain, skin, bone or heart or any other tissues. The histopathology of chancre differs from gummas—the later usually showing granulomatous inflammation, with central area of necrosis.^{4,10} Although rarely observable, *T. pallidum* can be demonstrated microscopically with immunostaining or by PCR.^{11,12}

Recent reports have identified patients in whom syphilis is associated with the development of inflammatory tumours. These lesions were not classical described as a finding of syphilis and seem to be different from gummatous lesions. These masses have been described in different locations such as the stomach,¹³ lungs,^{14,15} penis¹⁶ and liver.^{17,18} In contrast to gummas of tertiary syphilis this tumour-like masses differ in their histopathology, usually presenting with active inflammation, fibroblastic proliferation and plasma cell infiltration. They also typically have a higher burden of *T. pallidum* identifiable by immunostaining.¹⁸

The development of a liver mass seems to have been more frequently described in MSM. It has been postulated that the increased frequency in which the liver appears to be affected in MSM can be related to an increase delivery of *T. pallidum* to the liver through the portal circulation via anal intercourse.^{19,20} These masses have been more often described in earlier stages of the disease, as opposed to gummas that occur typically in tertiary syphilis.

In our case report the initial clinical scenario and laboratory testing suggested a diagnosis of secondary syphilis. The findings of rectal mass and liver masses associated to the presence of neurosyphilis led to some diagnosis uncertainty as these findings could represent tertiary syphilis. After careful review we have concluded that our case most likely represents syphilis in its early stages associated with inflammatory tumours and neurosyphilis.

Learning points

- Clinicians should be aware that syphilis in high-risk groups may not follow its typical course and atypical findings may be present, sometimes leading to diagnostic confusion.
- It is important to keep syphilis in the differential diagnosis as it is a relatively easy infectious disease to treat and failure to make a timely diagnosis may result in irreversible morbidity and even mortality.

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